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Those with diabetes, hypertension or CHD.

Reference: 1. Data on file. Pfizer Inc., New York, NY. **Detailed information available upon request.**

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Editorial

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Specialist in Cardiology

Dr. Bernard BL Wong

This is my real great honor and pleasure for me to join the Hong Kong Medical Diary as an editor. Cardiology is a rapidly advancing area in the medical arena. In every few months, there are new guidelines, investigation technologies, imaging modalities, medications and interventional devices popping up with a lot of supporting data and mega-trials. It is always a difficult task, even for the cardiologist to chase after all those new advances, season after season and year after year.

In this issue, we are really lucky to have an excellent panel of local radiologist and cardiologists -- Dr. Wai-hong Chen, Dr. Jay FL Kay, Dr. Albert WS Leung, Dr. Ivan YF Chan, Dr. Pui-yin Lee, Dr. Godwin TC Leung, Dr. Chi-ming Tam and Dr. Alex SB Yip. All of them are my very best friends and teachers. They really taught me a lot in the past decades.

I would also like to especially thank Dr. Ivan YF Chan, Dr. Pui-yin Lee, Dr. Godwin TC Leung, Dr. Chi-ming Tam and Dr. Alex SB Yip once again whose manuscripts will be published in the next March issue of the Hong Kong Medical Diary due to the limitation of pages in this issue. Thank you also to Dr. Godfrey CF Chan and Dr. Adrian YY Wu, the Co-Editors of the March issue for allowing the authors mentioned to place their manuscripts in their issue.

The primary aim of this February issue is to create a handy quick reference book, for the day in and day out clinical practice of all our dearest family practice colleagues and non-cardiology specialists. Bearing this in mind, all my best friends tried their very best in distilling the complicate new guidelines, new investigation technologies, imaging modalities, medications and interventional devices, writing them up in a simple, easy to digest way.

If this issue of the Hong Kong Medical Diary in Cardiology can help you in some day, some way of your daily clinical practice, then the aim of us was fulfilled.

We wish you all a prosperous, healthy and happy Year of the Pig.

Dr. Bernard BL Wong

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References: **1.** White WB, Lacourcière Y, Davidai G. Effects of the angiotensin II receptor blockers telmisartan versus valsartan on the circadian variation of blood pressure: impact on the early morning period. *Am J Hypertens.* 2004;17:347-353. **2.** Kario K. Clinician's Manual on Early Morning Risk Management in Hypertension. *London, England: Science Press*; 2004:50,55. **3.** Barnett AH, Bain SC, Bouter P, et al. Angiotensin-Receptor Blockade versus Converting-Enzyme Inhibition in Type 2 Diabetes and Nephropathy. *N Engl J Med.* 2004;351:1952-61. **4.** Redon J, Luque-Otero M, Martell N, et al. Renin-angiotensin system gene polymorphisms: relationship with blood pressure and microalbuminuria in telmisartan-treated hypertensive patients. *Pharmacogenomics J.* 2005;5:14-20. **5.** Neldam S, Edwards C, on behalf of the Athos Study Group. Telmisartan Plus HCTZ vs. Amloidopine Plus HCTZ in Older Patients With Systolic Hypertension: Results From a Large Ambulatory Blood Pressure Monitoring Study. *Am J Geriatr Cardiol.* 2006;15:151-160.

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Hypertension 2007' - Update on How to Choose and Prescribe the Best Medications for our Patients

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This article has been selected by the Editorial Board of the Hong Kong Medical Diary for participants in the CME programme of the Medical Council of Hong Kong (MCHK) to complete the following self-assessment questions in order to be awarded one CME credit under the programme upon returning the completed answer sheet to the Federation Secretariat on or before 28 February 2007.

Content:

Military action is important to the nation - it is the ground of death and life, the path of survival and destruction, so it is imperative to examine it.

~ The Art of War, Sun Tzu, 544-496BC

1. Introduction

Hypertension is one of the most important causes of stroke, coronary artery disease, peripheral artery disease, renal failure and congestive heart failure.[1] (Figure 1) In Hong Kong, based on the data collected in the year 1997, the prevalence is a bit less than 10% of the population[2]. There was no large scale epidemiology data available in the past 10 years. With reference to the ~ 10% increase of US prevalence[2] and the data of the other countries, my current Hong Kong prevalence estimation is ~ 25 - 30%. The prevalence in other countries is in Figure 2.

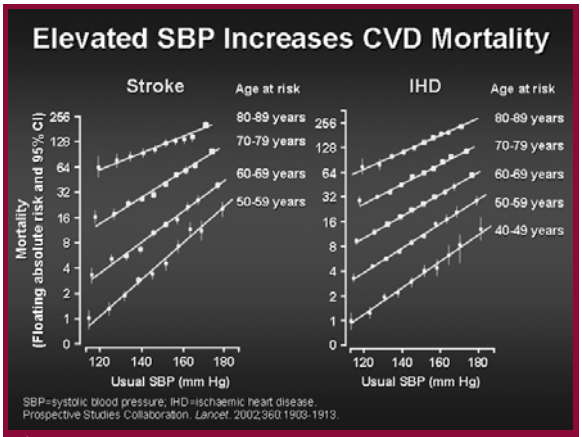


Figure 1

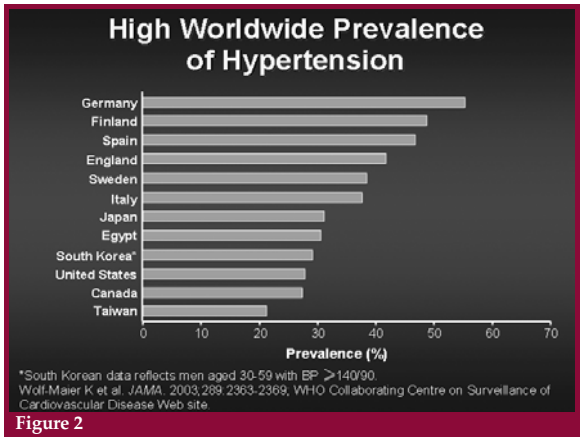


Figure 2

According to the National Health and Nutrition Examination Survey (NHANES), in US, the year 1999-2000, for 100 hypertensive citizens, only 70 of them were aware that they had hypertension, only 59 of them received medical treatment and only 34 of them with their blood pressure controlled (<140/90mmHg).[3]

So, in Hong Kong, for family doctors, the need for hypertension management is strong and the un-tapped market is huge.

In this article, we will have a quick glance on the recent updated international guidelines, a practical, straightforward and updated discussion on how to choose and prescribe anti-hypertensive medications. We will focus on the marvellous newly available anti-hypertensive medications. Once again, I hope that this article will be useful for their daily clinical practice to all my dearest colleagues.

For more detailed discussion on the historical development of hypertension concept and management, the development, rational and practical aspect of the guidelines, and the importance of systolic hypertension and life-style management, please kindly read my previous article "Hypertension- A guide to clinical practice, HKMA, CME Bulletin, Jan 2004 on http://www.hkma.org

"Leadership is a matter of intelligence, trustworthiness, humaneness, courage, and sternness."

~ The Art of War, Sun Tzu, 544-496BC

2. A quick glance on hot new guidelines - updated 2007

All four important guidelines were recently revised because of the numerous mega clinical trials in the recent 5 years:

World Health Organization - International Society of Hypertension
- WHO-ISH 2003' (Figure 3 & 4)[4]

European Society Of Cardiology - European Society of Hypertension
- ESC-ESH 2003 (Figure 5 & 6)[5]

Joint National Committee (United States)
- JNC VII 2003 (Figure 7 & 8)[6]

National Institute for Health and Clinical Excellence - Royal Hypertensive Society (United Kingdom)
- NICE 2006 (Figure 9)[7]

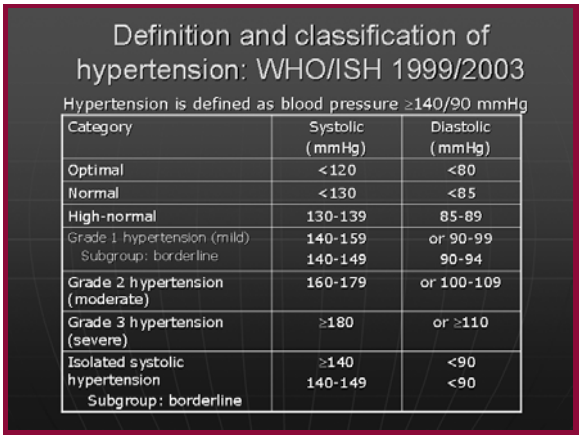
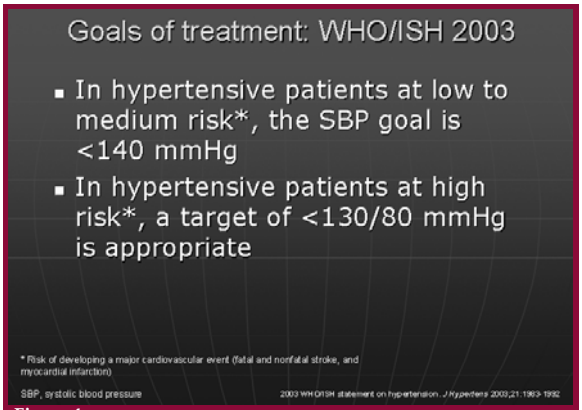


Figure 3



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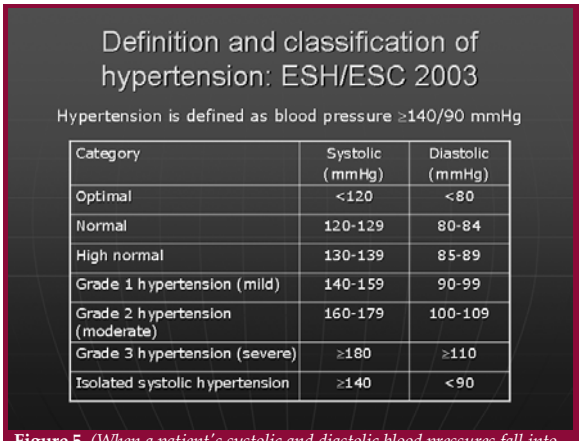
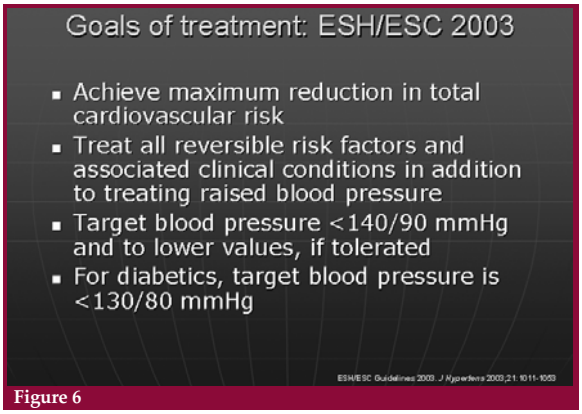


Figure 5. *(When a patient's systolic and diastolic blood pressures fall into different categories, the higher category should apply)*



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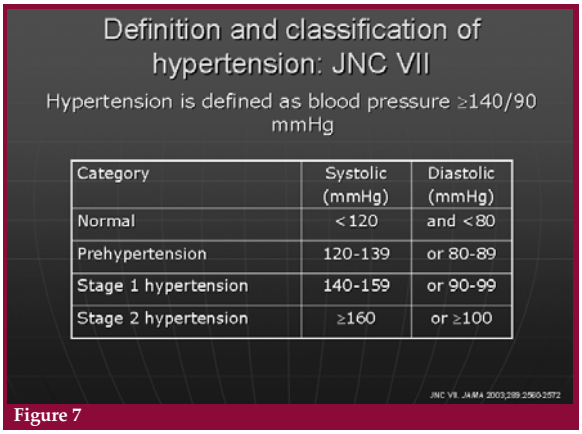

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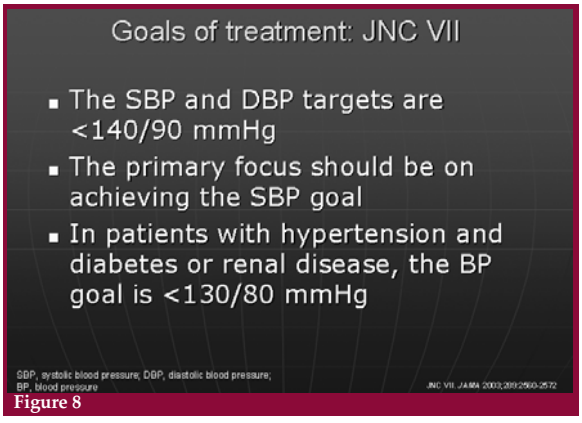

Figure 8

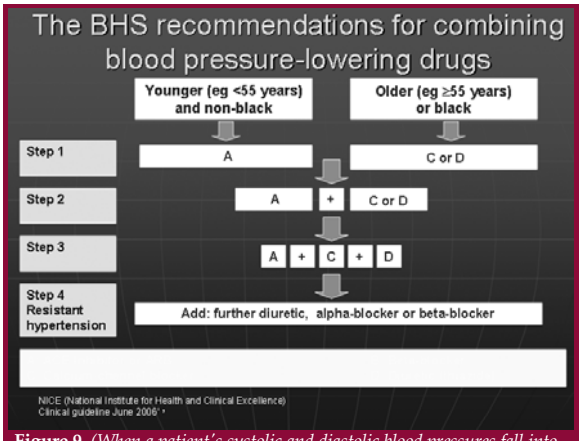

Figure 9. *(When a patient's systolic and diastolic blood pressures fall into different categories, the higher category should apply)*

We can all easily see that the following issue was unified throughout the whole world:
- Hypertension is Systolic BP ≧ 140mmHg and / or Diastolic BP ≧ 90mmHg (true for adult, all ages and both sexes)
- Optimal BP is Systolic BP < 120mmHg and Diastolic BP < 80mmHg
- Medication can start whenever your patient has Systolic BP ≧ 140mmHg and / or Diastolic BP ≧ 90mmHg
- The treatment target BP is Systolic BP < 140mmHg and Diastolic BP < 90mmHg.
- For high risk patients (diabetes and/or renal disease), the treatment target is further lowered to Systolic BP < 130 and Diastolic BP < 80mmHg[4, 5, 6, 7]

In my opinion, within the 4 international guidelines, the most recently updated treatment guideline of NICE 2006 is the easiest one to understand and use. (Figure 9)[7]
In figure 9, there are four classes of medications:

A. ARB or ACEI
B. Beta-blockers
C. Calcium channel blockers
D. Diuretics

For patients younger than 55, because of their stronger Renin- Angiotensin System (RAS), we can start with A (ARB or ACEI). C (calcium channel blockers) or D (diuretics) and will have a better result for patients older than 55 years old. A + C or D is the second step for patients with BP not reaching the target. The third step is A + C + D. For resistant hypertension, with the present of A + C + D, beta-blocker and alpha-blocker can then be added in the fourth step.

Because of the recent trials, there is a very recent change in the global practice and the NICE guideline:
Beta-blocker is not a first line drug anymore.
Beta-blocker is **only indicated for patients with hypertension plus**
- ischaemic heart disease and /or
- congestive heart failure
- young patients with ACEI/ARB intolerance[7]
(Figure 10 & 11)

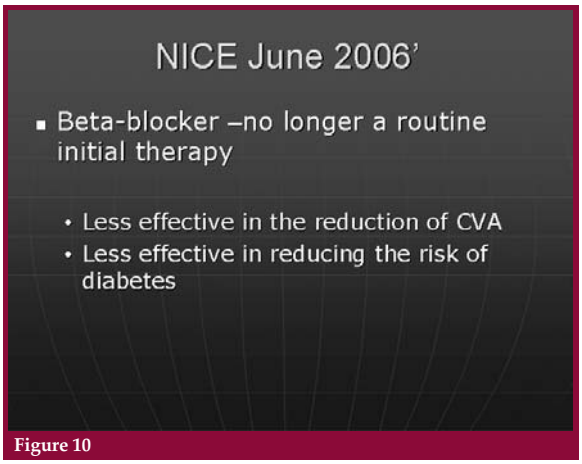

Figure 10

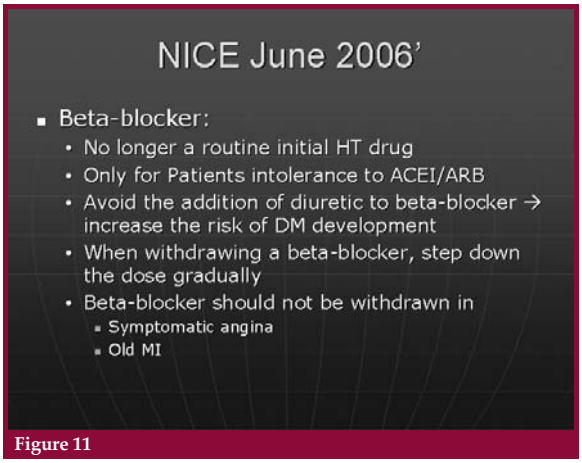

Figure 11

"The Way" means inducing the people to have the same aim as the leadership, so that they will share death and share life, without fear of danger

~ The Art of War, Sun Tzu, 544-496BC

3. Patient education - our key to success

During the first consultation... ...

For achieving an ultimate success, before the initiation of medical therapy, patients and their family must know and understand the followings important points:
- Hypertension is a chronic asymptomatic disease with dreadful outcomes (Framingham Risk Score table and ESC Heart Score table is very useful for explanation. Please refer to my article, Hypertension- A guide to clinical practice, HKMA, CME Bulletin, Jan 2004 on http://www.hkma.org)
- No curative treatment is available.
- In order to prevent the dreadful outcomes, the simple, single important thing is the permanent, persistent control of BP down to the target. Early and effective BP lowering rapidly reduces coronary heart disease (35%) and stroke event rate (45%)(ASCOT)[8]
- There are no such things as J shaped curve and hypotension. The keywords are:
  - o "the lower the better"
  - o "the lowest BP as tolerated"
- The medication side effects.
  - o "No free lunch!"
  - o All medications have side-effects
  - o New medications have fewer, milder side-effects and better compliance[9]
- The importance of Life-Style Management
  - o low salt diet and daily aerobic exercise > 30min
  - o lifestyle management can lower the blood pressure by ~ 5mmHg[10]
  - o Exercise can improve the cardiovascular morbidity and mortality up to 20-30%[11]
- The beauty of home monitoring
  - o In my clinic, I always have some simple, reliable and inexpensive electronic BP machines available for my patients to purchase
  - o All my patients regularly measure their morning and before sleep BP. They bring their recordings back during follow-up for discussion.
  - o This is a simple and useful way to improve self-

awareness, compliance and the accuracy of treatment.
- The cost of treatment.
  - o New medications are more effective in BP control, stroke prevention, milder in side effects but of course, more expensive. [7, 9]
  - o Let the patients and family to choose intellectually with our medical advice and according to their financial status.
  - o After all, the most important beauty of antihypertensive treatment is to control the blood pressure.
- Before the end of our first consultation, handouts and pamphlets on hypertension will be given for consolidation and reinforcement.

There when it (army) moves swiftly it is like the wind, when it goes slowly it is like a forest; it is rapacious as fire, immovable as mountains.
It is as hard to know as the dark; its movement is like pealing thunder.

~ The Art of War, Sun Tzu, 544-496BC

4. So many drugs - so many choices

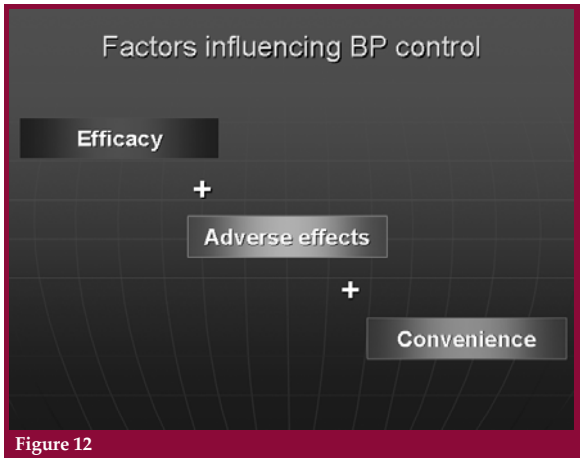

Figure 12

In this chapter, I will try to share with you a prescription habit based on a hybrid between
- most updated guidelines,
- most recently available trial results and
- my personal local experiences.

My personal local experiences may be different from yours. Cost is also a genuine consideration on prescription. New and better drugs are in no exception, a bit more expensive. Based on the evidence based data and your own personal experiences, you can easily develop a prescription pattern that can serve you the best.

The most important points affecting the choice of medications are illustrated on Figure 12.

Efficacy
- how fast and how low the blood pressure can be achieved

Adverse Effects
- o for ultra long term medical therapy, as hypertension, the side effects must be as minimal as possible to enhance the compliance

Convenience
- o Any long term medication more than once daily will not be successful.
- o Persistence rates with any type of antihypertensive drug decline quickly and are around 45% in the Netherlands and 55% in Italy after 1 year[9]
- o The percentage of fully adherent patients is approximately 30% after 1 year. Around 20-30% of patients are partially adherent. [9]

Now, let us go through the major medications one by one...

1. Angiotensin Receptor Blockers

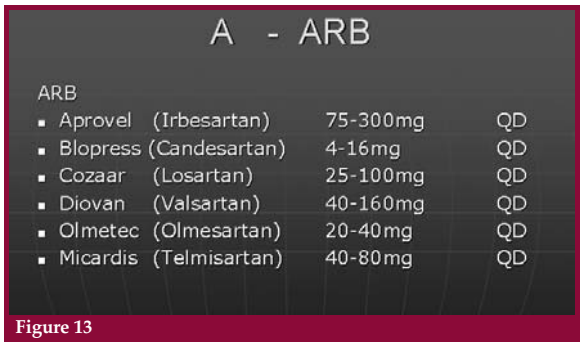

Figure 13

This is the newest class of medications. The prototype, Cozaar (Losartan) was marketed around 10 years ago.

Good Points:
- Effective medication for patients < 55 years old
- Extremely low in side effect rates
- Apart from hypertension, ARB is also effective in the morbidity and/or mortality reduction for patients with
  - o Congestive heart failure (CHARM)[12]
  - o Ischaemic Heart Disease (VALIANT)[13]
  - o Stroke (LIFE)[14]
  - o DM Nephropathy (Type II) (RENAAL)[15]

Bad Points:
- A bit more expensive (again, no free lunch)
- As a class, not very potent in BP lowering
- ≤ 3 % of patients will have dizziness

ARB is my favourite medication. It is effective in patients with simple or complicated hypertension[12, 13, 14, 15] and is virtually free from major and minor side effects. Most of my patients think that ARB is a very money valued medications for their long term combat with hypertension.

2. Angiotensin Converting Enzyme Inhibitor (ACEI)

Figure 14

ACEI is one of the most important milestones in the medical history. It is the predecessor of ARB. It opened a whole new world to the treatment of ischemic heart disease and congestive heart failure 25 years ago.

Good points:
- Cheap (hot market competition from generic companies)
- Effective for patients < 55 years old
- Apart from hypertension, ACEI is also effective in the morbidity and/or mortality reduction for patients with
  - Congestive heart failure (SOLVD[16], CONSENSUS[17])
  - Ischaemic Heart Disease (SAVE[18], HOPE[19], EUROPA[20])
  - Stroke (HOPE[19])
  - DM Nephropathy (Type I) [21, 22]

Bad points:
- Dry Cough
  - Very common in Asian populations (~ 15-50%)
  - Nocturnal and even daytime
  - Can be very bad, distressing and affecting the productivity and life-style of our patients
  - Can lower the rate of drug compliance for patients with cough
  - Higher optimal dose can not be achieved in many patients

ARBs with no side effect of cough. ARB has nearly all the capacity of ACEI. That is why ARB is gradually replacing ACEI, especially in the private sector. Nowadays, I seldom use ACEI in my clinic.

3. Beta-blockers

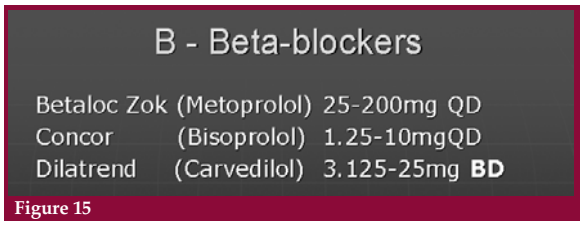

Figure 15

Among diuretics, Beta-blockers are one of the giants in the old days.

Good Points:
- Cheap (again, hot competition from generic companies)
- Effective in morbidity and mortality reduction for ischaemic heart disease[23] and congestive heart disease patients(CIBIS II)[24]
- Long history of usage with a lot of trials supports their efficacy in BP lowering (SHEP)[25]

Bad Points:
- Lethargy
- Reduce exercise tolerance (defeat our aim to get the patient on regular exercise)
- Cold hands and feet
- Erectile impotence (a real nightmare for male patients from 16 -100 years old!)
- Poor sleep and bad dreams (Atenolol cross the Blood Brain Barrier)
- Compared with CCB and ARB/ACEI, less favourable in stroke reduction and diabetes prevention.
- Adverse effect on the glucose and lipid profile, especially when using with diuretics[7]
- Adherence rates at 1 year are highest if individuals start therapy with ARBs, ACEIs and Calcium Channel Blockers.[28]

From the above points and the recent NICE guideline, I can't see a simple, single reason to use beta-blockers in simple hypertension apart from a cheaper price. For more than 3 years, I never use beta-blockers for simple hypertension. But they are actually invaluable good friends of mine for treating patients with ischaemic heart disease and congestive heart failure.

4. Calcium Channel Blockers

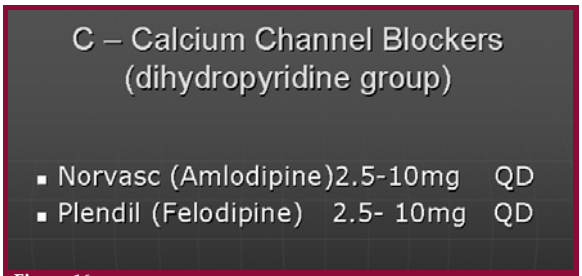

Figure 16

This is again, one of my favourite choices.

Good points:
- Very powerful in patients ≥ 55 years old
- Very powerful in Systolic BP reduction (as you can see in figure 1 and my article, Hypertension- A guide to clinical practice, HKMA, CME Bulletin, Jan 2004 on http://www.hkma.org, SBP is a more important killer than DBP)
- Plendil (felodipine) and Norvasc (Amlodipine) are safe for Congestive Heart Disease patients
- Apart from hypertension, Norvasc (Amlodipine) is also effective in ischaemic heart disease patients (CAMELOT)[26]
  - ↓30% cardiovascular death, nonfatal MI, resuscitated cardiac arrest, need for revascularisation, hospitalisation for angina and congestive heart failure, fatal and nonfatal stroke, transient ischaemic attack (TIA) and peripheral vascular disease (PVD)[25]
- Very low in side-effect rates and well tolerated

Bad points:
- A bit more expensive (again, No Free Lunch!)
- Mild flushing, palpitation, lower limb oedema (can be avoided by low salt diet)

A point to note:
I only mentioned the dihydropyridine Calcium Channel Blockers (CCB). Please kindly avoid the using of non-dihydropyridine CCBs. This sub-group of CCB included Herbesser (Diltiazem) and Isoptin (Verapamil). They are negative iontropic and chronotropic in nature. That means they slow down the pulse and decrease the pumping power of the heart. Serious side effects like bradycardia, heart block and heart failure can be the result. Again, I can not find a reason to use them in simple hypertension apart from money reasons. Actually they are the good friends for cardiologist in the treatment of tachyarrhythmias such as Supra-Ventricular Tachycardia (SVT)

5. Diuretics

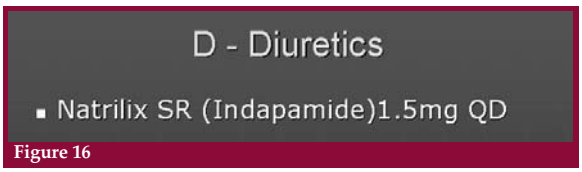

Figure 16

This is the oldest type of anti-hypertensive agent in common use. I only use Natrilix SR (Indapimide). In my experience, Lasix (Frusemide) and Moduretic (amiloride / hydrochlorothiazide) have a higher chance of getting electrolyte imbalance, fluctuation of blood pressure and gouty arthritis.

Good points:
- Cheap
- Effective in patients ≥55 years old
- Useful in hypertensive patients with lower limb edema and congestive heart failure

Bad points:
- Lethargy
- Reduce exercise tolerance (again, defeat our aim to get the patient on regular exercise)
- Hypokalaemia (can cause lethargy, leg cramps and arrhythmia)
- Polyuria (causing embarrassment, affects the life-style and leads to poor sleep)
- Precipitating Gouty Arthirtis
- Adverse effect on the glucose and lipid profile, especially when used with beta-blockers
- Poorer compliance and higher drop out rates.[28]

When used in combination (With CCB, ARB/ACEI) diuretics can be very useful in treating patients with hypertension and water retention.

6. Alpha Blockers

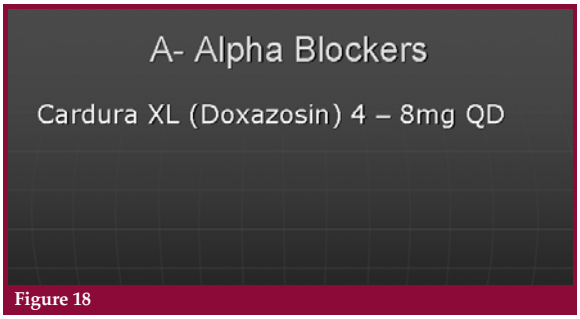

Figure 18

In elderly patients, Alpha-blockers can cause serious postural hypotension, dizziness and falls. This is one of the common cause of osteoporotic fracures. Fractures in elderly can have very gloomy outcomes. (up to 50% mortality in 1 year) In ALLHAT[27] trial, compared with the other group of medications, alpha blockers are also comparatively not as good in cardiovascular disease prevention. In my personal experience, as an alpha-blocker, Cardura XL (Doxazosin- slow release) has a more acceptable lower rate of postural hypotension attacks. I only use alpha-blockers in male hypertensive patients with true prostatism.

5. Combination Pills

Recent guidelines and my experience suggest that combination treatment can have lower side effect rates with a much higher BP lowering potency. Except the patients with very mild hypertension, I used to initiate the treatment with once daily, low dose combination pills for maximising the potency, minimising the side effects and achieving good compliance.

1. ARB + D

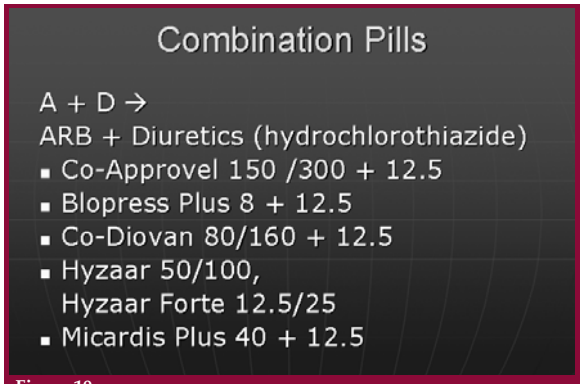

Figure 19

This is a very effective drug combination. Side effect rate is very low. Good for newly diagnosed patients. Because of the potassium conserving effect of ARB, very few patient need extra potassium supplement. They are my favourite combination pills

2. ACEI + D

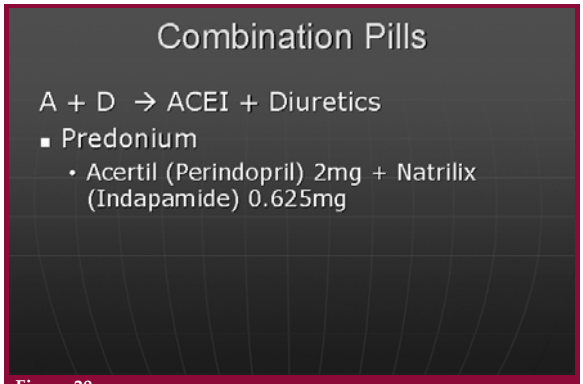

Figure 20

Same as ARB + D, this is a very effective pill. Unfortunately, dry cough is again not an uncommon problem.

3. B + D

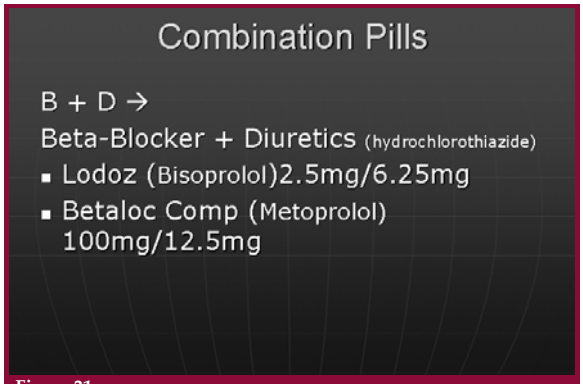

Figure 21

This is not a good choice for simple hypertensives. Lethargy, cold hands, erectile impotence, hypokalaemia, poorer lipid profile and even diabetes can be the results. But B + D is actually a very good combination for patients with hypertension and ischaemic heart failure.

4. B + C

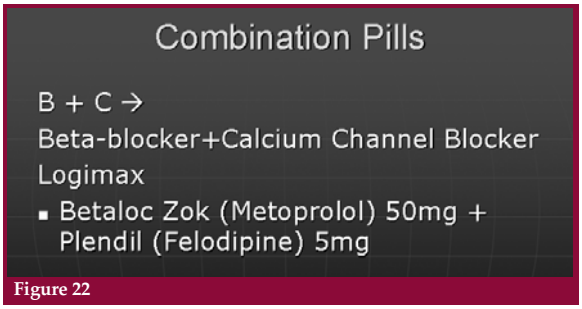

Figure 22

This is also not a perfect choice for simple hypertension. B + C combination is very useful for patients with hypertension and ischaemic heart disease.

The rule of military operations is not to count on opponents not coming, but to rely on having ways of dealing with them; not to count on opponents not attacking, but to rely on having what cannot be attacked

~ The Art of War, Sun Tzu, 544-496BC

5. One step further - treat the patient as a whole, don't forget the other risk factors

Nowadays, metabolic syndrome (Central obesity, hyperlipidaemia, hyperuricaemia, hypertension, impaired insulin sensitivity) is a very common problem in adults. Patients with multiple risk factors have a higher cardiovascular risk. We should treat all the risk factors as a whole to save their lives and their family. (Figure 23, 24 & 25)

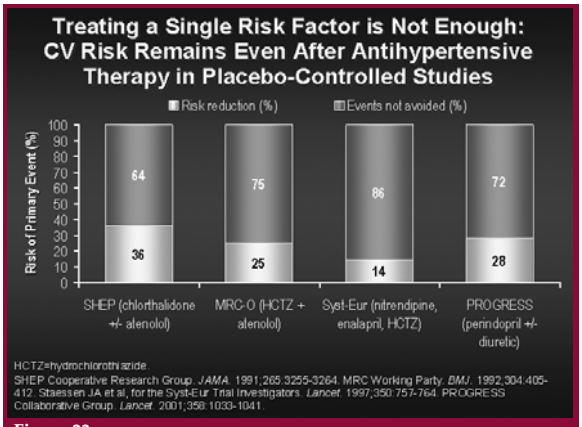

Figure 23

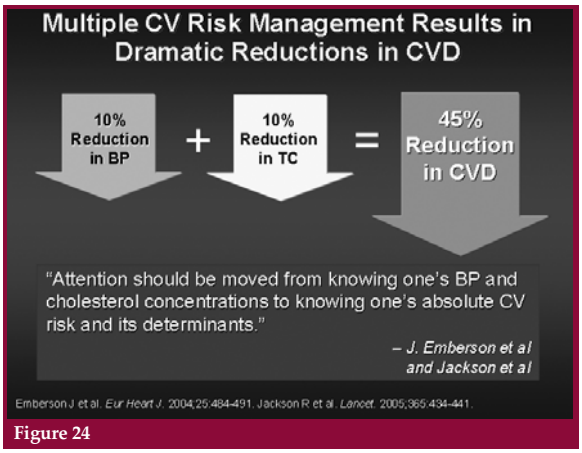

Figure 24

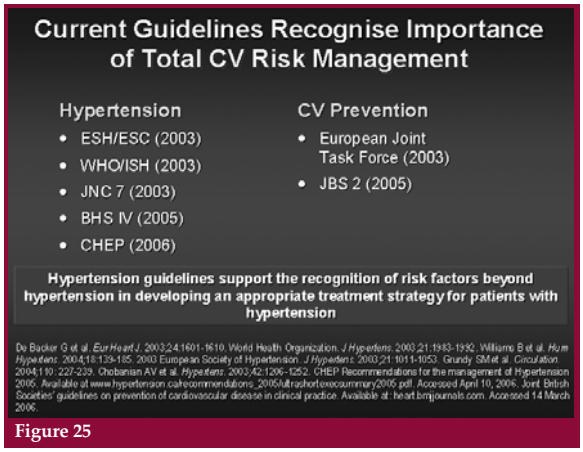

Figure 25

Some good news, there is a new combination drug Caduet (Lipitor + Norvasc). Caduet will be on the market in 2007'. This is truly a great move. The combination can further lower our patients' cardiovascular risks with a better compliance and a lower cost. I am looking forward that we are going to have more and more combination pills of this kind in the market.

In 2007, there is also another coming new drug with multiple effects, Acomplia (rimonabant). Coming from a new class, Acomplia regulates the over-activity of the central and peripheral endocannabinoid system. By selectively blocking CB1 receptor in central and peripheral tissues, rimonabant regulates energy balance, reduces abdominal obesity and improves glucose and lipid metabolism, thus resulting in the improvement of multiple cardiometabolic risk factors including blood pressure, lipid and glucose profile.[29]

The superior militarist foils enemies' plots; next best is to ruin their alliances; next after that is to attack their armed forces; worst is to besiege their cities.

~ The Art of War, Sun Tzu, 544-496BC[30]

6. Conclusion
- Intimate trust between Doctors and Patients + Patient education = Successful Hypertension Control

Hypertension is a very common and under-diagnosed disease in Hong Kong. It is the most important cause of disastrous diseases such as stroke, ischaemic heart disease, renal and heart failure. Hypertension can be easily controlled to the international target (<140/90mmHg) by life-style modification and medications. New medications such as calcium channel blockers and angiotensin receptor blockers are potent, user/Doctor - friendly, nearly side-effect free and money valued. Beta-blocker is no-longer a first line medication in our most up-dated guideline. The most important key for success is patient education delivered within an intimate trust between patients and doctors.

References

1. Levy D, Larson MG, Vasan RS, et al. The progression from hypertension to congestive heart failure. *JAMA* 1996; 275: 1557-1562
2. Janus ED. Clin Exp Pharmacol Physiol 1997;24:987-988;
3. Wang Y, Wang QJ. The preference of pre-hypertension and hypertension among US adults according to the new joint national committee guidelines: new challenges of the old problem. *Arch Intern Med* 2004; 164:2126-2134
4. 2003 WHO/ISH Statement on Hypertension.J Hypertens 2003;21:1983-1992;
5. ESH/ESC Guidelines 2003, J Hypertens 2003;21:1011-1053
6. JNC VII. *JAMA* 2003;289:2560-2572
7. Hypertension, Management of hypertension in adults in primary care: partial upodate of NICE (National Institute for Health and Clinical Excellence) Clinical Guideline 18, British Hypertension Society, Royal College of Physicians, June 2006
8. P. Sever et al, for the ASCOT investigators. *Lancet*, September 10, 2005
9. Sturkenboom M. et al. Presented at the 21st International Conference on Pharmacoepidemiology and Therapeutic Risk Management August 21-24 2005; Nashville, TN, USA
10. Whelton Sp, Chin A, Xin X, He J: effect of aerobic exercise on blood pressure: A metaanalysis of randomized controlled trials. *Ann Intern Med* 136:493, 2002.
11. Lee I-M, Skerett PJ: Physical activity and all-cause mortality-What is the dose response relation? *Med. Sci Sports exerc33*(6Suppl):S459,2001
12. Yusuf S. et al, CHARM Study, *Lancet* 2003; 362:759-66
13. Pfeffer MA, McMurray JJ, Velazquez EJ, Rouleau J-L, Kober L, Maggioni AP, Solomon SD, Swedberg K, Van de Werf F, White H, Leimberger JD, Henis M, Edwards S, Zelenkofske S, Sellers MA, Califf RM. Valsartan, captopril, or both in myocardial infarction complicated by heart failure, left ventricular dysfunction, or both. *N Engl J Med* 2003;349:1893-906
14. Dahlof B, Devereux RB, Kjeldsen S, et al., Losartan Intervention for End Point Reduction LIFE) , *Lancet* 2002; 359: 995-1003
15. Brenner BM, Cooper ME, de Zeeuw D, et al. Reduction of End Points in NIDDM with Angiotensin II Antagonist Losartan (RENAAL), *N Engl J Med* 2001;345:861-869,
16. The SOLVD investigators, Studies of Left Ventricular Dysfunction, *N Eng J Med* 1992; 327685-91
17. The Consensus trial Study group, Cooperative North Scandinavian Enalapril Survicval Study, *M Eng J Med* 1987;316:1429-35
18. Canadia Pfeffer MA, Braunwald E, Moye Effect of captopril on mortality and morbidity in patients with left ventricular dysfunction after myocardial infarction. Results of the Survival and Ventricular Enlargement Trial, *LA el al N Eng J Med* 1992; 327:669-77
19. HOPE study investigators, Effects of an angiotensin-converting-enzyme inhibitor, ramipril, on cardiovascular events in high-risk patients, *N Engl J Med* 2000;342:145-53
20. EURopean trial On reduction of cardiac events with Perindopril in stable coronary Artery disease Investigators, Efficacy of perindopril in reduction of cardiovascular events among patients with stable coronary artery disease: randomised, double-blind, placebo-controlled, multicentre trial (the EUROPA study), *Lancet* 2003;362:782-8
21. Parving HH. Hommel E, Jensen BR, Hansen HP: Long term beneficial effect of ACE inhibition on diabetic nephropathy in normotensive type I diabetic patients, Kidney Int 60:228-234, 2001
22. Kvetny J, Gregersen G, Pedersen RS: Randomized placebo-controlled trial of peridopril in normatensive, normoalbuminuric patients with type 1 diabetes mellitus. QJM 94:89-94, 2001
23. Chae CU, Hennekens CH: Beta blockers.In Hennekens CH (ed): Clnical Trials in Cardiovascular Disease: A Companion to Braunwald's Heart Disease. Philadelphia, WB Saunders, 1999, P84
24. The CIBIS II Investigators & Committees, *Lancet* 1999; 353:9-13
25. SHEP Cooperative Research Group, *JAMA* 1991;265:3255-64
26. Nissen SE, Tuzcu EM, Libby P, et al, for the Camelot investigators. Effect of antihypertensive agents on cardiovascular events in patients with coronary disease and normal blood pressure. The CAMELOT study: a randomized controlled trial. *JAMA*. 2004; 292:2217-2226.
27. ALLHAT Officers and Coordinators, Antihypertensive and Lipid -
20. Lowering treatment to prevent Heart Attack Trial (ALLHAT), *JAMA* 2002; 288: 2981-3007
28. Bloom BS., Continuation of initial antihypertensive medication after 1 year of therapy. *Clin Ther* 1998; 20:671-681
29. Eli V. Gelfand, Christopher P. Cannon, Rimonabant: A Cannabinoid Receptor Type I Blocker for the management of multiple cardiometabolic risk factors. *JACC* May 16, 2006 Vol 47, No 10, 1919-1926
30. The Art of War, Sun Tze, Translated by Thomas Cleary, Shambala Boston & London, 1991

MCHK CME Programme Self-assessment Questions

Please read the article entitled "Hypertension 2007' - Update on How to Choose and Prescribe the Best Medications for our Patients" by Dr. Bernard BL Wong, and complete the following self-assessment questions. Participants in the MCHK CME Programme will be awarded 1 CME credit under the Programme for returning completed answer sheets via fax (2865 0345) or by mail to the Federation Secretariat on or before 28 February 2007. Answers to questions will be provided in the next issue of The Hong Kong Medical Diary.

Questions 1-10: Please answer T (true) or F (false)

1. Hypertension is one of the most important causes of stroke, coronary artery disease, peripheral artery disease, renal failure and congestive heart failure.

2. Hypertension is Systolic BP≧ 140mmHg and / or Diastolic BP≧ 90mmHg (true for adult, all ages and both sexes)

3. Optimal BP is Systolic BP < 120mmHg and Diastolic BP < 80mmHg

4. For high risk patients (diabetes and/or renal disease), the treatment target is Systolic BP < 130 and Diastolic BP < 80mmHg

5. In NICE guideline June 2006', beta-blockers are not a first line drug anymore.

6. ARBs have dry cough as a common side effect

7. Beta-blockers would not cause lethargy and erectile impotence.

8. Diuretics would not cause polyuria

9. Alpha-Blockers have a superior stroke and ischaemic heart disease prevention power over the other types of anti-hypertensives

10. Patient education and intimate trust between doctors and patients is totally irrelevant for the long term success in hypertension control.

ANSWER SHEET FOR FEBRUARY 2007

Please return the completed answer sheet to the Federation Secretariat on or before 28 February 2007 for documentation. 1 CME point will be awarded for answering the MCHK CME programme (for non-specialists) self-assessment questions.

Hypertension 2007' - Update on How to Choose and Prescribe the Best Medications for our Patients

Dr. Bernard BL Wong

MBBS(HK), MRCP(UK), FHKCP(HK), FHKAM(Medicine), DME(Ireland), DCH(London)

Specialist in Cardiology

1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ 7 ☐ 8 ☐ 9 ☐ 10 ☐

Name (block letters):_____ HKMA No.: _____

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Answers to January 2007 issue

Recent Advances in the Management of Non-Small Cell Lung Cancer

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Cardiac Rehabilitation: Does It Really Matter?

Dr. Albert WS Leung

MBBS(HK), MRCP(UK), FHKAM(Medicine), FHKCP(HK), FRCP(Edin, Glasg)

Specialist in Cardiology

Dr. Albert WS Leung

Had you ever treated patients who survived a myocardial infarction (MI) by prescribing 'complete bed rest' for 3 to 5 days? Those days were gone, when the concept of cardiac rehabilitation (CR) drifted in. Nowadays, contemporary programmes have evolved into comprehensive multidisciplinary efforts that, in addition to exercise training, include modification of other risk factors as well as personal and vocational adjustment and education. However, in the United States, up to 90% of patients who could benefit from CR do not have it; of those who do, 25-50% drop out within weeks to months[1]. Factors contributing to this rate include transportation issues, motivation, comorbidities, misunderstanding of the value, reimbursement issues, and suboptimal referral rates. Neither is it better, across the Pacific, in our own city. What has gone wrong? Is the concept not evidence-based, not relevant, or not practical at all? As a matter of fact, every party in the medical service (the patient, the public, the service provider, the insurance, and the administrator) has to be well informed of what CR is.

The Past and the Present

In the 1910's, absolute bed rest for 6-8 weeks was the standard first step in recovery from MI. The rationale was that exertion could lead to ventricular aneurysm and ventricular rupture, and the hypoxia associated with exertion could lead to arrhythmia or recurrent MI. In the States, during World War II, successes in medical rehabilitation helped clinicians question the benefits of extended immobility. Early mobilisation first took the form of 'chair therapy' - patients to sit in a chair for 1-2 hours a day immediately after MI. This was gradually modified into the so-called phase I or inpatient CR, the goal of which was to condition the patient to carry out safely daily activities following discharge. The early programmes focused almost exclusively on exercise.

CR has now broadened to include all relevant aspects of secondary prevention, including physical activity counselling, exercise training, cessation of smoking, management of lipid levels, hypertension and diabetes, weight reduction, as well as psychosocial management. Apart from risk factor management, the goals of such a programmes include reduction in symptoms, reduction of risk of premature death, and improvement in quality of life. The 2006 update on the AHA/ACC guidelines for secondary prevention for patients with coronary and other atherosclerotic vascular disease has provided a summary on the mentioned goals[2]. Modern CR programmes include three stages: inpatient rehabilitation, outpatient rehabilitation, and maintenance. But the traditional terminology has described 4 phases.

Phase I CR, or inpatient CR, can be started as soon as a patient has been stabilised after initial treatment of an acute coronary syndrome (ACS) or coronary bypass graft surgery (CABG). It ranges from sitting in a chair, or take a few steps, to assisted walking, or even performing activities of daily living. Short stays nowadays in hospital make it difficult to do more than introduce patients to the disease process, the factors that maintain it, and strategies for reducing risk.

Phase II CR usually points to institution-based (usually hospital-based) programme conducted by a team of rehabilitation professionals. It lasts 2 to 3 months, and emphasises safe physical activity to improve conditioning with continued behaviour modification aimed at smoking cessation, weight loss, healthy eating, and other factors to reduce disease risk.

Phase III CR usually points to supervised rehabilitation lasting 6 to 12 months, preferably organised in a community service facility that can help establish a prescription for safe exercise and continue to emphasise risk factor reduction. This community-based approach allows a patient to have adjustment more geared towards his/her own real life situation, and is relatively less costly.

Phase IV CR points to long-term 'maintenance' programmes, often conducted in the same facilities as structured programmes provided in phase III, but with fewer staff. The model is usually a supervised health club, or a self-help patient group.

The traditional CR aims at patients who have experienced a MI or who have undergone CABG. In 1995, the Agency for Health Care Policy and Research Clinical Practice Guidelines[1] also recommended CR for patients with chronic heart failure, and for those who have undergone heart transplantation and heart valve surgery. With the expanded use of percutaneous coronary intervention (PCI), the need of CR for patients undergoing PCI has arisen from the horizon.

Evidence Base for CR

Considerably less clinical research has been focused on CR than on many other areas of cardiology. Yet there is sufficient evidence to support recommendation for CR. Numerous randomised clinical trials of exercise-based CR have been conducted in North America and Europe.

Virtually all of these trials had insufficient statistical power to demonstrate the efficacy of CR. Early meta-analyses[3,4] started to appear in 1988. In the latest meta-analysis[5], which included 48 trials with a total of 8940 patients, CR was associated with a significant reduction of 20% all-cause mortality and a significant reduction of 26% cardiac mortality. There were no significant differences in the rates of nonfatal MI and revascularisation. There were also greater reductions in total cholesterol level, triglyceride level, systolic blood pressure and self-reported smoking. Health related quality of life was similar among CR group and usual care group. The effect on total mortality was independent of coronary heart disease diagnosis, type of CR, dose of exercise intervention, length of follow-up, trial quality, and trial publication date. Many trials included in this meta-analysis were conducted in the last decade, in the present era of cardiovascular therapies and across the contemporary range of coronary artery disease (CAD) case mix, but these trials continued to report benefits of CR.

The risks associated with CR are low. Rates for cardiac arrest, acute MI, and cardiac death have been reported to be 8.9, 2.4, and 1.3 cases per million patient-hours of exercise[6]. For a typical programme serving 200-300 new patients per year, these event rates represent only one cardiovascular event about every 10 years. Studies of CR have demonstrated a lower cost per year of life saved (US$4,950 per life-year saved) than other commonly accepted standards of care, including thrombolytic therapy, CABG, and cholesterol-lowering medications, with one death prevented for every 32 to 72 person treated with CR[7]. In addition, CR has been shown to reduce costs associated with rehospitalisation[8].

The benefits of CR are multifactorial. Exercise itself appears to lower cardiovascular disease risk by effects on traditional coronary risk factors, as well as by improvement in cardiac physiology and biochemistry (fibrinolytic and endothelial function). In a review on exercise and the coronary circulation[9], it is found that in studies on CAD patients or in animal studies, exercise training can (a) improve endothelial dysfunction by restoring nitric oxide production; (b) lead to regression of coronary atherosclerosis; and (c) cause formation of collaterals.

Apart from exercise, many other factors account for the benefit of CR. Nutrition has direct effects on weight, serum lipids, blood pressure, blood sugar and insulin sensitivity, cardiac rhythm, endothelial function, and oxidative stress. At least 5 randomised trials have demonstrated that diet modification can provide significant reductions in cardiac events such as MI or sudden cardiac death[10-14]. Cessation of smoking yields a 50% reduction in the risk of a coronary event in the first year or two after quitting, with the risk of former smokers approaching that of non-smokers after 5-15 years. 2 meta-analyses have assessed the impact of psychosocial interventions in CR[15,16]. Both demonstrated a reduction in cardiac mortality and recurrent MI. Other key steps in CR included education as well as advice on sexual activity. Evidence on these aspects is inadequate.

Clinical trials under review focused in patients with CAD. Evidence on other patient categories is, however, growing. A review paper by Stewart et al[17] has given a comprehensive analysis on CR following PCI, heart transplant and heart valve surgery, and for chronic heart failure, as follows.

Despite the expanded use of PCI, there are few controlled studies of CR after these procedures. Theoretically, exercise training reduces the levels of fibrinogen and plasminogen activators and modulates platelet activation, thereby reducing the risk of restenosis and acute coronary syndromes after PCI. In one study[18], 93 patients were randomly assigned to receive a behaviourally oriented intervention or a control group. After 12 months, the intervention patients improved significantly on self-rated measures of smoking, exercise, and diet habits. Patients also lost weight, improved their exercise capacity, and experienced less chest pain during exertion.

Heart transplant patients experience persistent heart failure, diminished aerobic capacity, muscle atrophy, side effects of immunosuppressive medications, infections, rejection, and premature coronary atherosclerosis. Exercise training studies have demonstrated the potential for reversing or diminishing physiologic abnormalities in heart transplant patients. Improvements in aerobic capacity range between 20-50%. However, patients may still experience the consequences of their transplants. In a controlled study[19], the mean dose of prednisolone, the number of patients receiving antihypertensive medications, and the average number of rejection or infection episodes did not differ in the active treatment group (supervised programme) and the control group (home exercise). The published studies also do not address if CR can delay or prevent allograft arteriopathy, the diffuse atherosclerosis noted after heart transplantation.

The analysis on the existing trials of CR on chronic heart failure has shown that CR improves disease-related symptoms, quality of life, and clinical outcomes (including hospital re-admission and survival). Overall, prescribed exercise attenuates the fatigue and dyspnoea that limit exercise intolerance. Several mechanisms contribute to the improvement, including increased peak cardiac output, improved endothelial vasodilator function, and an improved neurohumoral axis. Exercise training also has direct effects on skeletal muscles. However, patients can still experience complications because of their overall increased risks due to LV dysfunction and an overactive sympathetic nervous system. To enhance safety, patients should undergo a period of supervised exercise to evaluate for potential heart failure related complications.

Heart valve surgery patients have no unique characteristics that differentiate them from patients with CAD. Cardiac haemodynamics and symptomatology with valve disease are similar to those of heart failure. After surgery, the average improvement without CR is one NYHA class. Many patients continue to exhibit abnormal changes in LV ejection fraction. The same paper has documented that exercise training is associated with improved aerobic capacity and physical working capacity in controlled trials on patients with valve prostheses. In particular, atrial fibrillation is a common arrhythmia in patients with heart valve surgery. Patients

with controlled atrial fibrillation can participate safely in exercise, but patient education to avoid the complications of anticoagulation therapy has to be emphasised.

CR in Hong Kong

In Hong Kong, CR service first appeared in the 1980's, but structured CR programmes were developed only after the white paper for rehabilitation was released in 1992, which elaborated clearly the government policies and expectations in rehabilitation. Since then, CR programmes, though limited in volume, spectrum and access, had been set up in a few public hospitals under Hospital Authority and in the community by the Community Rehabilitation Network of the Hong Kong Society for Rehabilitation.

The development continued in the late 1990's. A survey on the CR service in Hong Kong had been conducted in 2002 to review the situation after 10 years[20]. There were 18, 11, 7 and 5 public hospitals that had recruited patients to phases I, II, III and IV CR services respectively in the year 01/02. The community-based phases III and IV services were provided by the Community Rehabilitation Network as well as some patient self-help groups. The number of patients who had participated in the 4 phases of CR in the year 01/02 amounted to 4225, 1391, 396 and 428 respectively.

Patient categories included in various CR programmes were: acute MI, angina, post-CABG, post-PCI, heart failure, post-heart transplant, post-heart valve surgery, and post-device implantation (pacemaker or implantable cardioverter/defibrillator). The majority were comprehensive rehabilitation programmes utilizing exercise training, education, counselling, behavioural modification, psycho-social intervention and vocational counselling. In particular, Chinese-style supervised exercise such as Tai Chi was also incorporated. Apart from comprehensive programmes, case management model and home care had been provided to patients with special needs in pilot programmes, such as heart failure cases.

Another survey of a similar nature was conducted last year[21]. Only 14 public hospitals responded, and 10 of them had CR service. The result showed that the development on CR service had not gone further. Amongst these 10 hospitals, only 2025 patients had participated in CR programmes. The majority 1490 (73.6%) were CAD cases. The numbers participated in the 4 phases of CR in the year 2005 were 1088, 499, 206 and 222 respectively.

There was no formal survey on the provision of CR service in the private sector of Hong Kong until recently. A survey that was recently published demonstrated, however, that a small scale of service existed in the year 2005[22]. 9 private hospitals responded to the survey, and among them CR service was provided in 1, while 3 others were planning to implement CR programmes in the near future. The same survey also evaluated on the practices of private cardiologists. Out of the 21 responders, 6 had referred patients to join CR programmes in community-based institutes, private clinic settings or public hospitals. 17 (80.9%) cardiologists considered CR useful and 15 (71.4%) would consider referral of patients for CR should the service be available in more private hospitals.

The Barriers and the Way Forward

The proportion of patients that had participated in cardiac rehabilitation programmes remained low, if not very low, both globally and locally. The first potential barrier to CR participation is the failure of health-care providers (mainly doctors) to refer their patients to CR. A study[23] had demonstrated that some patients were referred more by doctors for CR than the others, namely younger age (<75 years), men rather than women, white patients, uncomplicated rather than complicated MI, presence of risk factors such as smoking, hypertension and high cholesterol, in-patient rather than out-patient, and having insurance cover.

In Hong Kong, CR is available in the public sector, but the discharge system and the workload of in-charge doctors may hinder referral of cardiac patients for CR. Assessment of CR referral should be included as a compulsory component of discharge protocols for hospitalised patients who are eligible for CR. In the private sector, more availability and access to CR service has to be encouraged. This can be solved when more private rehabilitation centres (hospital- or clinic-based) are available, or when access to community-based institutes is established.

Another major barrier is the attitude of patients towards CR, and their readiness to join the programmes. It has been estimated that less than 1/3 of patients referred to CR actually enroll in[24]. Drop-off rates are also substantial. Accountable patient factors include age, pre-morbid state, perceived benefits of CR, self-motivation, distance and transportation, family composition, social support, self-esteem, and occupation. In Hong Kong, the unique Chinese culture and belief of many patients on the way of rehabilitation is also important to the success of CR. Thus, more public education in the forms of talks, workshops and media programmes is essential. The latest Public Conference on Cardiac Rehabilitation, in conjunction with the 1st Asian Preventive Cardiology & Cardiac Rehabilitation Conference, taken place in December 2006, was the first of its kind to empower the public with updated knowledge on this topic.

More innovative and effective models of service delivery wait to be developed to improve the participation. We are encouraged by the development of the community-based rehabilitation programmes in the last decade. Apart from the existing Community Rehabilitation Network which is a non-government organisation, patient self-support groups such as Care for Your Heart and the Heart Club have been taking the important roles in community rehabilitation and their services should be further enhanced.

Apart from cardiology specialists, primary care physicians should also play a more definite role in providing and reinforcing the primary and secondary prevention services. The training of specialised personnel, designated staff and teams, clinical paths for management and collaboration between the providers are also prerequisite for success. We have got to augment both prevention and rehabilitation which is the key to control the epidemic of cardiovascular disease of the twenty-first century in Hong Kong.

References

1. Wenger NK, Froelicher ES, Smith LK, et al. Cardiac rehabilitation clinical practice guideline No.17. Rockville, MD: US Department of Health and Human Services, Public Health Service, Agency for Health Care Policy and Research, and the National Heart, Lung, and Blood Institute, 1995; Agency for Health Care Policy and Research Publication No. 96-0672.
2. Smith SC, Allen J, Blair SN, et al. AHA/ACC guidelines for secondary prevention for patients with coronary and other atherosclerotic vascular disease: 2006 update: endorsed by the National Heart, Lung, and Blood Institute. *Circulation*, May 2006; 113: 2363-72.
3. Oldridge NB, Guyatt GH, Fischer ME, et al. Cardiac rehabilitation after myocardial infarction. Combined experience of randomized clinical trials. *JAMA*, 1988; 260:945-50.
4. O'Connor GT, Buring JE, Yusuf S, et al. An overview of randomized trials of rehabilitation with exercise after myocardial infarction. *Circulation*, 1989; 80:234-44.
5. Taylor RS, Brown A, Ebrahim S, et al. Exercise-based rehabilitation for patients with coronary heart disease: Systematic review and meta-analysis of randomized controlled trials. *Am J Med* 2004; 116:682-92.
6. Van Camp SP, Peterson RA. Cardiovascular complications of outpatient cardiac rehabilitation programs. *JAMA* 1986; 256:1160-3.
7. Oldridge N, Perkins A, Marchionni N, et al. Number needed to treat in cardiac rehabilitation. *Cardiopulm Rehabil* 2002; 22:22-30.
8. Probstfield JL. How cost-effective are new preventive strategies for cardiovascular disease? *Am J Cardiol* 2003; 91:22G-27G.
9. Linke A, Erbs S, Hambrecht R. Exercise and the coronary circulation - alterations and adaptations in coronary artery disease. *Progress in Cardiovasc Diseases* 2006; 48:270-84.
10. Burr ML, Fehily AM, Gilbert JF, et al. Effects of changes in fat, fish, and fibre intakes on death and myocardial reinfarction: Diet and reinfarction trial (DART). *Lancet* 1989; 2:757.
11. Singh RB, Rastogi SS, Verma R, et al. Randomized controlled trial of cardioprotective diet in patients with recent acute myocardial infarction: Results of one year follow up. *BMJ* 1992; 304:1015.
12. Singh RB, Dubnov G, Niaz MA, et al. Effect of an Indo-Mediterranean diet on progression of coronary artery disease in high risk patients (Indo-Mediterranean Diet Heart Study): A randomisssed single-blind trial. *Lancet* 2002; 360:1455.
13. Ornish D, Scherwitz LW, Billings JH, et al. Intensive lifestyle changes for reversal of coronary heart disease. JAMA 1998; 280:2001.
14. de Lorgeril M, Salen P, Martin JF, et al. Mediterranean diet, traditional risk factors, and the rate of cardiovascular complications after myocardial infarction: Final report of the Lyon Diet Heart Study. *Circulation* 1999; 99:779.
15. Linden W, Stossel C, Maurice J. Psychosocial interventions for patients with coronary artery disease: A meta-analysis. *Arch Intern Med* 1996; 156:745.
16. Dusseldorp E, van Elderen T, Maes S, et al. A meta-analysis of psychoeducational programs for coronary heart disease patients. *Health Psychol* 1999; 18:506.
17. 17. Stewart KJ, Badenhop D, Brubaker PH, et al. Cardiac rehabilitation following percutaneous revascularization, heart transplant, heart valve surgery, and for chronic heart failure. *Chest* 2003; 123:2104-11.
18. Lisspers J, Sundin O, Hofman-Bang C, et al. Behavioral effects of a comprehensive, multifactorial program for life-style change after percutaneous transluminal coronary angioplasty: A retrospective, randomized controlled study. *J Psycho-Som Res* 1999; 46:143-54.
19. Kobashigawa JA, Leaf DA, Lee N, et al. A controlled trial of exercise rehabilitation after heart transplantation. N Engl J Med 1999; 340:272-7.
20. Lau ST, Lau CP. Cardiac Rehabilitation in Hong Kong: The local development. *J HK Coll Cardiol* 2003; 11(suppl2):PB64-65.
21. Chan NY, Lau ST, Lau CP, et al, on behalf of the Executive Committee, 1st Asian Preventive Cardiology and Cardiac Rehabilitation Conference.. A survey on the current status of cardiac rehabilitation service provision in the public hospitals in Hong Kong. Abstract, *J HK Coll Cardiol* 2006; 14(suppel2:B11)
22. Leung AW, Chan NY, Lau ST, et al, on behalf of the Executive Committee, 1st Asian Preventive Cardiology and Cardiac Rehabilitation Conference. A survey on the cardiac rehabilitation service in the private sector in Hong Kong. Abstract, *J HK Coll Cardiol* 2006; 14(supple2:B11).
23. Cortes O, Arthur HM. Determinants of referral to cardiac rehabilitation programs in patients with coronary artery disease: A systematic review. *Am Heart J* 2006; 151:249-56.
24. Thomas RJ, Witt RJ, Lopez-Jimenez F, et al. Quality indicators in cardiovascular care - The case for cardiac rehabilitation. *J Cardiopulm Rehabil* 2005; 25:249-56.

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Health Care Benchmarking

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What is Benchmarking?

Benchmarking is a process of comparison between the performance characteristics of separate, often competing organisations intended to enable each participant to improve its own performance in the marketplace. Its objectives are to obtain a clearer understanding of competitors and of customers' requirements. Benchmarking will also enable innovations (either of process or product) to spread more rapidly through an industry and across industries where appropriate (Beckford, 1998).

The benchmarking theory is built upon performance comparison, gap identification and changes in the management process (Watson, 1993; Camp, 1989; Karlof and Ostblom, 1993). By reviewing the benchmarking literature (Camp, 1989; Zairi, 1992; Smith et al., 1993; Rogers et al., 1995), it seems obvious that benchmarking:

- Helps organisations to understand where they have strengths and weaknesses depending upon changes in supply, demand and market conditions
- Allows organisations to realise what level(s) of performance is really possible by looking at others, and how much improvement can be achieved
- Helps organizations to improve their competitive advantage by stimulating continuous improvement in order to maintain world class performance and increase competitive standards
- Helps to better satisfy the customers' needs for quality, cost, product and service by establishing new standards and goals
- Promotes changes and delivers improvements in quality, productivity and efficiency; which in turn bring innovation and competitive advantage
- Is a cost effective and time efficient way of establishing a pool of innovative ideas from which the most applicable practical examples can be utilised
- To be keen on new developments within the related area, and improves the motivation of employees.

Despite these benefits, time constraints, competitive barriers, cost, lack of both management commitment and professional human resources, resistance to change, poor planning and short term expectations are regarded as the main problems affecting successful benchmarking research (Bendell et al., 1993). A poorly executed benchmarking exercise will result in a waste of financial and human resources, as well as time. Ineffectively executed benchmarking projects may have tarnished an organisation's image (Elmuti and Kathawala, 1997). Moreover, there is no single 'best practice' because it varies from one person to another and every organisation differs in terms of mission, culture, environment and technological tools available. Thus, there are risks involved in benchmarking others and in adopting new standards into one's own organisation. The 'best practice' should be perceived or accepted to be among those practices producing superior outcomes and being judged as good examples within the area. Finally, benchmarking findings may remove the heterogeneity of an industry since standards will themselves become globally standardised and attempts to produce differentiation may fail (Cox and Thompson, 1998).

Overall, benchmarking first requires senior management commitment, particularly to supporting actions arising from the exploration. Second, it requires staff to be trained and guided in the process to ensure that maximum benefit is obtained. Finally, it requires allocation of part of the relevant employees time to enable it to be carried out (Beckford, 1998).

Definitions

Benchmarking has been given many different definitions by different organisations and authors even though each aims to reach the same conclusion (see Table 1). It has been defined by Camp (1989) simply as "the search for industry best practice that leads to superior performance". In other words, benchmarking is a process of finding what best practices are and then proposing what performance should be in the future. The three principles of benchmarking are maintaining quality, customer satisfaction and continuous improvement (Watson, 1993).

Benchmarking studies are perishable and time sensitive. What is a standard of excellence today may be the expected performance of tomorrow. Improvement is a continuous process, and benchmarking should be considered as a part of that process. As a result, although different authors have defined benchmarking in different ways, all these definitions have a common theme, namely: the continuous measurement and improvement of an organisation's performance against the best in the industry to obtain information about new working methods or practices in other organisations (Kozak, 2004).

Benchmarking Process

As implied in the various definitions offered, benchmarking is a continuous process. It encourages the use of Plan-Do-Study-Act (PDSA) cycles when action planning and implementing improvements. The plan phase focuses on the various up front decisions such as the selection of functions/processes to benchmark and the type of benchmarking study on which to embark. In do, one delves in a self study to characterise the selected processes using metrics and documenting business practices. Furthermore, data (metrics and business practices) are collected on the company that is the benchmarking partner. Study refers to the comparison of findings via a gap analysis to observe whether negative or positive gaps exist between the benchmarking company and the benchmarking partner. Act refers to the launching of projects either to close negative gaps or maintain positive gaps. This is the stage that distinguishes benchmarking from "organised industrial tourism" (Pulsat 1994). The PDSA cycle is a recognised "framework for efficient trial-and-learning methodology" which emphasises action-based learning to generate knowledge and predict whether change will result in improvement (Langley et al 1996).

Types of benchmarking

The benchmarking literature can be mainly separated into two parts: internal and external benchmarking. Competitive, functional and generic benchmarkings are classified under external benchmarking (Camp, 1989; Zairi, 1992). The process is essentially the same for each category. The main differences are what is to be benchmarked and with whom it will be benchmarked.

Internal benchmarking

Internal benchmarking covers two way communication and sharing opinions between departments within the same organisation or between organisations operating as part of a chain in different countries (Cross and Leonard, 1994; Breiter and Kline, 1995). Once any part of an organisation has a better performance indicator, others can learn how this was achieved. Findings of internal benchmarking can then be used as a baseline for extending benchmarking to include external organisations (McNair and Leibfried, 1992; Karlof and Ostblom, 1993). All benchmarking processes should start by dealing with internal benchmarking because this requires an organisation to examine itself, and this provides a baseline for comparison with others (Breiter and Kline, 1995). Among advantages of internal benchmarking are the ability to deal with partners who share a common language, culture and systems, having easy access to data, and giving a baseline for future comparisons (Breiter and Kline, 1995). Therefore, the outcomes of an internal benchmarking can be presented quickly.

External benchmarking

External benchmarking requires a comparison of work with external organisations in order to discover new ideas, methods, products and services (Cox and Thompson, 1998). The objective is continuously to improve one's own performance by measuring how it performs, comparing it with that of others and determining how the others achieve their performance levels. This type of benchmarking provides opportunities for learning from the best practices and experiences of others who are at the leading edge.

COMPETITIVE BENCHMARKING refers to a comparison with direct competitors only. This is the most sensitive type of benchmarking activity because it is very difficult to achieve a healthy collaboration and cooperation with direct competitors and reach primary sources of information. It is believed to be more rational for larger organisations than smaller ones, as they have the infrastructure to support quality and continuous improvement (Cook, 1995). Its benefits include creating a culture that values continuous improvement to achieve excellence, increasing sensitivity to changes in the external environment and sharing the best practices between partners (Vaziri, 1992). It may however become difficult to obtain data from competitors and to apply lessons to be learnt from them. A further risk may include the tendency to focus on the factors that make the competitors distinctive instead of searching for the factors contributing to excellent performance (Karlof and Ostblom, 1993).

FUNCTIONAL BENCHMARKING refers to comparative research and attempts to seek world class excellence by comparing business performance not only against competitors but also against the best businesses operating in similar fields and performing similar activities or having similar problems, but in a different industry (Davies, 1990; Breiter and Kline, 1995). For instance, British Rail Network South East employed a benchmarking process to improve the standard of cleanliness on trains. British Airways was selected as a partner because a team of 11 people cleans a 250 seat jumbo aircraft in only 9 min. After the benchmarking exercise, a team of ten people was able to clean a 660 seat train in 8 min (Cook, 1995).

GENERIC BENCHMARKING refers to the comparisons of business function that are same regardless of business. This means that a hotel organisation's accounting department would look at the accounting department of a manufacturing organisation that has been identified as having the fastest operations. It is believed to be easier to obtain data in such arrangements, as best in class organisations are more likely to share their experiences. However, generic benchmarking can take a long time to complete, and research outcomes may need a lot of modification in order for organisations to set their own standards (Cook, 1995).

Benchmarking in Health Systems

All professionals involved in health care are under a duty of care, which involves ensuring the uniform provision of a high quality health service. A widely accepted definition of quality is "the degree to which health services for individual and populations increase the likelihood of desired health outcomes and are consistent with current professional knowledge" (Institute of Medicine, 2001b). The quality of health care is a complex multidimensional concept in constant need of analysis and clarification (Attres, 1993; Gillies, 1996).

Benchmarking was translated to the health system from

the management field and has been used to improve quality in the health services from the mid to late 1990s (Phillips, 1995; Bullivant, 1998 and Camp, 1998). Bullivant (1996) identifies common pitfalls to successful benchmarking, including among others gaining senior management "buy-in", getting started and maintaining momentum.

The comparison of data either within or between health care systems relies on data that may be derived at different levels within the health care system Benchmarking indicators in health have been defined as a measurement tool used to monitor and evaluate the importance of governance, management, clinical and support functions (Wait and Nolte, 2005). Many methodological challenges in the field of benchmarking related to the selection and quality of indicators used to make comparisons both within and between health systems (Mckee and James, 1997; Goddard et al., 2000; Walshe, 2003). Though benchmarking has become an intrinsic part of most developed health care systems, the impact of benchmarking initiatives on improvements in system performance and their integration within the existing policy processes remain to be elucidated (Blalock, 1999; Goddard et al., 2000).

Performance benchmarking is an activity of comparing performance levels to identify gaps in performance. Process benchmarking is the identification of root causes, which lead to achievement of superior performance. Patient experience benchmarking focuses upon meeting patient expectations (Kozak, 2004; Ellis, 2004).

Competitive benchmarking uses performance measures to inform how well or badly a person or company is performing against direct competitors. Comparative benchmarking focuses upon how similar functional activities are handled by different organisations. This removes the competitive edge and therefore provides a greater potential for learning. Collaborative benchmarking involves the sharing of knowledge about a particular activity, with all hoping to improve based upon what they learn (Ellis, 2004).

Clinical practice benchmarking is a process benchmarking which involves the structural comparison and sharing of best practice in clinical aspects of care (Ellis, 2000). It provides a quality assessment and continuous quality improvement approach that supports the development of quality care (see Figure 1) (Ellis, 1995; Ellis and Morris, 1997).

Benchmarks traditionally identified from `the leader in the field' (Camp 1989). In clinical practice, however, benchmarking arriving at the "benchmark of the best practise" demands the acceptance of all levels of evidence shown in Table 2 (Ellis, 1995; Ellis and Morris 1997). Unlike organizational benchmarks where best practice refers to actual systems and processes (Codling 1992; Zairi and Leonard 1994), clinical practice benchmarks include external consideration of what the standard of excellence consists of. This means that best practice can be aspirational, seeking to meet the identified expectations of a quality health service rather than limiting developmental activity to what is currently achieved by "leaders in the field". Although challenging the principles of objectivity and measurability stated in some benchmarking literature as essential (Watson 1993), clinical practice benchmarking encompasses fundamental benchmarking descriptors (Codling 1992; Bogan and English 1994; Zairi and Leonard 1994), supporting continuous quality improvement through comparison and sharing, moving benchmarking further along the sophistication axis shown in Figure 2.

As an example of benchmarking in health service, clinical practice benchmarking is being used by paediatric units in 27 National Health Service Trusts in the north west of England to promote the utilisation of available evidence in to practise (Ellis, 2000). The group was formed in response to members' concerns that there appeared to be inconsistencies in the quality of care across the UK. In addition, resources were being wasted through repetition of effort as practitioners in all areas strived independently to ensure delivery of evidence-based care in the same areas of practice, e.g. paediatric pain control. Figure 3 highlights the inequalities in practice at the commencement of clinical practice benchmarking. Figure 4 indicates that after 24 months of clinical practice benchmarking activity, there is apparently less variance in the benchmarking scores awarded by practitioners, which may suggest greater consistency in practice in the particular areas considered. In addition, for some factors, the median scores are also improving which suggests that the quality of care may also be improving. Networking promotes general exchange of information and also creates a wider supportive culture, especially important in areas of specialist practice where practitioners can feel isolated and ill informed. Benchmarks are re-scored every year and the evaluation of the project suggests not only that practice is developing, but also that by working together, sharing developments and innovations, practitioners are helping to ensure that wherever they are cared for, patients can expect a similar high standard of care.

Clinical governance activity is concerned with providing a systematic approach to improving and maintaining quality in service delivery and care provision. Essence of Care (Department of Health, 2001) involves the structural comparison and sharing of qualitative good practice. It supports local clinical governance activity to help improve quality (Ellis, 2001). It is a sophisticated clinical practice benchmarking approach, which was envisaged to become an integral and effective part of health service benchmarking to support continuous improvement in the quality of patient care and experiences (Ellis, 2001).

Essence of Care consists of a series of best benchmarks relating to areas of fundamental aspects of care that are crucial to the quality of patient experience, for example, privacy and dignity (Matykiewicz and Ashton 2005). Although in arriving at benchmarks of best practice all evidence types shown in Table 2 are taken into account, Essence of Care benchmark compilation expands evidence, with the inclusion of the opinions, experiences and indeed expectations of patients and their carers as the true respected authorities on what constitutes best practice. Learning from others, therefore, is necessarily expanded to learning with

others to develop innovative and new practice to meet patients' expectations and needs, rather than just replication of current good practice (Ellis, 2004).

Although the subjective patient experience is mentioned as central to the quality of the health service (Department of Health, 2000; Phillips, 1995), the use of the Essence of Care is inconsistent. There is an apparent continuing preoccupation in health service with measurement that can support quantitative comparison and elements of competition (Bullivant 1998). This reinforces the traditional view of acceptable benchmarking activity as a "management by fact, data driven" approach rather than a "management by gut" intuition based approach (Watson 1993). Camp (1989) states that considering what satisfies the customer in each individual practice will ensure improved overall performance that benefits customers. This view would support the health service in recognising the value of Essence of Care benchmarking activity accepting that benchmarking generally is not primarily viewed as being undertaken for performance monitoring or to provide comparative data but that it is accepted as a continuous quality improvement approach (Ellis, 2001; Matykiewicz and Ashton 2005). Valuable comparison and sharing can occur without objective measurement. With the necessary ownership, commitment and resource support of the staffs, Essence of Care activity should be encouraged and supported to improve the quality of health care (Ellis 2004).

Conclusion

Benchmarking is a valuable technique for quickly lifting the performance of an organisation. Benchmarking activity is not only about auditing practice to ensure practice is achieving required measurable outcomes but supports open comparison and sharing to allow continuous improvement and development.

The modern health service is being encouraged to ensure uniform provision of high quality health care. Benchmarking pushes the boundaries of best practice ever onwards. Practitioners, aware of developments elsewhere, can develop practice with minimal effort, concentrating resources on new areas for practice development. The potential of benchmarking in the health service has been developed from the quantitative measurement of performance and consideration of processes to the qualitative attainment of best practice around patient experience. The perceived immeasurability and subjectivity of Essence of Care and clinical practice benchmarks means that these benchmarking approaches are not always accepted or supported by health service organisations as valid benchmarking activity. Further research and applications are needed to ensure that benchmarking in health fulfills its objective, namely to further our understanding of where to focus policy efforts in order to improve the performance of health care systems.

Table 1. Benchmarking definitions.

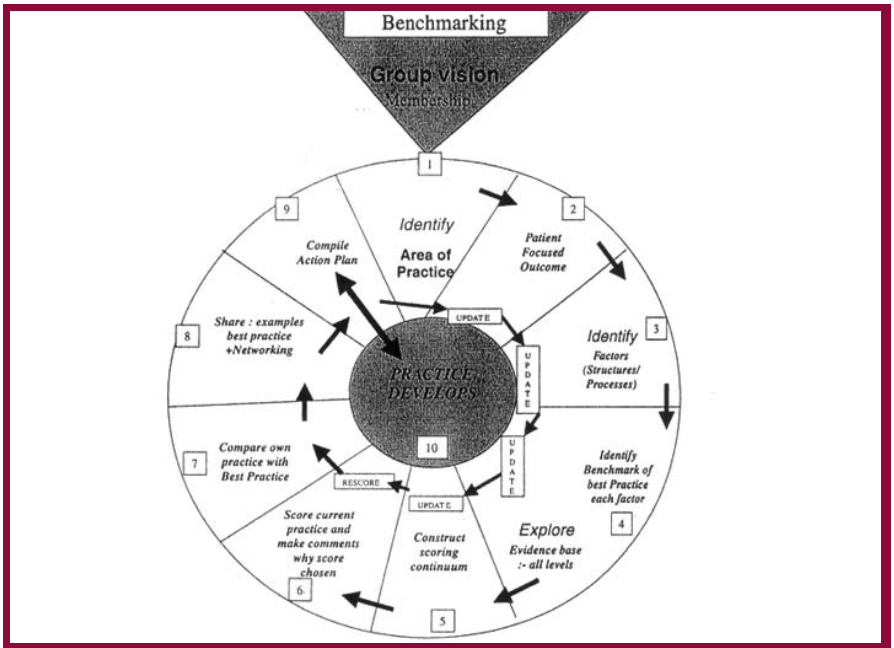
Authors	Definitions
Camp (1989)	The continuous process of measuring products, services and practices against the toughest competitors or those companies recognised as industry leaders.
Geber (1990)	A process of finding the world class examples of a product, service or operational system and then adjusting own products, services or systems to meet or beat those standards.
Vaziri (1992)	A continuous process comparing an organisation's performance against that of the best in the industry considering critical consumer needs and determining what should be improved.
Watson (1993)	The continuous input of new information to an organisation.
Kleine (1994)	An excellent tool to use in order to identify a performance goal for improvement, identify partners who have accomplished these goals and identify applicable practices to incorporate into a redesign effort.
Cook (1995)	A kind of performance improvement process by identifying, understanding and adopting outstanding practices from within the same organisation or from other businesses.
APQC[1] (1999)	The process of continuously comparing and measuring an organisation against business leaders anywhere in the world to gain information that will help the organisation take action to improve its performance.

[1]APQC stands for American Productivity and Quality Center.

Table 2. Classification of types of evidence used to identify benchmarks in clinical practice benchmarking (Ellis, 2000).

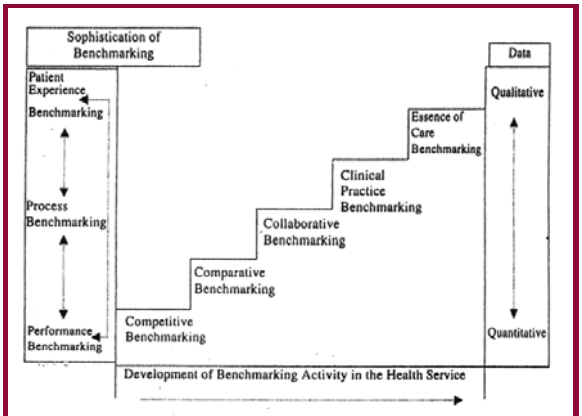
Classification of Evidence
1. NHS Centre for Research and Dissemination or Cochrane database review (systematic reviews)
2. Large scale well designed primary studies randomised controlled trials and other controlled trials
3. Large scale primary studies using other methodologies
4a. Descriptive studies and reports (including national and local standards, guidelines, customer surveys, support groups)
4b. The opinions and experience of respected authorities based on clinical experience, professional consensus

Figure 1. Clinical practice benchmarking cycle for continuous quality improvement towards best possible practice (Ellis, 2000)



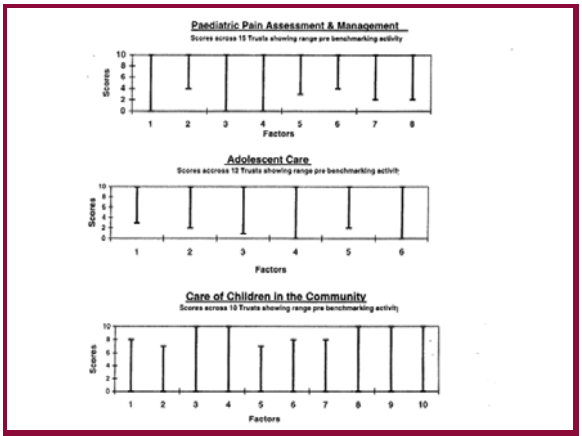
The patient focused outcome for each considered area of practice is agreed. Structural and process are then identified as factors that would support the attainment of the outcome. Through the consideration of all available evidence, the best possible practice within each factor is then arrived at and accepted as the benchmark of best practice.

Figure 2. Benchmarking activity in the health care service (Ellis, 2004)



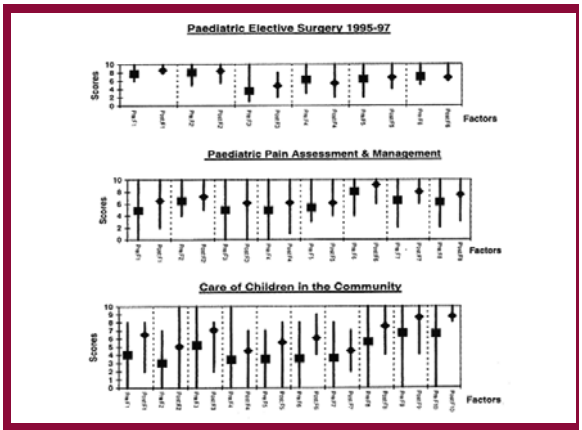
The potential of benchmarking in the health service has been developed from the quantitative measurement of performance and consideration of processes to the qualitative attainment of best practice around patient experience. The benchmarking activity used by the health service has been changed from types of benchmarking that focus upon performance to inclusion of types that can include consideration of process and more recently patient experience.

Figure 3. Practice benchmarking scores before quality improvement benchmarking activity (Ellis, 2000).



Each chart relates to a particular area of practice, e.g. adolescent care. Along the x axes are the different structures and processes that professional consensus identified essential for the attainment of patient focused outcomes that area of practice. They appear as Factor 1 (F1), Factor 2 (F2), etc. The y-axes relate to the scores awarded that factor. Practitioners were asked for each factor to compare their actual practice against a continuum of practice descriptors with a 10 score signifying attainment of the benchmark of best practice. Scores for the number of Trusts stated have been collated, to show for each factor range of scores, self awarded by practitioners across north west.

Figure 4. Benchmark scores comparing pre and post-benchmarking quality improvement activity in a specified area of practice: showing the range of scores and the median scores for each factor (Ellis, 2000).



= range of scores;
= division between different factors;
= prebenchmarking activity median score;
= postbenchmarking activity median score.

The y-axes show the scores awarded and the x axes relate to the structures and processes, the factors. The `pre' column relates to scores prior to any actual quality improvement activity, and the `post' column relates to scores after 24 months of quality improvement activity. The clinical practice benchmarking cycle has been completed and the inner update circle commenced with benchmarks re scored. The dotted line divides the different factors. After benchmarking activity, the range is closing in most factors.

References

Attree, M. 1993. An analysis of the concept "quality" as it relates to contemporary nursing care, International Journal of Nursing Studies, 30, 355 69.

American Productivity and Quality Center. 1999. What is best practice? http://www.apqc.org retrieved on 12 January.

Blalock, A. 1999. Evaluation research and the performance management movement. From estrangement to useful integration? Evaluation, 5(2), 117-49.

Beckford, J. 1998. Quality: A Critical Introduction, Routledge, Florence, KY, USA, 256-63.

Bendell, T., Boulter, L. and Kelly, 1993. Benchmarking for Competitive Advantage, Financial Times Pitman Publishing, London.

Bogan, C. and English, M. 1994. Benchmarking for Best Practices. Winning through Innovative Adaptation, McGraw Hill, New York.

Breiter, D. and Kline, S. 1995. Benchmarking quality management in hotels, FIU Hospitality Review, 13(2), 45 52.

Bullivant, J. 1996. Benchmarking in the UK National Health Service, International Journal of Health Care Quality Assurance, 9(2), 9-14.

Bullivant, J. 1998. Benchmarking for Best Value in the NHS, NHS Confederation, Financial Times Healthcare, London.

Camp, R. 1989. Benchmarking: the Search for Industry Best Practices that Leads to Superior Performance, ASQC Quality Press, Milwaukee, Wisconsin.

Camp, R. 1998. Global Cases in Benchmarking, Best Practices from Organisations Around the World, American Society for Quality Control Quality Press, Quality Resources, Milwaukee, New York.

Codling, S. 1992. Best Practice Benchrnarking: a Management Guide, Gower, Hampshire.

Cook, S. 1995. Practical Benchmarking: a Manager's Guide to Creating a Competitive Advantage, Kogan Page, London.

Cox, A. and Thompson, I. 1998. On the appropriateness of benchmarking, Journal of General Management, 23(3), 1 20.

Cross, R. and Leonard, P. 1994. Benchmarking: a strategic and tactical perspective, In: Dale, B.G. (ed.), Managing Quality, 2nd edn, Prentice Hall, New Jersey, 497 513.

Davies, P. 1990. Benchmarking, Total Quality Management, December, 309 10.

Department of Health. 2000. The National Health Service Plan for England: A Plan for Investment a Plan for Reform, Department of Health, London.

Department of Health. 2001. Essence of Care, Department of Health, London.

Ellis, J. 1995. Using benchmarking to improve practice, Nursing Standard, 9, 25 8.

Ellis, J. and Morris A. 1997. Paediatric benchmarking: a review of its development, Nursing Standard, 12, 43 6.

Ellis, J. 2000. Sharing the evidence: clinical practice benchmarking to improve continuously the quality of care. Journal of advanced Nursing, 32 (1), 215 25.

Ellis, J. 2001. Introducing a method of benchmarking nursing practice, Professional Nurse, 16(7), 1202-03.

Ellis, J. 2004. Evaluating the Effectiveness of Clinical Practice Benchmarking in Improving the Quality of Health Care, Unpublished PhD, University of Central Lancashire, Lancashire, UK.

Elmuti, D. and Kathawala, Y. 1997. An overview of benchmarking process: a tool for continuous improvement and competitive advantage, Benchmarking for Quality Management and Technology, 4(4), 229 43.

Geber, B. 1990. Benchmarking: measuring yourself against the best, Training, November, 36 44.

Gillies A. 1996. Improving the Quality of Patient Care, Wiley, Chichester.

Goddard, M., Mannion, R. and Smith, P. 2000. Enhancing performance in health care: a theoretical perspective on agency and the role of information, Health Economics, 9,95-107.

Hutton, R. and Zairi, M. 1995. Effective benchmarking through a prioritization methodology, Total Quality Management, 6(4), 399 411.

Institutes of Medicine. 2001b. Envisioning the National Health Care Quality Report, National Academy Press, Washington, DC.

Karlof, B. and Ostblom, S. 1993. Benchmarking: a Signpost of Excellence in Quality and Productivity, John Wiley & Sons, Chichester.

Kleine, B. 1994. Benchmarking for continuous performance improvement: tactics for success. Total Quality Environmental Management, Spring, 283 95.

Kozak, M. 2004. Destination Benchmarking: Concepts, Practices and Operations, CABI Publishing, MA, USA, 1-19.

Langley, G. et al. 1996. The improvement Guide-A Practical Approach to Enhancing Organizational Performance, Jossey Bass, San Francisco, 6.
Matykiewicz, L. and Ashton, D. 2005. Essence of Care benchmarking: putting it into practice, Benchmarking: An International Journal, 12(5), 467-81.

Mckee, M. and James, P. 1997. Using routine data to evaluate quality of care in British hospitals, Medical Care, 35(10), 102-11.

McNair, C. and Leibfried, K. 1992. Benchmarking; a Tool for Continuous Improvement, Harper Business, New York.

Phillips S. 1995. Benchmarking: providing the direction for excellence, British Journal of Health Care Management 1, 705-07.

Pulsat, B. 1994. Process Improvements Through Benchmarking, The TQM Magazine, 6(2): 37-40.

Rogers, D., Daugherty, P. and Stank, T. 1995. Benchmarking programs: opportunities for enhancing performance, Journal of Business Logistics, 10(2), 43 63.

Smith, G., Ritter, D. and Tuggle, W. 1993. Benchmarking: the fundamental questions, Marketing Management, 2(3), 43 8.

Vaziri, K. 1992. Using competitive benchmarking to set goals, Quality Progress, October, 81 5.

Walshe, K. 2003. International comparisons of the quality of health care; what do they tell us? QSHC, 12,4-5.

Watson, G. 1993. Strategic Benchmarking: How to Rate Your Company's Performance Against the World's Best, John Wiley & Sons, Canada.

Wait, S. and Nolte, S. 2005. Benchmarking health systems: trends, conceptual issues and future perspectives, Benchmarking: An International Journal, 12(5), 436-48.

Zairi, M. 1992. The art of benchmarking: using customer feedback to establish a performance gap, Total Quality Management, 3(2), 177 88.

Zairi, M. 1996. Benchmarking for Best Practice: Continuous Learning through Sustainable Innovation, Butterworth Heinemann, Oxford.

Zairi, M. and Leonard, P. 1994. Practical Benchmorking. The Complete Guide, Chapman & Hall, London.

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Aspirin Resistance: Is it Real and Does it Matter?

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Dr. Wai-hong Chen

Abstract

Platelets play a pivotal role in the pathophysiology of ischaemic complications of atherosclerotic cardiovascular disease. Aspirin is an oral antiplatelet drug that has been shown to reduce adverse clinical events across the wide spectrum of patients with atherothrombotic disease. However, recurrent ischaemic events still occur in a significant proportion of patients despite aspirin treatment. The concept of aspirin resistance therefore emerges. Although uniformed definitions and standardised assays are not yet available, numerous studies have documented the inter-individual variability in platelet responsiveness to aspirin. Evidence is also accumulating to demonstrate that hypo-responsiveness to aspirin in the laboratory (i.e. resistance) is associated with adverse clinical events in different patient populations. Clinical application of aspirin resistance will require proof from prospective randomised trials that modifications of antiplatelet therapy based on tests of antiplatelet responsiveness will improve the outcomes of patients with aspirin resistance.

Introduction

Platelets play a pivotal role in mediating thrombotic complications of atherosclerotic vascular disease and percutaneous coronary intervention (PCI). Platelets adhere to the subendothelium via interaction with collagen and von Willebrand factor at sites of spontaneous or iatrogenic plaque disruption. After adhesion, platelets undergo conformational changes and release agonists with prothrombotic and/or vasoactive properties such as thromboxane A_2 (TxA_2) and adenosine diphosphate (ADP), which result in amplification and propagation of platelet activation and aggregation, eventually leading to thrombus formation in combination with coagulation factors.

Aspirin is the cornerstone of oral antiplatelet therapy for preventing ischaemic events of atherothrombotic disease. Aspirin inhibits platelet cyclooxygenase-1 by irreversible acetylation of a serine residue at position 530, which prevents the conversion of arachidonic acid to TxA_2. The antithrombotic effect of aspirin is resulting from the decreased production of TxA_2, a potent vasoconstrictor and platelet agonist. The Antithrombotic Trialists' Collaboration reported that aspirin therapy was associated with 15% reduction in vascular mortality, 34% reduction in myocardial infarction (MI), and 25% reduction in stroke among high-risk patients with atherothrombotic disease.[1] Aspirin has also been shown to reduce the acute ischaemic complications of coronary angioplasty.[2-4]

Although the effectiveness of aspirin in reducing ischaemic events is well established, there are still a significant proportion of patients experiencing recurrent events despite aspirin treatment. Together with the recognition of laboratory variability in the response to aspirin, the concept of aspirin resistance emerged and has aroused attention in recent years. Despite the lack of universal agreement, the term aspirin resistance generally refers to the inability of aspirin to prevent ischaemic vascular events or the laboratory phenomenon of reduced effect of aspirin on one or more tests of platelet function. In addition to disagreement regarding definition(s), the clinical relevance of aspirin resistance is also uncertain. The remainder of this article is devoted to examine the evidence concerning the relationship of aspirin resistance and adverse clinical events among patients treated with aspirin.

Aspirin Resistance and Clinical Relevance

Variability in the response to aspirin has been recognised for decades. Aspirin's antiplatelet effect is usually quantified by assays of platelet aggregation or measurements of markers of platelet activation. Using different methodologies and varied definitions, the prevalence of hypo-responsiveness (i.e. resistance) to aspirin has been reported to vary from 5% to 60% among patients with atherosclerotic diseases involving different vascular beds.[5-16] The mechanisms of aspirin resistance are not clearly defined. Multiple clinical, pharmacodynamic, cellular, and genetic factors alone or in combination are likely to be involved.[17,18] A number of prospective studies relating laboratory measures of aspirin resistance to adverse clinical outcomes have been reported and are summarised in Table 1. Grotemeyer et al[19] determined aspirin responsiveness in 180 stroke patients 12 hours after an oral intake of 500 mg aspirin. Patients with a platelet reactivity index 1.25 were categorised as aspirin responders while those with an index >1.25 were defined as secondary aspirin non-responders (i.e. aspirin-resistant). All patients were prescribed aspirin 500 mg three times daily and were followed for 24 months. Stroke, MI, or vascular death were major outcome measures. Complete follow-up was obtained in 174 patients (96%). One-third of the patients were noted to be aspirin-resistant. Major events were noted in 29 patients: 5 (4.4%) in the aspirin responder group versus 24 (40%) in the aspirin-resistant group ($p < 0.0001$).

Mueller et al[9] evaluated 100 patients with intermittent claudication undergoing elective percutaneous balloon angioplasty. Aspirin was prescribed at a dose of 100 mg daily. They utilised the method of corrected whole blood aggregometry to define a normal response to aspirin as at least 20% reduction in platelet function with both ADP and collagen as agonists. Aspirin responsiveness was noted to fluctuate among the studied population on repeated monitoring. The incidence of aspirin resistance was 60% at each time point of measurement. At 52-week follow-up, 8 patients in the aspirin-resistant group were found to have reocclusion at the angioplasty site, compared with none of the patients with a normal response to aspirin (87% increase in risk, p = 0.0093). Eikelboom et al[20] performed a nested case-control study on 976 aspirin-treated patients, with documented or at high-risk of cardiovascular disease, from the Heart Protection Prevention Evaluation (HOPE) trial. Aspirin responsiveness was divided into quartiles by urinary 11-dehydrothromboxane B2 levels, a marker of in vivo thromboxane generation. After 5 years of follow-up, those patients in the upper quartile had 1.8-fold increase in risk for the composite of MI, stroke, or cardiovascular death (odds ratio [OR] 1.8; 95% confidence intervals [CI] 1.2 to 2.7; p = 0.009) when compared to those in the lower quartile, and the association was independent of traditional risk factors. There was a 2-fold increase in the risk of MI and 3.5-fold increase in the risk of cardiovascular death as well. Gum et al[21] enrolled 326 stable patients with cardiovascular disease treated with aspirin 325 mg daily for ≥ 7 days and defined aspirin resistance as a mean aggregation of ≥ 70% with 10 M ADP and a mean aggregation of ≥20% with 0.5 mg/ml arachidonic acid by optical platelet aggregation. Aspirin resistance was noted in 17 patients (5.2%). After a mean follow-up of 1.8 years, major events (death, MI, or stroke) occurred in 4 (24%) patients in the aspirin-resistant group, compared with 30 (10%) patients in the aspirin-sensitive group (p = 0.03). The Kaplan-Meier time-to-event curves for event-free survival showed late divergence of the event curves that remained to be explained. Multivariate analysis demonstrated that, in addition to other risk factors like increasing age, history of congestive heart failure, and elevated platelet count, aspirin resistance was an independent predictor of adverse outcomes (hazard ratio [HR] 4.14, 95% CI 1.42 to 12.06, p = 0.009). Chen et al[22] examined aspirin responsiveness in patients undergoing elective PCI treated with aspirin at 80-300 mg daily for at least 7 days, clopidogrel pretreatment with a loading dose of 300 mg at least 12 hours before intervention, and procedural anticoagulation using heparin. Using the aggregation-based point-of-care VerifyNow Aspirin, 29 (19.2%) out of the 151 enrolled patients were found to be aspirin-resistant, as defined by an aspirin reaction unit (ARU) ≥ 550. Patients with aspirin resistance were at increased risk of myocardial necrosis (OR 2.9; 95% CI 1.2 to 6.9; p = 0.015) determined by creatine kinase-myocardial band elevation, when compared with aspirin-sensitive patients. The mechanism was explored in a substudy by the same group, showing an inverse linear relationship between coronary flow reserve measured by the corrected Thrombolysis In Myocardial Infarction frame count method and ARU (r = -0.227, p = 0.014).[23] This observation implies that insufficient aspirin-induced platelet inhibition is associated with increased propensity of platelet thrombus formation during iatrogenic plaque rupture by PCI. The attendant distal embolization with or without local platelet-dependent thrombosis will lead to microvascular obstruction which is measurable by reduced CFR. After reporting the predictors and prevalence of aspirin resistance among 468 stable patients with coronary artery disease (CAD) using VerifyNow Aspirin,[16] Chen et al followed this cohort prospectively and found that after a mean follow-up of 379 ± 200 days, patients with aspirin resistance (n=128; 27.4%) were at increased risk of the composite outcome of cardiovascular death, MI, unstable angina requiring hospitalisation, stroke, and transient ischaemic attack compared with patients who were aspirin-sensitive (15.6% vs 5.3%, HR 3.12, 95% CI 1.65 to 5.91, p < 0.001).[24] Cox proportional hazard regression modelling identified aspirin resistance, diabetes, prior MI, and a low haemoglobin to be independently associated with major adverse long-term outcomes (HR for aspirin resistance 2.46, 95% CI 1.27 to 4.76, p = 0.007).

Conclusions

It is incontrovertible that inter-individual variability in platelet responsiveness to oral antiplatelet drugs exists. Analogous to biological responses to other pharmacological agents, the response to clopidogrel has been shown to display a continuous distribution[25] while similar response to aspirin may exist. On the basis of the aforementioned studies, there is substantial evidence illustrating hypo- or non-responsiveness to aspirin measured in the laboratory (i.e. resistance) is associated with adverse spontaneous (cardiovascular death, acute coronary syndromes, stroke or peripheral arterial occlusion) or procedure-related (myocardial necrosis after PCI or reocclusion after peripheral angioplasty) clinical events in diverse populations of patients with atherothrombotic disease in stable or unstable phase. Nevertheless, the currently available data are flawed by some major limitations. The samples size of these reports is small. Confounding variables are not adequately controlled by the study designs. Different definitions of aspirin resistance are used. Variable aspirin dosage, uncertain treatment compliance, and lack of pretreatment platelet activity assessment are noted in aspirin studies. Clinical application of aspirin resistance will require studies on larger populations that define aspirin resistance using consistent and reproducible assays, and correlate the measurements with clinical outcomes which can be improved by alterations in antiplatelet strategy (e.g., increasing dose of antiplatelet agent, adding or substituting second antiplatelet agent). Such prospective randomised trials are currently underway. The Clopidogrel for High Atherothrombotic Risk and Ischaemic Stabilisation, Management, and Avoidance (CHARISMA) trial comparing clopidogrel and aspirin versus placebo and aspirin for high-risk primary or secondary prevention was reported recently. Urinary 11-dehydrothromboxane B2 levels were checked in a substudy, enabling prospective assessment of the addition of clopidogrel to aspirin in reducing adverse events associated aspirin resistance.[26] The ASpirin non-responsiveness and Clopidogrel Endpoint Trial (ASCET) evaluates whether switching to clopidogrel will be superior to continued aspirin therapy in improving clinical outcomes among aspirin-resistant patients with angiographically documented CAD.[27] The Research

Evaluation to Study Individuals who Show Thromboxane Or P2Y12 Receptor Resistance (RESISTOR) trial will investigate whether modifying antiplatelet regimens could prevent myonecrosis post-PCI in patients with aspirin and clopidogrel resistance. The practice of antiplatelet therapy tailored to individual response may usher soon upon validation by these trials.

References

1. Antithrombotic Trialists' Collaboration. Collaborative meta-analysis of randomised trials of antiplatelet therapy for prevention of death, myocardial infarction, and stroke in high risk patients. *BMJ* 2002;324:71-86.
2. Barnathan ES, Schwartz JS, Taylor L, et al. Aspirin and dipyridamole in the prevention of acute coronary thrombosis complicating coronary angioplasty. *Circulation* 1987;76:125-34.
3. Schwartz L, Bourassa MG, Lesperance J, et al. Aspirin and dipyridamole in the prevention of restenosis after percutaneous transluminal coronary angioplasty. *N Engl J Med* 1988;318:1714-1719.
4. Lembo NJ, Black AJR, Roubin GS, et al. Effect of pre-treatment with aspirin versus aspirin plus dipyridamole on frequency and type of acute complications of percutaneous transluminal coronary angioplasty. *Am J Cardiol* 1990;65:422-426.
5. Mehta J, Mehta P, Burger C, Pepine CJ. Platelet aggregation studies in coronary artery disease. Part 4. Effect of aspirin. *Atherosclerosis* 1978;31:169-175.
6. Grotemeyer KH. Effects of acetylsalicylic acid in stroke patients. Evidence of nonresponders in a subpopulation of treated patients. *Thromb Res* 1991;63:587-593.
7. Pappas JM, Westengard JC, Bull BS. Population variability in the effect of aspirin on platelet function. Implications for clinical trials and therapy. *Arch Pathol Lab Med* 1994;118:801-804.
8. Buchanan MR, Brister SJ. Individual variation in the effects of ASA on platelet function: implications for the use of ASA clinically. *Can J Cardiol* 1995;11: 221-227.
9. Mueller MR, Salat A, Stangl P, et al. Variable platelet response to low-dose aspirin and the risk of limb deterioration in patients submitted to peripheral arterial angioplasty. *Thromb Haemost* 1997;78:1003-1007.
10. Hurlen M, Seljeflot I, Arnesen H. The effect of different antithrombotic regimens on platelet aggregation after myocardial infarction. *Scand Cardiovasc J* 1998; 32:233-237.
11. Gum PA, Kottke-Marchant K, Poggio ED, et al. Profile and prevalence of aspirin resistance in patients with cardiovascular disease. *Am J Cardiol* 2001;88:230-235.
12. Andersen K, Hurlen M, Arnesen H, Seljeflot I. Aspirin non-responsiveness as measured by PFA-100 in patients with coronary artery disease. *Thromb Res* 2002;108:37-42.
13. Macchi L, Christiaens L, Brabant S, et al. Resistance to aspirin in vitro is associated with increased platelet sensitivity to adenosine diphosphate. *Thromb Res* 2002;107: 45-49.
14. Grundmann K, Jaschonek K, Kleine B, et al. Aspirin non-responder status in patients with recurrent cerebral ischemic attacks. *J Neurol* 2003;250:63-66.
15. Wang JC, Aucoin-Barry D, Manuelian D, et al. Incidence of aspirin nonresponsiveness using the Ultegra Rapid Platelet Function Assay-ASA. *Am J Cardiol* 2003;92:1492-1494.
16. Lee PY, Chen WH, Ng W, et al. Low-dose aspirin increases aspirin resistance in patients with coronary artery disease. *Am J Med* 2005;118:723-727.
17. McKee SA, Sane DC, Deliargyris EN. Aspirin resistance in cardiovascular disease: a review of prevalence, mechanisms, and clinical significance. *Thromb Haemost* 2002;88:711-715.
18. Bhatt DL. Aspirin resistance: more than just a laboratory curiosity. *J Am Coll Cardiol* 2004;43:1127-1129.
19. Grotemeyer KH, Scharafinski HW, Husstedt IW. Two-year follow-up of aspirin responder and aspirin non-responder. A pilot study including 180 post-stroke patients. *Thromb Res* 1993;71:397-403.
20. Eikelboom JW, Hirsh J, Weitz JI, et al. Aspirin-resistant thromboxane biosynthesis and the risk of myocardial infarction, stroke, or cardiovascular death in patients at high risk for cardiovascular events. *Circulation* 2002;105:1650-1655.
21. Gum PA, Kottke-Marchant K, Welsh PA, et al. A prospective, blinded determination of the natural history of aspirin resistance among stable patients with cardiovascular disease. *J Am Coll Cardiol* 2003;41:961-965.
22. Chen WH, Lee PY, Ng W, Tse HF, Lau CP. Aspirin resistance is associated with a high incidence of myonecrosis after non-urgent percutaneous coronary intervention despite clopidogrel pretreatment. *J Am Coll Cardiol* 2004;43:1122-1126.
23. Chen WH, Lee PY, Ng W, et al. Relation of aspirin resistance to coronary flow reserve in patients undergoing elective percutaneous coronary intervention. *Am J Cardiol* 2005;96:760-763.
24. Chen WH, Cheng X, Lee PY, et al. Aspirin resistance and aadverse clinical events in patients with coronary artery Disease. *Am J Med* 2006, in press.
25. Serebruany VL, Steinhubl SR, Berger PB, et al. Variability in platelet responsiveness to clopidogrel among 544 individuals. *J Am Coll Cardiol* 2005; 45:246-251.
26. Bhatt DL, Topol EJ. Clopidogrel added to aspirin versus aspirin alone in secondary prevention and high-risk primary prevention: rationale and design of the Clopidogrel for High Atherothrombotic Risk and Ischemic Stabilization, Management, and Avoidance (CHARISMA) trial. *Am Heart J* 2004;148:263-268.
27. Pettersen AA, Seljeflot I, Abdelnoor M, Arnesen H. Unstable angina, stroke, myocardial infarction and death in aspirin non-responders. A prospective, randomized trial. The ASCET (ASpirin non-responsiveness and Clopidogrel Endpoint Trial) design. *Scand Cardiovasc J* 2004;38:353-356.

Table 1. Prospective Studies on Clinical Relevance of Aspirin Resistance

Investigators	Population studied	ASA dose (mg/day)	Definition of ASA resistance	Incidence of ASA resistance	Adverse Clinical Events
Grotemeyer et al[19]	Stroke patients (n=180)	1500	Platelet reactivity index >1.25	33%	~10-fold increased risk of vascular death, MI, or stroke at 2 years
Mueller et a[l9]	PAD patients (n=100)	100	≥20% reduction in platelet function using CWBA	~ 60%	87% increased risk of reocclusion at angioplasty site at 1 year
Eikelboom et al[20]	Patients with CAD, stroke, PAD, or DM plus ≥1 CV risk factor(s) (n=976)	Not specified	Quartiles of urinary 11-dehydro-thromboxane B2 levels	Not specified	1.8-fold increased risk of cardiovascular death, MI, or stroke at 5 years
Gum et al[21]	Patients with stable CV disease (n=325)	325	≥70% ADP-induced and ≥20% AA-induced optical platelet aggregation	5.2%	~4-fold increased risk of death, MI, or stroke at 1.8 years
Chen et al[22]	Patients undergoing elective PCI (n=151)	80-300	ARU≥550 in point-of-care platelet aggregation assay	19.2%	2.9-fold increased risk of CK-MB elevation after PCI
Chen et al[24]	Patients with CAD (n=468)	80-300	ARU≥550 in point-of-care platelet aggregation assay	27.4%	2.5-fold increased risk of death, MI, stroke, TIA, or UA hospitalisation at 1.0 years

AA = arachidonic acid; ADP = adenosine diphosphate; ARU = aspirin reaction unit; ASA = aspirin; CK = creatine kinase; CV = cardiovascular; CWBA = corrected whole blood aggregometry; DM = diabetes mellitus; MI = myocardial infarction; PAD = peripheral arterial disease; PCI = percutaneous coronary intervention; TIA = transient ischaemic attack; UA = unstable angina

Diploma in Child Health Examination (DCH) 2007

The Hong Kong College of Paediatricians (HKCPaed) and the Royal College of Paediatrics and Child Health (RCPCH) will hold a Joint Diploma in Child Health Examination in Hong Kong this year, awarding DCH (HK) and DCH (International) to successful candidates.

The Examination is divided into two parts, Written (MRCPCH Pt IA) and Clinical. The MRCPCH Part 1A Examination is held three times a year in Hong Kong. The next MRCPCH Part 1A Examination will be held on **Tuesday, 8 May 2007.** The examination fee is **HK$3,200** for Part IA. Candidates who wish to enter the examination must hold a recognized medical qualification in Hong Kong.

Application: Candidates who wish to sit the examination in Hong Kong **MUST** apply through the Hong Kong College of Paediatricians (HKCPaed). For application details, please visit the HKCPaed website at www.paediatrician.org.hk or call the College Secretariat at 28718871.

Deadline for Application: Friday, 23 February 2007

Important Notice

New Clinical Examination for DCH from March 2006

A new format of the DCH clinical examination has been adopted since March 2006. Details of the new format and other relevant information can be viewed on the RCPCH website at: www.rcpch.ac.uk

Dermatological Quiz

Dr. Lai-yin Chong

MBBS(HK), FRCP(Lond, Edin, Glasg), FHKCP, FHKAM(Med)

Yaumatei Dermatology Clinic, Social Hygiene Service

Dr. Lai-yin Chong

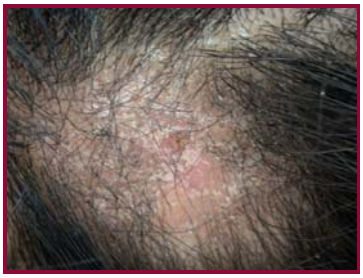


Fig 1
Patchy alopecia with scaling

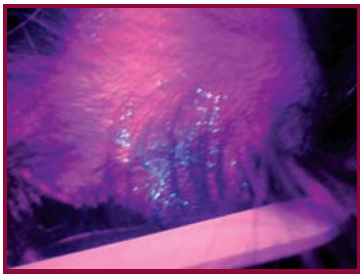


Fig 2
Same patient showing the same site

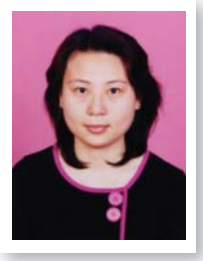
A 10-year-old girl was referred from the Student Health Service with a preliminary diagnosis of alopecia areata. She had patchy hair loss at the vertex in recent one month. There is no significant family history, but she had a cat at home.

Questions:

1. What is your diagnosis?
2. What is the clinical examination shown in Fig 2?
3. What are the other differential diagnoses for patchy alopeica?
4. How do you manage this patient?

(See P. 37 for answers)

The new FMSHK team



Ms. Carmen Y.C. Cheung has been appointed as the Executive Officer of FMSHK since 3 January 2007, a role she comes to after over 10 years experience in administrative and secretarial works. She obtained her Degree in Computing Information System in 1999.

In her leisure time, Carmen likes to listen to Chinese flute music and to travel to different places with her husband.

The Federation Annual Dinner 2006 - A GoGo to Count Down

FMSHK hosted The Federation Annual Dinner 2006 on 31 December 2006 at Run Run Shaw Hall, Hong Kong Academy of Medicine. It provided an opportunity for everyone to take a walk down memory lane and experience life in the 1960's. There was lively entertainment including songs from the 60's played from a live band, 60's fashion show, vintage car display, delicacies and games. One of the highlights of the evening was a special performance by Ms Irene Rhyder, who captured everyone with her voice and songs.

Please visit our homepage www.fmshk.org to view and order more exciting photos of the event

Count Down Ceremony from left to right: Dr Chan Chi Kuen, 1st Vice President, FMSHK; Dr Dawson Fong, President, FMSHK; Dr Grace Tang, President, Hong Kong Academy of Medicine; Dr Luk Kwok Fai; Dr Chan Chok Wan, President, The Hong Kong Society of Child Neurology & Developmental Paediatrics

Vintage car show from left to right: Dr Albert Lee, Dinner Chairman, FMSHK; Mr Nelson Lam, Honorary Treasurer, FMSHK; Dr the Hon. Kwok Ka Ki, Legislative Council Member; Dr Dawson Fong, President, FMSHK; Dr Richard Lo; 1954 Mercedes-Benz 300S

60's fashion show: Dancers and models from Hong Kong Institute of Vocational Education - Shatin

Special performance from Ms Irene Rhyder

News from Member Societies:

Hong Kong Society of Nephrology
Dr LUI Sing Leung has replaced Dr WONG Kim Ming, Francis as the society's representative effective from December 1st 2006.

The Hong Kong College of Paediatricians.
New office-bearers for the year 2006-2009 are as follows: President: Prof. Louis Chung Kai LOW, Vice President: Dr Alex Kwok Hing CHAN, Hon. Secretary: Dr WONG Sik Nin, Hon. Treasurer: Dr Frederick Wai Keung KO

The Hong Kong College of Family Physicians
New office-bearers for the year 2007 are as follows: President: Dr IP Kit Kuen, Andrew, Vice President (General Affairs): Dr LO Chun Hung, Raymond, Vice President (Education & Examinations): Dr CHAN Hung Chiu, Hon. Secretary: Dr LAM Wing Wo, Edmund, Hon. Treasurer: Dr TSOI Wai Wang, Gene.

The Hong Kong Orthopaedic Association
New office-bearers for the year 2007-2008 are as follows: President: Dr FUNG Kwai Yau, President Elect: Dr YIP Siu Fai, Henry, Vice President: Dr CHAN Kwok Pui, Brian, Hon. Secretary: Dr WONG Nan Man, Raymond, Hon. Treasurer: Dr CHAN Chi Fai, Samson.

Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
				★HKMA Council Meeting 1	★健康服務助理員訓練課程 (TC-HCA-0306) ★Cardio Rhythm 2007 2	★Cardio Rhythm 2007 ★3rd HKMA Exercise Prescription Certificate Course (Module 8-10) 3
★Cardio Rhythm 2007 ★HKMA Structured CME Programme Year 06/07 (XI)- Endocrinology & Neurosurgery 4	★A Lady with Urinary Incontinence ★The Role of the Medical Expert in Personal Injury Litigation ★HKMA CME Luncheon Lecture on New Laws on Smoking Ban and Smoking Cessation Programme for Patients 5	★HKMA Newsletter Editorial Meeting 6	★健康服務助理員訓練課程 (TC-HCA-0306) 7	★HKMA Structured CME Programme with Hong Kong Sanatorium & Hospital Year 2007 (II) - Therapeutic Efficacy of Tomotherapy: The Hong Kong Sanatorium & Hosptial Experience 2005-2006 8	★健康服務助理員訓練課程 (TC-HCA-0306) 9	★3rd HKMA Exercise Prescription Certificate Course (Module 11-14) ★HKMA Refresher Course for Health Care Providers 2006/2007 (VI) - Common Skin Conditions and Their Imitators ★Cancer Imaging 2007 - Joint Meeting of the International Cancer Imaging Society & Hong Kong College of Radiologists 10
★Cancer Imaging 2007 - Joint Meeting of the International Cancer Imaging Society & Hong Kong College of Radiologists ★2nd Certificate Course in Recent Medical Advances for General Practitioners ★HKMA Structured CME Programme Year 06/07 (XI)- Cardiology 11	12	13	★健康服務助理員訓練課程 (TC-HCA-0306) ★The 2007 WHO Classification For Brain Tumours: Some Key Points For Neurosurgeons 14	★Public Private Interface - Electronic Patient Record (PPI-ePR) Pilot Project Sharing Session 15	★健康服務助理員訓練課程 (TC-HCA-0306) 16	17
18	19	20	21	22	23	24
25	26	27	★健康服務助理員訓練課程 (TC-HCA-0306) 28			

Date / Time	Function	Enquiry / Remarks
1 THU 8:00 pm	**HKMA Council Meeting** Organised by: The Hong Kong Medical Association # HKMA Headquarter Office, 5/F., Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Christine WONG Tel: 2527 8285
2 FRI (7,9,14,16,28) (3,4)	**健康服務助理員訓練課程 (TC-HCA-0306)** Organised by: College of Nursing, Hong Kong **Cardio Rhythm 2007** Organised by: Hong Kong College of Cardiology & Chinese Society of Pacing and Electrophysiology # Hong Kong Convention & Exhibition Centre, Wanchai, Hong Kong	Sugar Tel: 2572 9255 Fax: 2838 6280 Secretariat Tel: 2559 5888 Fax: 2559 6910 Email: info@cardiorhythm.com Website: www.cardiorhythm.com
3 SAT 1:00 pm	**3rd HKMA Exercise Prescription Certificate Course (Module 8-10)** Organised by: The Hong Kong Medical Association, Department of Health, Physical Fitness Association of Hong Kong, China & Queen Elizabeth Hospital Chairman: Dr. Y.S. CHAN & Dr. C.F. YEUNG Speaker: Various # Lecture Theatre, G/F, Block M, Queen Elizabeth Hospital, Kowloon	Miss Gloria CHEUNG Tel: 2527 8285 (Registration fee is required) 2 CME Points
4 SUN 2:00 pm	**HKMA Structured CME Programme Year 06/07 (XI)-Endocrinology & Neurosurgery** Organised by: The Hong Kong Medical Association & Queen Elizabeth Hospital Chairman: Dr. T.C. SHIH Speaker: Various # Lecture Theatre, G/F, Block M, Queen Elizabeth Hospital, Kowloon	Miss Nina HUNG Tel: 2861 1979 (Registration fee is required) 3 CME Points
5 MON 7:30 pm - 8:30 pm	**A Lady with Urinary Incontinence** Organised by: Hong Kong Urological Association Chairman: Dr CHU Sau Kwan Peggy Speaker: Dr KWOK Shan Chun # Seminar Room, G/F, Block A, Queen Elizabeth Hospital, Kowloon	Dr CHAN Kwok Keung Sammy / Ms Siddy MA Tel: 2958 6006 Fax: 2958 6076 1 CMP Point for College of Surgeons of Hong Kong
7:00 pm - 8:30 pm	**The Role of the Medical Expert in Personal Injury Litigation** Organised by: British Medical Association & The New Medico-Legal Society of Hong Kong Speaker: Various # 8/F, AON China Building, 29 Queen's Road Central, Hong Kong	Ms. Maseedis KAY Tel: 3420 6683 Email: maseedis.kay@sp.hk (Registration fee is required)
2:00 pm	**HKMA CME Luncheon Lecture on New Laws on Smoking Ban and Smoking Cessation Programme for Patients** Organised by: The Hong Kong Medical Association & Tobacco Control Officer, Department of Health Chairman: Dr. T.C. SHIH Speaker: Prof. LAM Tai Hing and Dr. WONG Wang Christine # Crystal Ballroom, Basement 3, Holiday Inn Golden Mile, Hong Kong	Miss Dorothy KWOK Tel: 2527 8452 (Pre-registration is required) 1 CME Point
6 TUE 8:00 pm	**HKMA Newsletter Editorial Meeting** Organised by: The Hong Kong Medical Association Chairman: Dr. H.H. TSE # HKMA Headquarter Office, 5/F., Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Tammy TAM Tel: 2527 8941
8 THU 2:00 pm	**HKMA Structured CME Programme with Hong Kong Sanatorium & Hospital Year 2007 (II) - Therapeutic Efficacy of Tomotherapy: The Hong Kong Sanatorium & Hospital Experience 2005-2006** Organised by: The Hong Kong Medical Association & Hong Kong Sanatorium & Hospital Chairman: Dr. B.L. WONG Speaker: Dr. TEO Man Lung Peter # HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road Central, Hong Kong	Miss Nina HUNG Tel: 2861 1979 (Registration fee is required) 1 CME Point
10 SAT 1:00 pm	**3rd HKMA Exercise Prescription Certificate Course (Module 11-14)** Organised by: The Hong Kong Medical Association, Department of Health, Physical Fitness Association of Hong Kong, China & Queen Elizabeth Hospital Chairman: Dr. Y.S. CHAN & Dr. C.F. YEUNG Speaker: Various # Lecture Theatre, G/F, Block M, Queen Elizabeth Hospital, Kowloon	Miss Gloria CHEUNG Tel: 2527 8285 (Registration fee is required) 2 CME Points
2:30 pm	**HKMA Refresher Course for Health Care Providers 2006/2007 (VI) - Common Skin Conditions and Their Imitators** Organised by: The Hong Kong Medical Association & Our Lady of Maryknoll Hospital Chairman: Dr. T.C. SHIH Speaker: Dr. CHONG Lai Yin # Our Lady of Maryknoll Hospital, 118 Shatin Pass Road, Wong Tai Sin, Kowloon	Ms. Clara TSANG Tel: 2354 2440 2 CME Points
(11)	**Cancer Imaging 2007 - Joint Meeting of the International Cancer Imaging Society & Hong Kong College of Radiologists** Organised by: International Cancer Imaging Society & Hong Kong College of Radiologists Course President: Ms. Lilian LEONG Speaker: Various Venue: Hong Kong Academy of Medicine Jockey Club Building, 99 Wong Chuk Hang Road, Aberdeen, Hong Kong	Mrs. Maureen WATTS Tel: 44 (0) 208 661 3420 Fax: 44 (0) 208 661 3901 E-mail: Maureen.Watts@icr.ac.uk Website:http://www.icimagingsociety.org.uk Ms. Diane LEE Tel: 2871 8788 Fax: 2554 0739 E-mail: enquiries@hkcr.org Website: http://www.hkcr.org
11 SUN	**2nd Certificate Course in Recent Medical Advances for General Practitioners** Jointly organised by the Family Medicine Unit, the University of Hong Kong and the Family Medicine Division, Hong Kong Sanatorium and Hospital Speakers: Various	Hospital Administration Department Tel: 2835 8800 Fax: 2835 8008 E-mail:hospadm@hksh.com, Website:http://www.hksh.com/CME.pdf
2:00 pm	**HKMA Structured CME Programme Year 06/07 (XI)-Cardiology** Organised by: The Hong Kong Medical Association & Kwong Wah Hospital Chairman: Dr. T.C. SHIH Speaker: Dr. LAM Tse Fun Cathy and Dr. TAM Chi Ming # Lecture Theatre, 10/F, Yu Chun Keung Memorial Medical Centre, Kwong Wah Hospital, Kowloon	Miss Nina HUNG Tel: 2861 1979 (Registration fee is required) 3 CME Points
14 WED 7:30 pm	**The 2007 WHO Classification For Brain Tumours: Some Key Points For Neurosurgeons** Organised by: Hong Kong Neurosurgical Society Chairman: Dr. Gilberto K.K. LEUNG Speaker: Prof. NG Ho Keung # Seminar Room, G/F, Block A, Queen Elizabeth Hospital, Kowloon	Dr. Y.C. PO Tel: 2990 3788 Fax: 2990 3789 2 CME Points
15 THU 1:00 pm	**Public Private Interface - Electronic Patient Record (PPI-ePR) Pilot Project Sharing Session** Organised by: The Hong Kong Medical Association & the Hospital Authority Chairman: Dr. C.P. HO Speaker: Various # HKMA Dr. Li Shu Pui Professional Education Centre, 2/F., Chinese Club Building, 21-22 Connaught Road Central, Hong Kong	Ms. Candy YUEN Tel: 2527 8285 1 CME Point

Meetings

27/03/2007	**Clinical Nurse Specialist Group Evening Forum - Managing Urinary Incontinence** Organised by: College of Nursing, Hong Kong Enquiry: Secretariat Tel: 2572 9255 Fax: 2838 6280
19-20/5/2007	**8th Regional Osteoporosis Conference** Organised by: The Osteoporosis Society of Hong Kong & The University of Hong Kong (The Osteoporosis Centre & Research Centre of Heart, Brain, Hormone & Health Aging) Chairman: Prof. Annie KUNG # 6/F, Old Wing, The Hong Kong Convention & Exhibition Center Enquiry: Ms. Cissy SOONG Tel: 2855 4353 Fax: 2855 1701
13-17/6/2007	**The 21st Congress of International Association of Paediatric Dentistry IAPD** Organised by: Hong Kong Society of Paediatric Dentistry # Hong Kong Convention & Exhibition Centre Enquiry: Mr. Daniel CHOK Tel: 2871 8896 Fax: 2871 8898 Email: info@iapd2007.com Website: http://www.iapd2007.com
12/7/2007	**The 1st Nursing Forum** Organised by: Department of Surgery, Li Ka Shing Faculty of Medicine, The University of Hong Kong; American College of Surgeons, Hong Kong Chapter & Nursing Studies, Li Ka Shing Faculty of Medicine, The University of Hong Kong # Underground Lecture Theatre, New Clinical Building, Queen Mary Hospital, Pokfulam, Hong Kong Enquiry: Forum Secretary Tel: 2855 4885 / 2855 4886 Fax: 2819 3416 Email: hksf@hkucc.hku.hk Website: http://www.hku.hk/surgery
12-14/7/2007	**The 50th Hong Kong Surgical Forum** Organised by: Department of Surgery, Li Ka Shing Faculty of Medicine, The University of Hong Kong & American College of Surgeons, Hong Kong Chapter # Underground Lecture Theatre, New Clinical Building, Queen Mary Hospital, Pokfulam, Hong Kong Enquiry: Forum Secretary Tel: 2855 4885 / 2855 4886 Fax: 2819 3416 Email: hksf@hkucc.hku.hk Website: http://www.hku.hk/surgery
17-18/11/2007	**Annual Scientific Meeting in Anaesthesiology 2007 - Expanding the Boundaries** Organised by: The Hong Kong College of Anaesthesiology & The Society of Anaesthetists of Hong Kong # Hong Kong Convention and Exhibition Centre Enquiry: CMPMedica Pacific Limited Tel: 2559 5888 Fax: 2559 6910 Email: meeting.hk@asia.cmpmedica.com Website: www.hkca.edu.hk/asm2007.htm

Courses

2,7,9,14,16,21,23,28,30/03/2007	**健康服務助理員訓練課程 (TC-HCA-0306)** Organised by: College of Nursing, Hong Kong Enquiry: Sugar Tel: 2572 9255 Fax: 2838 6280
14/3/2007 - 14/7/2007	**Professional Certificate in Clinic Operation** Organised by: Hong Kong Institute of Vocational Education - Sha Tin Speaker: Lecturers from Department of Applied Science, HKIVE(ST) # Hong Kong Institute of Vocational Education (Sha Tin Campus), 21 Yuen Wo Road, Sha Tin, New Territories Enquiry: Mr. YAU Yiu Shu & Ms. Joyce CHAN Tel: 2256 7114 Fax: 2256 7109 Email: joychan@vtc.edu.hk Website: http://stas.vtc.edu.hk or http://www.fmshk.org
18/3/2007, 22/4/2007 20/5/2007, 17/6/2007	**2nd Certificate Course in Recent Medical Advances for General Practitioners** Jointly organised by the Family Medicine Unit, the University of Hong Kong and the Family Medicine Division, Hong Kong Sanatorium and Hospital Speakers: Various, Enquiry: Hospital Administration Department Tel: 2835 8800, Fax: 2835 8008, E-mail:hospadm@hksh.com, Website: http://www.hksh.com/CME.pdf
18,19/5/2007	**IOF Osteoporosis Diagnosis Course** Organised by: The Osteoporosis Society of Hong Kong, The University of Hong Kong (The Osteoporosis Centre & Research Centre of Heart, Brain, Hormone and Healthy Aging) Chairman: Prof. Annie KUNG # 6/F, Old Wing, The Hong Kong Convention & Exhibition Centre Enquiry: Ms. Cissy SOONG Tel: 2855 4353 Fax: 2855 1701

Answer to Clinical Quiz

Answer :

1. This girl was suffering from tinea capitis, as shown by the patchy and scaly alopecia at the vertex. Skin scraping was positive for fungal hyphae and culture showed microsporum canis. Tinea capitis is uncommon in Hong Kong. It is seldom seen in adults here and more commonly occurs in children with pets at home.

2. The hair showed green fluorescence under Wood's light, a characteristic feature in tinea capitis caused by zoophilic dermatophytes. Wood's light examination is a quick and useful method for mass screening during an outbreak of tinea capitis in school.

3. Other causes include alopecia areata and trichotillomania (non-scarring and non-inflammatory); discoid lupus erythematosus and lichen planus (scarring and inflammatory).

4. Tinea capitis should be treated with oral antifungals, such as griseofulvin or terbinafine. The source should be traced and the pet should be treated as well. With zoophilic infection like microsporum canis, the child can normally be allowed to go to school as infectivity from human to human is small. But with anthropophilic species, which is actually predominant in tinea capitis, the infected child is better to be kept at home to prevent further spread.

Dr. Lai-yin Chong
MBBS(HK), FRCP(Lond, Edin, Glasg), FHKCP, FHKAM(Med)
Yaumatei Dermatology Clinic, Social Hygiene Service

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