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The Singapore Family Physician

Diabetes and the Family Physician

Tan CB

Diabetes is a common chronic condition seen by family physicians. With increasing affluence and changes in dietary habits, the prevalence of diabetes has increased from 4.7% in 1992[1] and to 9.0% in 1998[2]. Diabetes is a chronic condition with potentially serious disabling complications. It ranks among one of the top 10 leading causes of death in Singapore. It is a major risk factor for coronary artery disease, cerebrovascular disease and peripheral vascular disease, and one of the major cause of blindness, end stage renal failure and limb amputations in Singapore.

In the recent National Health Survey conducted in 1998, the prevalence rate of diabetes was 9.0%. Sixty-one percent who were found to be diabetic in the survey had not been previously diagnosed. Of those who were known diabetics, 53.2% had poor sugar control. The mean HbA1c among the known diabetics was 8.5%. Fifteen percent of survey population had impaired glucose tolerance.

This has significant implications for Family Physicians in Singapore. Firstly, being frontline healthcare providers, Family Physicians play an important role in the early detection of diabetes. Family Physicians must play a proactive role in the screening of at risk population for diabetes. This would include patients with a family history of diabetes, obese patients, those with previous history of gestational diabetes, patients with Syndrome X, patients with polycystic ovary syndrome and obese patients with acanthosis nigricans[3]. The American Diabetic Association also recommended screening for those aged about 45, hypertensive patients and those with dyslipidaemia (low HDL and/or high triglycerides)[4].

Secondly, those diagnosed to have diabetes should be treated rigorously. Lifestyle modifications, dietary control, weight management, cardiovascular risk management and drug therapy should be instituted to achieve euglycaemia. Diabetic complications should also be screened regularly. Blindness can be prevented if diabetic retinopathy is detected early. Early detection of microalbuminuria and the use of ACE inhibitors help to reduce the deterioration of nephropathy. The National Diabetes Commission had drawn up practice guidelines in 1993 for good management of diabetes and is due to publish a review of the practice guidelines this year.

Family Physicians are key players in the management of diabetes in Singapore. The majority of the diabetic patients in Singapore are being treated by Family Physicians. However, it is not possible for Family Physicians to manage the patients entirely on their own. The recruitment of other healthcare providers like nurses, dieticians, podiatrists as well as specialists in the care of the diabetic patients will help provide better care and hence reduce morbidity and mortality of our diabetic patients.

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Dr Tan Chee Beng
Honorary Editor

President's Column

A Chair or a Department of Family Medicine?

Lim LH

I would like to inform members about the developments that have taken place since the Extraordinary General Meeting (EGM) held on 24 September 1999, when the following three resolutions were passed by the House:

Resolution 1
That the College initiates the move to set up a Department of Family Medicine at the National University of Singapore

Resolution 2
That the College spearheads the fund raising towards a target sum of S$3million for the Endowment Fund for the Chair in Family Medicine.

Resolution 3
That a donation of at least S$250,000 be made by the College from its existing funds to the proposed Endowment Fund for the Chair in Family Medicine.

In pursuing the setting up of an independent Department of Family Medicine with a full time Chair within the Faculty of Medicine, the College's Executive Committee members have held formal and informal meetings and discussions with Professor Lim Pin, Vice-Chancellor, Professor S S Ratnam, Director, Graduate School of Medical Studies, A/Professor Tan Chorh Chuan, Dean, Faculty of Medicine and Professor Lee Hin Peng, Head, Department of Community, Occupational and Family Medicine.

Professor Lee Hin Peng has advised the College Council that he supports the setting up of a Chair in Family Medicine but that this Chair would be a Research Chair or even a visiting Chair which could be filled by a Family Medicine academic from overseas or locally who is recognised internationally for his research in Family Medicine, and who can oversee and direct Family Medicine Research in Singapore. Such research is lacking at the moment.

To go about this, Professor Lee has suggested that the initial sum of money required from the College to set up such a Research Chair is S$1 million, which will be used to start an Endowment Fund with the National University of Singapore's Bursar's Office. Once this minimum sum has been raised, a selection committee will be set up to make an appointment for the Chair.

Faced with this new knowledge, Council met to decide whether it wants to expend the same amount of time and effort to achieve what has effectively now become a part time Chair that could be filled by anyone the University chooses to appoint.

Thus, it is now clear to Council that the College is not in a position to set up a Department or a full time Chair in Family Medicine because we do not have a strong research base. Council is now of the view that we should set up a Research Center within the College premises.

However, the three resolutions passed at the Extraordinary General Meeting still stand, but will only be implemented at the appropriate time. Also, an alternative route should be sought to help set up the independent department with a full time Chair in Family Medicine. Council also agreed that the fund raising for this independent department and full time Chair should be started.

Now that a College Research Center would be set up within the College premises, funds will have to be raised for this project. The fund raising for the Chair and the independent Department of Family Medicine would be kept separate from that of the Research Center. Council has also appointed A/Professor Goh Lee Gan to be Director of the Research Center with Dr Kwan Yew Seng as his Deputy Director.

I will be keeping members informed of the progress made in these very exciting, yet important events in the College's milestones.

A/Professor Lim Lean Huat
President
17th Council (1999-2001)

17th Sreenivasan Oration

Medicine & Politics – Do They Mix?

Tan CB

In many countries, a politician commands the least respectability whereas a medical doctor, usually the highest. The politician is usually suspected of his intentions and he may also be viewed as power-hungry, dictatorial, dishonest, cunning and self-serving. Rarely is he associated with idealism or honesty. A doctor, on the other hand, is usually regarded as a respected, compassionate individual.

The topic today "Medicine and Politics – Do They Mix" set me thinking whether Singapore MP doctors have been effective parliamentarians. How he juggles between the two contrasting roles is what many have asked me time and again. The fact that the College of Family Physicians has asked me to make this oration on this subject must be testimony to my long stay in Parliament.

This is my 5th term in Parliament and by the turn of the century I would have served 20 years as a Member of Parliament for the constituency of Ayer Rajah. You must be wondering how I lasted so long. I have been criticised and praised for my very controversial views and stand on many issues which many of you must be aware of –amongst which are issues like streaming in our schools, restructured hospitals, nominated Members of Parliament and of course, foreign talent.

I must have held the dubious honour of having been taken to task by practically every Front Bench Minister. Do I feel hurt? Of course I get wounded and at times it gets very painful. I remember my first encounter in the House with Dr Goh Keng Swee DPM in 1980. I was a new MP then and it was my maiden speech. I criticised his education system accusing him of coming up with a policy of streaming which I felt would lead to a stratification of our society and dividing Singaporeans into class divisions. This was a serious allegation and Dr Goh went to the House the next day with a newspaper cutting of what I said and blasted me so hard that I sat there speechless and wounded. It was a baptism of fire and I woke up to the realisation that politics and medicine are two different worlds.

At the end of the day, why I stayed so long is because I believe the leadership knows I have no hidden agenda and that I reflected the ordinary Singaporean's feedback to the government. My presentation of such feedback has very often been direct, blunt and too frank and that has made the front bench members all worked up.

However, I feel politics has changed my life a great deal. I am more worldly-wise, more mature in my understanding and evaluation of things around me. I look at issues not purely from the view of an inward looking medical practitioner but from the more practical and helicopter view of a politician.

Having said all that, politics at times makes me mad and medicine keeps me sane. I enjoy seeing my patients every morning.

Let me now paint the two roles and let you judge whether the two can mix.

A doctor faces issues of life and death everyday. His decisions are usually instinctive based on well-honed skills. The results are usually apparent immediately or within days. But his influence is limited to almost a one-to-one situation.

A politician has to have a wider perspective of his wards. He has to take into account the societal context. He acts within the laws of governance and party policies. Politics embraces the whole of the social fabric and medicine is only a part of it, albeit a large concern of most people.

Doctors work hard and are not afraid of hard work. Politicians also work hard, especially in Singapore, and are also workaholics. That's where the similarity ends. The doctor works hard for the individual but the politician works hard for the masses. A doctor's training places the importance of the individual's needs, i.e. patients always come first. That is why it is not unusual for doctors to prescribe treatment to a patient without cost implication and affordability.

Dr Tan Cheng Bock
Member of Parliament
Ayer Rajah Constitutency
and Practising Family
Physician

A politician looks at health management in a different way, eg., when an order for an extra MRI machine is made by the doctor, the politician questions such a need when he sees that the same cost for this MRI machine could be put to better use in a health prevention program to serve more Singaporeans. But the doctor is often so captured by the need to better the treatment for a few selected patients that he sometimes fails to balance the needs of his patient with that of the nation's ability to pay for all. The politician looks at the total health care delivery system and wants to know the unit health cost. This is because he has a fixed budget to spend for use by many competing health institutions.

This does not mean the politician is not sympathetic to the doctor's need. He has to weigh this need with others. He has to decide for the majority and put aside his own personal feelings for the minority. It hurts at times but this has to be the way.

Doctors generally lack exposure to general management and financial accountability. His scientific training is very biased towards his patient's medical needs. He practises in much the same way as a shopkeeper. Even if he practises in an institution, he hardly plays a significant role in management. He is thus not an organisational man, unlike the politician in Singapore.

When I became a politician I had a conflict within me – an intra-personal conflict. My doctor's training, my compassion for people was tested and stretched to the limits.

I was torn between my compassion as a doctor and what I must do as a politican. My doctor's instinct tells me that the caning of illegal immigrants is not right and pushing away the boat people back into the open sea is a thought that I didn't want to entertain. However, my political instincts tell me that I must support these policies because if I don't, this island will be flooded with illegal immigrants and boat people. Our health, social, educational and security systems will be heavily taxed leading to a possible breakdown. So in the end, my individual prejudices must be put aside and the interest of our nation must come first. This intra-personal conflict is not easy to resolve. I guess it helps me to add a humane side to the politician's hard-headedness and firmness.

I shall now give you an insight into the work and expectation of an elected Member of Parliament. This can be categorised into within Parliament and outside Parliament.

Within Parliament, an MP is expected to attend all Parliament sittings unless he is overseas or ill and has obtained leave of absence from the Speaker. Parliament sits in the afternoon from 12.30pm to 4.30pm. This seldom happens as the meeting usually lasts way beyond 4.30pm. Parliament sits at least once a month and lasts one to three days except during the Budget and during the Presidential address debate when the sitting can last 2 weeks or so. Apart from the usual Parliament sitting there are select committee meetings and some of these meetings can stretch to as late as 11.30pm like the one I was involved in on Affordability of our Health System.

Then there are special meetings called by Government Parliamentary Committees and other Parliamentary groups. These meetings include dialogue sessions with overseas MPs. Also, there are state and official functions to attend.

As the leader of a group, like myself who chairs the Singapore-South East Asian Parliamentary group, I am expected to meet my Asian counterparts and lead delegations abroad.

Outside Parliament, there is the constituency work to attend to, like the weekly Meet-the-People's Sessions, grassroot meetings and functions including the 7th Moon Festival, Mooncake Festival, etc. You need to be away from home at least 3 nights per week.

Since the devolution of municipal matters to the MPs, we have had to organise and administer town councils. Recently, CDCs were created to enhance the development of community bonding.

As Chairman of the Bukit Timah Community Development Council, I have an added 6 constituencies to take care of and that excludes my town council work where I serve as Vice-Chairman. How effective you are at the ground

will be reflected by your performance at the general election. This is especially so if you are a single seat MP like myself. Group Representation MPs also need to work hard as you might bring down the votes of the group if you neglect your division.

In addition, there are non-parliamentary duties to perform. I am involved with the Land Transport Authority. I sit on the Executive Board planning Singapore's land transport. This takes away 2 of my afternoons every month. It is a government appointed job and not one by choice. It is of course an honour to sit on such a board. Therefore, you can see that it is not easy being an elected MP, it requires stamina and staying power and an ability to juggle many diverse roles.

So if you are also doctor-politician you have a practice to attend to, in addition to all the work as a Member of Parliament. How are you going to manage your practice, your parliamentary duties and very importantly, your family, is something you have to work out.

One of the impediments against doctors in politics is the inability to create time on his own. A lawyer can ask his client to see him later, a businessman can postpone a meeting but a doctor cannot tell his sick patient to come back later as he has to attend a meeting. So something must give way if a doctor is in politics.

I shall use myself to illustrate how a doctor-MP copes with the sudden change in his life style. The day I got elected I ceased being a private individual. You suddenly become public property and what you say and do are under the watchful eyes of the press and those who voted for you. You are torn between your loyalty to your patients and your responsibility as a parliamentarian. I did not have a night practice but those nights were quickly filled up by numerous grassroots and official engagements. Initially, I felt lost shuttling between my practice, my constituency and home. At one stage I was tempted to give up medicine and join the government but that would mean making politics my full time career. This conflict endured for a while but my love for medicine overrode politics. To me politics is a calling and medicine is my first love. As doctor-politician, I was faced with a dilemma. How I conduct myself and my practice will be watched by my medical colleagues. I had to avoid being accused of using my position to get patients. In Singapore, there are basically 2 types of practice i.e. contract and family practice. Contract practice involves getting contracts from companies to 'feed' the clinics and that means having to be close to company executives and CEOs and worse still, competing with your colleagues for such contracts. Invariably, if a doctor-politician gets such contracts he will be accused of being on an uneven playing field. It is true that many want to be close to a politician for whatever reasons and contracts could be given on such a basis. I decided from the very beginning that the better way out of this was to avoid contract practice and gave up what little contract practice I had and stuck to family practice. I also advise my patients from Ayer Rajah to see other doctors in case of any conflicts of interest.

With so many competing duties to perform, I had to make a swift decision. I decided to close one of my 3 clinics, took in a full time partner, and engaged 2 locum doctors to help me run my remaining 2 clinics. It was a relief though my income was halved but as I chose to take this path, I never regretted it. I wanted to do the job well and never counted the costs.

I went into politics with my eyes wide-opened at age 40 years because I had financial security and had developed a certain degree of maturity, independence and a better understanding of things around me at that age. I told myself: I do not want to die MBBS, that the world has more to offer than the 4 walls of my clinic and I wanted to see changes. I knew that if I cannot change the system from without, I have to see certain change from within. Today I am left with one clinic and a partner to help me and a very relaxed practice to contend with. I have reached a level of maturity that I am able to manage all my varied roles and prioritize my engagements. I have reached what I call a stable-state and have the confidence to take challenges better.

One of the most difficult adjustments to make in the beginning was my family life. My 2 children missed me a lot when I entered politics. My young

son missed me at bedtime as I used to read to him. He complained to his mother that I no longer tell him Tarzan stories. My advice to those who want to enter politics is you must make sure you have an understanding wife. My ability to give my best in politics is due to my wife who has to take care of the home and the education of my children in my absence. She has done well and my 2 children have grown up balanced and well adjusted. I am very proud of them.

Since 1980, the year I entered Parliament, 8 doctors were elected into the Chamber. There are now 3 elected MP doctors, one serving his 2nd term, one the 1st term and myself doing my 5th term. Judging by this brevity of their stay in Parliament (most stay only one term), the medical profession in politics has not been very successful.

Having said all that I hope I have not frightened away would-be politicians. You ask yourself, doctors come from the cream of the school cohort yet how many can be seen and counted upon in the hierarchy of society other than those in their own medical field. So few can be counted upon in terms of public services, in leadership roles in public and private sectors.

Why? My answer is doctors are too comfortable, they have status and financial security, they do not have much time and they are too inward looking in life.

In the beginning of my speech I said that in most countries, politicians are viewed with suspicion, disdain, etc. However, in Singapore, politicians are respected because of the high level of integrity and performance that the PAP demands of them. So a successful doctor-politician here, perhaps can claim to be almost at the top of scale of respectability. I hope this can be an inducement to those of you who have the enthusiasm and passion to contemplate joining politics.

Finally, if you do enter politics, my advice is be yourself and you cannot go wrong.

Thank you and good afternoon.

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Diagnosis and Classification of Diabetes Mellitus – A Brief Update

Tan KT

Summary

Keywords: **Diabetes Mellitus, Diagnosis, Classification, Oral Glucose Tolerance Test**

Introduction

Diabetes Mellitus is a common clinical problem seen often by the primary health care physician. While the diagnosis of overt diabetes is not often difficult, the exact criteria by which to diagnose and classify the disease can sometimes be confusing especially in the light of recent changes in guidelines. A brief update of these changes and their clinical application is presented.

Diagnostic Criteria (Old)

Although Diabetes Mellitus as a disease has been known and described for several centuries, widely accepted criteria for diagnosis and classification was only placed on a firm footing through the publications of the National Diabetes Data Group in America in 1979[1] and the World Health Organisation in 1980[2]. These two publications, although differing slightly in a few respects, remained the major source of reference for the diagnosis of this disease for many years. Minor modifications were made by WHO is 1985[3] but the main points were unchanged.

Basically, the diagnosis was based on a fasting plasma glucose of 140 mg% or higher and a 2 hour post-glucose reading of 200 mg% or higher. Subjects with classical symptoms of thirst, polyuria and weight loss could be diagnosed on the basis of a single abnormal reading but asymptomatic subjects required two separate readings or a formal 75g oral glucose tolerance test (GTT). The criteria for GTT were largely the same – again a fasting reading of 140 mg% or more being diabetic and 2 hour post glucose reading of 200 mg% or higher being diagnostic. The category of Impaired Glucose Tolerance (IGT) was for subjects with a normal fasting glucose level but a 2 hour reading of between 140 mg % to 200 mg% (Table 1)

Diagnostic Criteria (New)

In 1997, the Expert Committee from the American Diabetes Association (ADA) published their recommendations[4] and this was followed by a provisional report from a WHO consultation group in 1998 . Hopefully, the final report from WHO will be published within the next 12 months.

	Fasting Plasma Glucose	2 Hour post Glucose
Normal	Less than 140 mg% (7.8 mmol/l)	Less than 140 mg% (7.8 mmol/l)
IGT	Less than 140 mg% (7.8 mmol/l)	140-200 mg% (7.8 – 11.1 mmol/l)
Diabetes	140 mg% (7.8 mmol/l) or higher	200 mg% (11.1mmol/l) or higher

Table 1: Old Diagnostic Criteria for Diabetes Mellitus (WHO 1980)

One major change involves a lowering of the fasting plasma glucose level from 140 mg% to 126 mg%. The 2 hour mark remains unchanged at 200 mg%. The other change involves the introduction of a new category – called Impaired Fasting Glucose (IFG). This denotes a group where the fasting glucose level is near that of the diabetic level and may lead to diabetes later on in life. For IFG, the fasting blood glucose is between 110 mg% and 126 mg% (see Table 2).

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	Fasting	2 Hour Post Glucose
Normal	Less than 110 mg%	Less than 140 mg%
Impaired Fasting Glycaemia (IFG)	110 mg% to 125 mg%	
Impaired Glucose Tolerance (IGT)	Less than 126 mg%	140 mg% to 199 mg%
Diabetes	126 mg% or higher	200 mg% or higher

Table 2: New Diagnostic Criteria for Diabetes Mellitus (ADA 1997 & WHO 1998)

Rationale for the changes in Recommendation

There were a couple of major reasons for the change in criteria of diagnosis.

a. Studies have also shown that fasting level of 140 mg% represented a higher degreee of glucose intolerance than the post-prandial level of 200 mg%[6]. In other words, many individuals with fasting blood glucose under 140 mg% would have post glucose levels of greater than 200 mg%. It is estimated that the level of 126 mg% would be closer to representing similar levels of glucose intolerance.

b. The studies into the complications or diabetes mellitus in relation to the fasting blood glucose showed that the original criteria of 140mg% predicted the occurrence of microvascular complications but correlation with macrovascular complications was at a lower level of fasting plasma glucose[7]. It had been recognised for some time that patients classified as Impaired Glucose Tolerance test were already at higher risk for macrovascular complications. Since macrovascular complications play a major role in accounting for increased mortality in diabetic patients, it is clear that under the old criteria, these patients would not be classified as diabetic and would escape the attention they require to lower their risks for macrovascular complications.

Gestational Diabetes Mellitus (GDM) or Diabetes in pregnancy

The ADA had always adopted a different criteria from the WHO is the diagnosis of Gestational diabetes Mellitus (GDM). The 2 hour 100g GTT original studies by Sullivan and Mahan[8] remain the criteria of choice by the ADA while the WHO continued to adopt the 75g GTT. Here in Singapore we have tended to follow the WHO criteria. In the recent recommendations, both groups elected not to change the criteria for GDM.

New Classification of Diabetes Mellitus

There were a few main changes in the classification of categories of diabetes. The previous terms of non-insulin-dependent diabetes mellitus (NIDDM) and insulin-dependent diabetes mellitus (IDDM) were dropped as these tended to cause some confusion with insulin treated patients regarded as insulin dependent. Instead, type 1 and type 2 diabetes are the recommended terms. Type 1 denotes diabetes in which there is progressive beta cell destruction whereby there is ultimately severe insulin deficiency and requirement for insulin therapy for survival. Autoimmune type 1 diabetes constitute the majority of this category although it is recognised that there is a smaller idiopathic group of type 1 diabetes where there is no evidence of autoimmunity. Type 2 diabetes which is by far the largest category denotes the group characterised by insulin resistance and relative insulin deficiency. This group may also manifest

with various degrees of glucose intolerance ranging from mild glucose intolerance to severe hyperglycaemia requiring insulin for control and in some cases (although rarely) even ketoacidosis requiring insulin for survival.

The third category includes other types of diabetes – for example those caused by drugs, chemical, surgery or pancreatic diseases. The fourth and last category is Gestational Diabetes Mellitus which denotes a group where glucose intolerance is first recognised or diagnosed during pregnancy regardless of its actual etiology. Following delivery, glucose intolerance of these subjects would have to be re-evaluated.

Implications for Current Clinical Practice

1. **Diagnosis and Screening of Diabetes Mellitus** – the new guidelines suggest more reliance on the use of fasting blood glucose for the diagnosis of diabetes mellitus rather than oral Glucose Tolerance Test. It introduces a new category called Impaired Fasting Glycaemia (IFG). What are we to do with patients in this category? What is the likelihood of this category of patients developing diabetes mellitus later in life? These questions have not been fully answered. The WHO group recommended the retention of the older class called Impaired Glucose Tolerance (IGT). This, I think, is reasonable. The rate of conversion from IGT to overt diabetes has been worked out previously from many studies (average of about 3% per annum). To diagnose IGT would require a patient to undergo a formal 75 g oral Glucose Tolerance Test (GTT) which is rather cumbersome and not so pleasant for the patient. Until we gather more data on the long-term outcome of patients classified under IFG, we would probably have to rely on GTT to reclassify these persons and advise them accordingly if they have IGT.

2. **Progress of Diabetes from normal to IFG or IGT to overt Diabetes**. The new classification clearly recognises that diabetes is a progressive disease. Regardless of etiology (eg autoimmune or otherwise) diabetes starts with mild glucose intolerance and then progresses to more severe glucose intolerance requiring oral hypoglycemic agents and ultimately many would require insulin treatment for control or even for survival. The old method of using the terms insulin-dependent or non-insulin-dependent is clearly outdated and should be replaced. The recognition of this progressive nature of diabetes also suggests that the rate of progress may be retarded perhaps by early intervention or more aggressive treatment. Data is now coming on stream to support this and in the years to come would likely influence how we manage diabetes. For the present, the aim should be to control diabetes as well as possible from the outset so that the disease would hopefully not progress as quickly to more severe degrees of glucose intolerance.

3. **Type 1 or Type 2?** The recognition that both type 1 and type 2 diabetes may start off at a mild stage which would appear clinically as requiring only diet or perhaps only oral agents may cause difficulty in our attempts to diagnose whether the patient has type 1 or type 2 diabetes. The glucose level or the age per se would not help us to differentiate type 1 or from type 2 in say a patient of 30 years old. Only time would tell where type 1 would progress (usually) inexorably to total reduction of insulin production and insulin requirement for survival, whereas type 2 would usually progress more slowly and rarely reach dependence on insulin for survival. There is still no clear solution to this question. The use of auto-antibodies to identify patients who have autoimmune type (the majority of type 1 diabetes have an autoimmune origin) can only solve the problem partially. Some patients with type 1 diabetes have no autoimmune markers at all and this is especially so in the Asian population. The use of weight and family history can be helpful to an extent – type 2 being more strongly associated with familial occurrence and obesity whereas type 1 tends to occur in leaner subjects. Still, these are not clear cut criteria. The only way is to keep an open mind, follow-up the patient closely and see if the blood glucose level is well

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controlled. For these young diabetic patients, early introduction to insulin, if good control is not achieved in a few months, may be justified.

Conclusion

The introduction of new criteria for diagnosis and classification is welcomed. They represent another step forward in the understanding and management of diabetes. There are still grey areas that need more clarification. Hopefully, this will come over the next few years.

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Primary Prevention of Type 2 Diabetes Mellitus – Implications for Current Clinical Practice

Tan KT

Summary

Type 2 diabetes mellitus is a disease of increasing prevalence in Singapore. By the time overt diabetes is diagnosed, treatment is often difficult and in the long term good control is difficult to maintain. Animal models of diabetes demonstrate that diabetes appear to start with insulin resistance leading, in the presence of excessive nutrition, to hyperinsulinaemia and later hyperglycaemia and beta cell failure. This situation may be reversible by early intervention. The stage of Impaired Glucose Tolerance is identifiable by oral Glcuose Tolerance Test in susceptible individuals and intervention at this stage may prevent ultimate progression to diabetes. The Diabetes Prevention Programme is an ongoing randomised placebo controlled trial designed to examine the possibility of preventing the onset of diabetes by intensive lifestyle alteration or the introduction of oral Metformin. This trial will only end in 2002 but there are implications for our current clinical practice. By the time diabetes is diagnosed, very few patients will succeed in being controlled by diet alone. The early and aggressive control of blood glucose levels appears to be the most logical approach to preserve beta cell function. Screening for Impaired Glucose Tolerance in certain groups at risk of developing insulin resistance, IGT and diabetes should be seriously considered and if IGT is detected, they must be monitored and followed-up.

Keywords: **Diabetes Mellitus, Primary Prevention, Impaired Glucose Tolerance, Metformin**

Introduction

Diabetes Mellitus is a chronic illness, which is increasing in prevalence. In Singapore, population based surveys over the past 25 years have shown a progressive increase in prevalence, starting from 1.9%[1] in 1975, increasing to 4.7% in 1984[2], to 8.6% in 1992[3] and finally to 9.0% in 1998[4]. Clearly, Type 2 Diabetes Mellitus which is by far the more common variety seen here is of immense public health importance.

Although diabetes is a common illness, treatment of diabetes and prevention of its many devastating long-term complications have been difficult. Poorly or sub-optimally controlled hyperglycaemia is one of the most common clinical problems encountered in many primary health care clinics.

The difficulty in achieving and maintaining good glycaemic control is well demonstrated in the United Kingdom Prospective Diabetes Study (UKPDS)[5] . In the UKPDS, patients assigned to be treated intensively showed an initial fall in the HbA1c level but this later increased with time. The intensively treated group only managed a mean HbA1c of 7.0% which is above normal even though it is better than the conventionally treated group (HbA1c mean of 7.9%).

It appears that with time, diabetes is a progressive disease and with time, there is inexorable worsening of the disease regardless of the methods introduced to control it. The next logical step would be to examine whether the disease can be managed at an earlier or pre-diabetic stage. The possibility of the prevention of type 2 diabetes is therefore extremely attractive.

Pathogenesis of Type 2 Diabetes

It is worthwhile to briefly review the pathogenesis of type 2 diabetes as the understanding of pathogenesis would give us the underlying principles for our attempts at preventing this disease.

It has been well accepted that type 2 diabetes is the result of an interplay of genetic susceptibility and environmental factors. The genetic basis of the disease has been supported by twin and family studies. Type 2 diabetes has been demonstrated to have a very high concordance rate for

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monozygotic twins as compared to dizygotic twins underscoring the role of genetic factors[6]. There is also a high incidence of diabetes in children born to families where both parents have diabetes.

It has also been well accepted that hyperglycaemia in type 2 diabetes is the result of the twin problems of insulin resistance with relative insulin deficiency. The question is whether insulin resistance or islet cell dysfunction is the initial lesion.

One elegant animal model of diabetes that demonstrates the parallel that occurs in human beings is the dessert sand rat, *Psammomys obesus*.[7] It is actually not a rat but a gerbil living in the desert. The sand rat in the wild is not obese or hyperglycaemic but when they are fed laboratory rodent diet, they rapidly develop obesity, insulin resistance and then beta cell failure. They appear to go through four stages. In the initial stage (A), the animals are normoglycaemic and normoinsulinaemic. In stage B, the animals become hyperinsulinameic but still manage to maintain normal glycaemia. In stage C, the animals gain weight and become hyperinsulinaemic and hyperglycaemic. Insulin resistance is not preceded by obesity. Finally in stage D, the animals lose weight, glucose levels rise further and the insulin levels falls. Therefore, it appears that in these genetically predisposed animals which in the wild, do not have excess of food, diabetes does not occur. In the presence of abundance of food, hyperinsulinaemia occurs to compensate for insulin resistance until eventually hyperglycaemia ensues and subsequent beta bell failure occurs.

It appears that almost 40 years after its introduction, Neel's 'thrifty genotype' hypothesis is still relevant[8]. In type 2 diabetes the genetic predispostion leads initially to insulin resistance. In the presence of 'overnutrition', the beta cell hyperfunction leads to hyperinsulinaemia, which initally, is sufficient to maintain normoglycaemia but ultimately fails and hyperglycaemia or overt diabetes is the outcome.

It has been further demonstrated in the animal model above that the progression of this sequence of events may be averted by diet restriction. Animals in Stages B and C showed reversibility in hyperinsulinaemia and hyperglycaemia when transferred to a restricted diet. The parallel in human beings had been demonstrated eloquently in the Australian aborigines who showed improved glucose tolerance when transferred to their native surroundings and resumed their native lifestyle and diets[9]. The key is therefore early intervention before irreversible damage occurs.

Progression of Impaired Glucose Tolerance to Overt Diabetes

The stage of Impaired Glucose Tolerance as defined by the oral Glucose Tolerance Test has been recognised since the advent of the WHO criteria for diagnosis of diabetes[10]. Many studies have looked at the subsequent rates of progression from IGT to overt diabetes. The rates vary depending on the ethnic and clinical background and range from 2.3 per 100 patient years in the Japanese, 3 per 100 patient years for Caucasians and Mexican Americans, 4.7 for Nauruans, 4.0 for women with a history of previous gestational diabetes and between 10 to 11 for Asian Indians and Pima Indians. A figure often quoted is 3% per year for 10 years of follow-up.

Strategies for Intervention of Impaired Glucose Tolerance ranged from weight reduction and increased exercise to oral sulphonylureas and even surgery for gastric banding[11]. The results have been variable. The Da Qing Study reported that diet and exercise helped to reduce progression to diabetes[12]. Another study in Sweden showed that diet and physical activity helped to improve glucose tolerance[13]. However, a study in New Zealand over two years did not show any value of diet and exercise in improving fasting blood glucose[14].

One paper which showed the tantalizing possibility of prevention diabetes by oral Tolbutamide was published in 1980[15]. In the group treated with diet, about 30% developed diabetes after 10 years but in the group treated with tolbutamide, none became diabetic. The criteria of impaired glucose tolerance was different from that of the WHO because the cases were recruited prior to these criteria being published.

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Diabetes Prevention Programme (1996 –2002)

Some of the difficulties in intervention studies in impaired glucose tolerance (IGT) include the slow rate of progression to diabetes (which requires many years of follow-up), the difficulty in achieving and maintaining significant changes in lifestyle, the need to find an effective and safe agent for intervention and the large number of patients required to be recruited to ensure a definite answer to the question.

These difficulties may find their answer in a trial which is in progress at present – the Diabetes Prevention Programme[16]. This trial, which started in 1996, aims to recruit 3000 subjects with Impaired Glucose Tolerance based on 75 gram oral GTT. To ensure a reasonably high conversion rate from IGT to diabetes, the ethnic mix will be selected such that they can assume a 6.5% per year conversion to diabetes. This is important as previous studies may have lacked the ability to demonstrate the value of intervention because of a slow conversion rate.

Subjects would be assigned at random to one of four groups - conventional treatment (standard advice on diet and exercise), intensive lifestyle intervention, treatment with metformin and treatment with troglitazone (both placebo controlled). The last group was later removed (vide infra) and the trial is now proceeding with only three groups.

In the group assigned to intensive lifestyle intervention, the targets are to achieve and maintain a weight reduction of at least 7% of the initial body weight and to achieve and maintain a level of physical activity of at least 150 minutes per week of moderately intensive exercise (like walking or cycling).

The choice of drug intervention fell to Metformin and Troglitazones. Both are agents with the ability to reduce insulin resistance rather than to increase insulin production. Sulphonylureas were considered less suitable because of the possibility of hypoglycaemia which can be life-threatening.

Metformin is not associated with hypoglycaemia and has been demonstrated to reduce hepatic glucose production[17], to reduce hyperinsulinism and insuln resistance in polycystic ovary syndrome[18] and may have positive effect on blood pressure and lipid profile[19].

Troglitazone, which is member of the group of Thiazolidienediones (the 'glitazones'), was also selected for its ability to enhance tissue sensitivity to insulin. In clinical studies on polycystic ovary syndrome, IGT and type 2 diabetes, troglitazone lowers both fasting and post-prandial insulin levels[20, 21]. In a short-term study, subjects treated with troglitazone for three months were shown to convert from IGT to normal glucose toleracne in about 80%[22]. Unfortunately, in 1998, after 585 subjects were assigned to the troglitazone group, one participant treated with troglitazone developed hepatic failure and subsequently died inspite of a liver transplant. The troglitazone group was eliminated from the trial.

Implications for Current Clinical Practice

The Diabetes Prevention Programme will not be completed until 2002. In the meantime, are there clinical applications for us from the research into prevention of type 2 diabetes?

1. Approach to Screening for Diabetes Mellitus and follow-up of high risk groups

Population based surveys in Singapore and other countries have always shown a significant proportion of diabetic that are undiagnosed prior to screening and a further number are in the group of IGT. While the jury is still out on whether screening for diabetes should be carried out for the whole adult population, it would seem logical that a few high risk groups must already be strongly considered for screening.

The American Diabetes Association in its 1998 Position Statement recommended screening for type 2 diabetes for the following categories[23]

- Family history of diabetes (ie parents or siblings with diabetes)

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- Obesity (more than 20% over ideal body weight or BMI more than 27)
- Race/ Ethnicity (African-Americans, Hispanic Americans, Native Americans, Asian Americans, Pacific Islanders)
- Age 45 years or more
- Previous IGT or IFG
- Hypertension (140/90 mm Hg or higher)
- HDL Cholesterol 35 mg/dl or less and/or a triglyceride level 250 mg/dl or more
- History of GDM or delivery of babies over 9 pounds

In Singapore, we have yet to formally introduce guidelines for the screening of diabetes mellitus. From the National Health Survey (1998) we know that screening for diabetes would potentially diagnose the individuals with undiagnosed diabetes (about 5% of population) and those with IGT (15% of population). It may be premature to suggest screening of the whole population but certainly the following groups can be considered:

a. Individuals from families where both parents have diabetes
b. Severely obese individuals (BMI more than 30)
c. Individuals with previous gestational diabetes
d. Individuals with Syndrome X or Metabolic Syndrome
e. Individuals with Polycystic ovary syndrome
f. Individuals with acanthosis nigricans especially with concomitant obesity

As the prevention of type 2 diabetes gains firmer footing, this list is likely to become longer.

2. Approach to patients with diagnosed Impaired Glucose Tolerance

While the introduction of drug therapy for IGT must await the outcome of this trial, the use of lifestyle modification can certainly be adopted. It must be emphasised that the rates quoted for progression from IGT to overt diabetes are for instances where patients have been offered conventional dietary advice. What would the progression rate be if no advice or follow-up is given? Presumably it could be higher. The minimum that these patients should have would be advice on diet, weight reduction (where relevant), increase physical exercise and reduction of concomitant risk factors like smoking, hypertension and hyperlipidaemia.

3. Approach to the treatment of newly-diagnosed type 2 diabetes

We have discussed the attractiveness of early intervention in diabetes. The possibility of intervening at a point where insulin resistance is still reversible and where beta cell failure is not already advanced has been shown in animal models. By the time diabetes is diagnosed, many patients already have long-term complications, but until we screen more patients for diabetes, this would be their earliest contact with medical intervention.

If we are on the threshold of introducing drug therapy for IGT, is it still logical to use diet only for treating newly-diagnosed diabetes? I think we need to seriously question this age-old practice. While it is too early to say that all patients should embark on drug therapy, we must certainly be aware that on a longer term basis, practically all patients would require drug intervention in order to achieve euglycaemia which would include a normal level of HbA1c. We must accept nothing short of this.

We have seen that conventional dietary advice would still not totally prevent progression from IGT to diabetes. How much can conventional dietary advice alone prevent progression of diabetes to more severe diabetes? I do not undervalue the role of dietary control. Dietary control is still the cornerstone of management of type 2 diabetes. My argument is to question whether diet alone will suffice to bring about excellent control, which would be the best insurance against further beta cell loss and dysfunction. It is glucose toxicity from even mild degrees of hyperglycaemia that we want to prevent.

The risk of earlier drug intervention is obviously hypoglycaemia. I have long lost count of the number of patients who, on being introduced to drug therapy of newly-diagnosed diabetes, suffer a frightening episode of hypoglycaemia and for years later on become very reluctant to embark on drug therapy. Our patients are already mightily biased culturally against long-term drug therapy. At the point where diabetes is newly diagnosed, they are at their most receptive. Their receptiveness covers both diet and drug therapy. If dietary control is introduced, they are likely to be able to achieve good control for a short while (but would be unable to maintain it later). However, they would have learned that dietary control can bring about normal blood glucose (sometimes) and this discourages them from accepting drug therapy later (I could do it previously, I want to try again). They do not realise that the situation of glucose toxicity results in progression of disease and they often cannot achieve perfect control again with diet alone.

The choice of drug intervention, if this is to be considered, is therefore important. The biggest disadvantage of sulphonylureas is still its propensity to cause hypoglycaemia. While many of us do not believe in traditional herbal medicine, we must accept while they may do nothing for the hyperglycaemia, they certainly do not cause hypoglycaemia, which is a very visible and upsetting adverse effect for the patient and relatives. In this respect, metformin again is an attractive agent. I find it useful from the outset to get patients to accept that long-term drug therapy is almost unavoidable for diabetic patients. The main disadvantage of Metformin is its gastrointestinal side effects. All patients must be warned of the possible gastrointestinal side effect. If forewarned, patients accept these side effects better because they are much less frightening than hypoglycaemia. The alpha glucosidase inhibitors can also be used without fear of hypoglycaemia. However, they do not directly reduce insulin resistance and that is a distinct disadvantage. The gastrointestinal complaints from these agents are also as frequent as that from metformin.

The comments here refer to a fairly young (less than 60 years of age) patient with newly-diagnosed diabetes and no complications from diabetes. For elderly patients, who may have other concomitant disease, the consideration may be different.

My position is "Don't give diabetes a second chance!" Intervening at the point of diagnosis of overt diabetes is already a late stage. Any more pussy footing around will lose us invaluable time in the fight to preserve glucose tolerance.

Conclusion

Not long ago, I was asked by a family physician at a meeting whether I would treat a person with IGT using drugs like Metformin. I told him that before we can adopt that as clinical practice there must be conclusive evidence from trials such as the Diabetes Prevention Programme. He told me that if he himself was the person who had IGT, he might be tempted to try drug therapy.

Diabetes is certainly not one of the better diseases to be suffering from. I think that prevention is a real possibility and the results for the Diabetes Prevention Programme will hopefully translate hope to clinical practice.

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Diagnosis and Management of Familial Hypercholesterolaemia and Familial Combined Hyperlipidaemia

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The mean cholesterol level in the non-diabetic Singapore population was found to be 5.18 mmol/l in the 1992 National Health Survey[1]. The recent 1998 Survey showed that there was no significant reduction in mean cholesterol levels (5.5 mmol/l) in our population (unpublished). Most are due to dietary indiscretion and a sedentary lifestyle. However, there is a sub-population of subjects with high cholesterol because of an inherited lipid disorder. It is estimated that about 250 million persons in the world are exposed to a very high risk of dying at young age because they carry one or more genes that promote inherited lipid disorders. These disorders include Familial Hypercholesterolemia (FH, estimate 10 million) and Familial Combined Hyperlipidemia (FCH, estimate 40 million).

FH is a disorder of cholesterol metabolism with an autosomal dominant mode of inheritance, characterised by the presence of xanthomas and premature atherosclerosis[2]. Tendon xanthomas are pathognomonic of FH but are insensitive diagnostic markers. Hence diagnostic criteria should focus on LDL cholesterol. This disorder is caused by mutations in the gene that encodes the low-density lipoprotein (LDL) receptor, a protein that maintains cholesterol homeostasis. The frequency of heterozygote FH is estimated to be 1:500 in most populations, whilst homozygote FH is thought to be about 1:1,000,000. Homozygous FH are rare and may not be seen by General Practitioners as they are often under the care of Paediatricians and if they survive to adults, would be under the care of Lipidologist/Cardiologist. Heterozygote FH are more commonly encountered and would often be undiagnosed unless the attending physician is aware of such an entity. Diagnosis of FH can be made on clinical evidence and lipid panel without complex genetic analysis.

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The mean LDL cholesterol in such heterozygote FH is often above the 90th percentile of the age standardised population means. Demonstration of mutations in the LDL-receptor gene is confirmatory and useful but not mandatory. Clinically overt coronary artery disease (CAD) usually occurs at the mean age of 45-48 years in males and 55-58 in females. For details of lipid levels in the diagnosis of FH, please see Table 1.

FCH is also an autosomal dominant disorder caused, in all likelihood, by a combination of several genes. It is estimated to occur at a frequency of 1% to 2% (approximately 60,000) in Singapore. FCH subjects present with increased levels of apolipoprotein B containing lipoproteins, including very low density lipoprotein and low density lipoprotein. They frequently exhibit insulin resistance and small dense LDL particles. This aggregate of risk factors translates into premature atherosclerosis and it is not surprising that 10-20% of those with CAD under the age of 60 years have FCH. This lipid disorder is characterised by variability in lipid expression and some members express high triglyceride (TG), others have high cholesterol and the rest may exhibit mixed hyperlipidaemia. FCH is now believed to be inherited with multigenic mode of inheritance.[3]

Treatment of inherited lipid disorders

Modern medication such as the statins, can now normalise lipid levels in persons affected with these familial lipid disorders[4]. The results of recent large clinical trials have demonstrated that the use of such drugs can prevent early, fatal and non-fatal myocardial infarctions and stroke and can prolong high-quality life for many decades[5, 6]. In the absence of therapy to reduce LDL cholesterol, many patients with FH will develop premature atherosclerosis and this is likely to occur at a time when they are productive members of society. LDL cholesterol in most adult FH patients exceed 6.5 mmol/l (250 mg/dl) and even in the absence of other cardiovascular risk factors based on the NCEP and EAS guidelines, should be treated aggressively. The use of 3 particular statins, i.e. lovastatin, simvastatin and atorvastatin, over the approved therapeutic range of these

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Table 1: Cholesterol levels for the diagnosis of FH in mg/dl (mmol/l)

Age	40s	30s	20s	<18
New Cases				
Total Cholesterol	360 (9.3)	340 (8.8)	290 (7.5)	270 (7.0)
LDL cholesterol	260 (6.7)	240 (6.2)	210 (5.4)	200 (5.2)
Family members of known cases				
Total Cholesterol	300 (7.8)	280 (7.2)	240 (6.2)	220 (5.7)
LDL cholesterol	215 (5.6)	195 (5.0)	175 (4.5)	165 (4.3)

Table 2: Lipid levels for the diagnosis of FCH in Singaporean men and women (figures represent the 90th percentile of the Singapore population)

Total cholesterol mg/dl (mmol/l)	LDL cholesterol mg/dl (mmol/l)	Triglyceride mg/dl (mmol/l)
250 (6.6)	175 (4.6)	228 (2.6)

3 drugs (≤80 mg), can reduce LDL cholesterol by more than 40% in FH patients[7-9]. The LDL cholesterol target in FH patients without CAD or symptoms of CAD should be 3.4 to 4.1 mmol/l (130 to 155 mg/dl) whilst those with known CAD should be treated to target as in the NCEP guidelines (LDL <2.5 mmol/l or 100 mg/dl). Aggressive lipid lowering with statin therapy has been shown to be as effective but may require higher doses. Desirable LDL cholesterol levels in FH patients whose baseline values exceed 7.8 mmol/l (295 mg/dl), or in those patients with multiple risk factors or known CAD, are often not achieved with monotherapy. Combination therapy of bile acid sequestrant with statins may be safe and effective. In Severe heterozygote FH and homozygote FH, LDL apheresis maybe the therapy of choice either alone or in combination with statin [10-12].

Subjects with FCH may be started on either a fibrate or statin, depending on the predominant lipid disorder. In many instances, monotherapy may not be adequate in achieving desirable lipid levels. Combination of statins and fibrates are powerful and effective and local experience has shown it to be safe if used judiciously monitored appropriately. The incidence of myopathy during statin-fibrate combination has turned out to be less frequent than had been thought [13] and can be safely used in FCH patients. Other combinations include statin with nicotinic acid, with bile acid sequestrants.

Statin treatment in children and women with FH

There are no long-term studies on the safety of statin therapy in children with FH. The short-term data suggest that it is safe[14]. The current recommendation is to use resins in children and adolescents and restrict the use of statins to those aged 18 years and above. It is appropriate to exercise restraint in the use of drugs in young female patients during pregnancy, lactation and those who plan to get pregnant. Female heterozygote FH subjects with strong family history of premature atherosclerosis (myocardial infarction in females less than 60 years) may need drug therapy early.

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Unfortunately, recent surveys have also shown that the majority of persons affected with these treatable inherited lipid disorders are undiagnosed, untreated or poorly treated. Without identification and further intervention, most of them will die at ages of 35 to 65 years. Effective intervention through early diagnosis and durg therapy results in longer life expectancy as well as more productive lives without heart attacks. The MED-PED program, **M**ake **E**arly **D**iagnoses to **P**revent **E**arly **D**eaths, was officially launched during the 1999 National Week, in conjunction with the National Heart Association. MED PED is an international humanitarian program which collects data, provides education to the public and to health care providers and co-ordinates research in genetic disease. MED PED now includes local registries in over 31 countries including the Unites States, Britain, Ireland, Hungary, Israel, Canada and many others. Personnel from the National University Hospital and the Singapore General Hospital are involved in the programme comprising cardiologists, lipidologists and biochemists. Other doctors from both the private and public sector will eventually be recruited into the program. A local committee has been formed to decide on plans to oversee the implementation and maintenance of a registry for FH in Singapore and to organise educational activities. Members of the committee are already involved in the education of physicians on lipid disorders and have been using these opportunities to inform them about the programme.

The activities of MED PED in Singapore will be:

1. Facilitate the accurate diagnosis of FH and FCH in individuals with severe hypercholesterolaemia and premature coronary artery disease. Currently, genetic diagnosis is available in the National University Hospital and the Singapore General Hospital. Physicians familiar with the diagnostic criteria based on the family history and biochemical features can also make the diagnosis based on accepted guidelines.

2. Maintain a computer registry of cases that allows tracking of cardiovascular events as well as follow up.

3. Provide training and educational material for primary care physicians to build up a registry of physicians with experience in the treatment of FH and FCH. MED PED will not take over the care of patients but rather provide consultative support services for physicians involved in the care of patients with FH and FCH.

4. Assist primary care physicians in contacting relatives of index cases to encourage appropriate screening or treatment.

5. Work with government, lay or professional organisations (Singapore National Heart Association, Ministry of Health) to increase public awareness of this treatable disorder and to develop stable funding for the activities needed to find and help patients with FH and FCH.

6. Coordinate research into these disease conditions and promote collaboration with other MED PED centres internationally.

Why is treatment of FH and FCH different form polygenic hypercholesterolaemia?

The cornerstone of treatment of hyperlipidaemia is diet and lifestyle changes and this holds true

even for those individuals with FH and FCH. However, unlike the polygenic hypercholesterolaemia, the majority of individuals affected with inherited lipid disorder would need some form of drug therapy. Untreated, most would develop coronary artery disease by the fourth decade in males and the fifth decade in females. The dosage of statins needed to achieve desirable LDL levels in FH would generally be higher and often require expanded doses e.g. 40 to 80 mg of simvastatin and atrovastatin. In many instances, combination therapy would be required, even with the use of expanded dose statins. These combinations include statins with resin binders, nicotinic acid and its derivatives, and fibrates. In FCH, monotherapy is often inadequate and a substantial proportion may require combination therapy to achieve desirable lipid levels. The combination of statin with fibrate is believed to be safe and particularly useful in patients with FCH. Treatment in inherited lipid disorders would need to be lifelong, as cholesterol levels would invariably rise if drug treatment were ceased. Monitoring for side effects such as transaminitis and raised creatine kinase should be implemented as in the usual lipid management. Although treatment is expensive, studies have also shown that the cost of not treating such inherited dyslipidaemia with its consequent myocardial infarction and procedures is even more costly. A study from Harvard[15] has in fact shown that cholesterol reduction for primary prevention in FH is cost effective and carries similar benefit as the secondary prevention from the Scandinavian Simvastatin Survival Study[16].

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The Singapore Family Physician

Activities of Daily Living Skills (ADL) Aids for the Elderly

Venkataraman S

Providing care for the elderly requires a holistic approach to ensure each individual patient's needs are met. A vital aspect of rehabiliation also involves the introduction of adaptive aids to provide more independent function. Adaptive aids are an essential part of integrating the functional physical strength and skills present with the required aid or adaptive equipment prescribed. Practical tips suggested here are not to oversimplify the role of adaptive aids in providing for the elderly, but to serve as an introduction to the wide variety of adaptive aids to cater to each specific functional need of the elderly.

Caring for the elderly person requires the caregiver to be aware of all available sources of help. Part of this knowledge also entails the ability to identify areas requiring functional assistance and what these areas of deficiencies could be. The Family Physician plays a key role in helping the family to identify these functional difficulties and to recommend the appropriate use of these adaptive aids. The aged person should be encouraged to be an active participant in his/her daily care. Independence without utilizing adaptive aids is most desirable but many elderly patients may be unable to begin or complete even a simple task without the use of some aid.

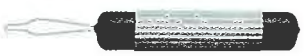
The following is a list of ADL aids which is available for the elderly to help them overcome some of their functional disabilities.

Wheelchair tracks: A simple 2 piece narrow ramp that is available in 6" or 7" width and portable. Primarily used for the outdoors and provides a non-skid and sturdy surface to manoeuver a wheelchair, for example, in or out of a van.

Wheelchair ramp: Portable and can be used both indoors or outdoors. Non-skid surface and safety tracks with 2.5" integral side guards and threshold plates are available. Can be a temporary or permanent fixture for easy wheelchair movement.

Jar opener: Can be wall mounted or fixed under a counter with mounting screws to provide convenient opening or closing of any type of capped jar or bottle cap.

Grip handle button hook: Facilitates manipulating of buttons during dressing task using one hand. Built up handle with surface ridges to provide adequate grip and tactile sense to skin surface.



Denture brush: 3" long denture brush secures to most surfaces for easy cleaning. 2 rubber suction cup feet provide a firm hold on counter while scrubbing.

Zipper pull: Clips to any zipper tab for easier grasping.

Long handled shoehorn: Enables individual to put on or remove his shoes without difficulty.

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Practice Tips in Caring for the Elderly

Sock aid with cord handle: Facilitates the donning or doffing of socks without slipping or requiring excessive bending over. The patient simply pulls the sock over the semi-plastic trough and manipulates the cord string upward to pull the sock.

Portable door knob turner: Durable, molded piece of plastic increases leverage to help turn rotating door knobs. The hook like shape is designed for using pull-type handles and even works on car doors. A rubber lining on the interior surface prevents the turner from slipping off doors.

Key turner: Provides extra leverage for turning keys. Curved built-up handles are easier to grip.

Transfer Board: Transfers safely and proves to be a sturdy surface to move from one place to another independently. The 3/8" base is tapered at both ends and laminated for easy sliding.

Work tray: Clear tray attaches to all types of wheelchairs with velcro straps. Tray measures 19" x 24" and is 1/4" thick.

Wall grab bars: Provides extra support and safety in bathroom/toilet. Grab bars are easy to install and can be tailored to the users' specific neds. Wide range of styles and sizes are available.

Long Handled shower hose: Enables independent showering while seated on the tub or commode seat. Built-up handle with textured grip surface for proper grasping is provided.

Tub seat: Thick high-density foam cushion for comfortable seating; suction foot pieces; water resistant and easy to clean. Backrest and side rail available for grasp for support. Lightweight frame and height adjustable buttons are present.

Bed pull-up: Enables the person sit up independently. The 40" long loop webbing attaches to the end of the bed or frame. A hook and loop strap keeps the buckle secure.

Light switch extension: Makes turning on and off light switches easy for the wheelchair bound or persons with limited reach.

Book butler: Provides a good holding surface and enables both hands to be free for page turning or wiring.

Giant Push button telephone adapter: Attaches to standard push button telephone with enclosed tape.

Rheumatic pen: Writing pen requiring no excessive pressive to grip the handle surface.

Practice Tips in Caring for the Elderly

Long handled reacher: 	Unique design that has flexible claws with round rubber grips to provide hold. Claws open to 3.5" wide and holds items of various shapes and sizes weighing up to 2 pounds. Ideal for one or two handed use.
Long handled comb: 	Reaching the back of the head or combing hair is simplified with this aid. The long handled comb adjusts to the length desired for easy combing. 8" handle is made from strong lightweight plastic.
Elastic Shoe laces:	Ideal for those with decreased hip flexion or dexterity. Lace the shoes in normal manner.
One handed cordless can opener:	Lightweight rechargeable can opener requires no pressure to operate. Easy to clean and easy storage.
Loop scissors: 	Provides sufficient grasp on loop for cutting. A self-opening spring strap handle is ideal for a weak grasp.
Pill bottle opener:	Opening presecription bottles with simple lid lifting techniques.
Milk carton opener:	Designed for impaired hand usage for opening cartons.
Milk carton holder:	Slip the carton holder for secure grip; uses one hand and is made of sturdy plastic.
Rocker knife: 	Provides easy cutting of meat/food with minimal pressure needed; large comforable angled handle
Weighted cup:	Designed for those with tremors or limited hand control; provides stability and decreases spillage with controlled liquid flow lid
Foodguard: 	The food guard attaches to the plate for easier scooping of food without spilling.
Dycem matt:	Non-slip surface material to prevent sliding of food utensils, for example, during meals
Long handled sponge	Facilitates independent bathing skills and enables reach to the back or toe areas

Conclusion

Some of these ADL aids may not appear to be very sophisticated but if used appropriately they allow the elderly person greater independence and this means a lot to the individual.

My fingers stiff and wrinkled
With a stooped posture I stand
My feet appear swollen and rigid to move
Grey and parched is my skin
But I still see, hear and taste
Painfully slow I may be
I still am - a human being

The Singapore Family Physician

Practical Tips on Swallowing Disorders for the Elderly

Viswam P

Swallowing disorders otherwise known as Dysphagia is fairly common in the following conditions:

- ✦ Neurological Disorder (e.g. CVA, Head Injury)
- ✦ Progressive Disorder (e.g. Parkinsons)
- ✦ Dementia
- ✦ Cervical Oesteophytes (when large bony spurs impinge on pharynx)
- ✦ Head & neck cancers

Some Common Signs of Dysphagia

- Excessive coughing before, during or after swallows.
- Excessive oromotor movements to get the bolus to move posteriorly, in an attempt to swallow.
- Decreased oromotor movements, as the person may have difficulty in chewing or have decreased tongue movement, or poor lip seal.
- Delayed swallows, whereby the person may have cleared bolus from oral cavity without having yet triggered a swallow reflex.
- Wet sounding or gurgly voice after swallows
- Unexplained spikes in temperature
- Weight loss due to decreased food intake (probably unable to tolerate the given consistency of food).
- Dehydration

General guidelines for safe swallow

- Identify "difficult" food consistencies and avoid it (e.g. thin liquids, grainy textures, mixed consistencies)
- Thicken liquids if necessary with thickener. Usually thicker liquids are better managed than thin liquids
- Proper positioning during feeding, whether in bed or on a chair.
  Sitting upright is the ideal position with the head slightly flexed.
  If in bed, the person should be propped up to at least 45°.
  A tube fed person should also be propped up to avoid reflux.

- For those persons who have an attention/ concentration deficit (e.g. after head injury), a quiet environment with fewer distractions (auditory and visual) may be more appropriate during feeding.
- Check and clear food if pocketed in the weaker cheek probably due to the decreased tone on one side.
- Solids should be avoided or cut up finely if chewing ability is decreased.
- Never feed a person who is not fully awake. Small frequent feeds may be better than a long session, where the individual is not able to maintain alertness.
- Modify food consistency whenever necessary and give in controlled manageable amounts.
- Watch for breathing - swallowing coordination especially for those who have apnea and dyspnea.

Sometimes such persons may have to be reminded to consciously hold his/her breath during swallows.

CHOKING AND ASPIRATION PNEUMONIA are two main problems which can be prevented in the elderly.

There could be silent aspirators, who show no outward signs of dysphagia but if suspected, further investigations could reveal the problem (e.g. a videofluroscopic study)

It is important also that caregiver learn the **HEIMLICH MANOUVER** to prevent choking in the elderly.

A speech-language therapist should be consulted if further advice on assessment and management is required.

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The Singapore Family Physician

Practical Tips on Recommending and Teaching the Elderly on the Use of Walking Aids

Seema S

The aim of prescribing walking aids for the elderly is to provide them with safety, security and function with the least expenditure of energy.

The main reasons for recommending walking aids for the elderly are as follows:

- balance problems due to medical conditions like recent CVA,
- pain due to age related musculoskeletal changes, e.g. osteoarthritis,
- fatigue due to cardiopulmonary causes
- functional immobility if the elderly has been bed ridden for a long period of time.

It is important to consider the following as part of the assessment:

- pre morbid status
- medical diagnosis
- social history
- architectural barriers
- elderly's ambulatory goals
- balance
- muscle strength
- cognition
- vision and hearing
- endurance

The choice of walking aid should be individualised according to the needs and requirements of the elderly.

The most common types of walking aids usually recommended are:

Walkers: Walkers are available in different types, ie. standard, folding, wheeled, etc. The use of walkers provide greater stability and support along with mobility. The advantage of walkers is that they are lightweight and adjustable. The folding frame which collapsible, is good for travel and can be stored easily. But the main disadvantages are that they are not practical on stairs and the normal reciprocal arm swing gait pattern is lost.

Canes: Canes are also available in various types like, T-cane, J-cane, quads cane. They are used to improve balance and stability. The advantages are they are lightweight, adjustable and parctical on stairs but the disadvantage is that because of a small base of support, they provide less stability as compared to walkers.

After a proper selection of walking aid has been done; teaching the elderly on the use of aids is an essential part and this entails the following:

Proper fit: the aid should be of proper height. For walking frame, the height is adjusted in standing position such that the wrist crease of the person corresponds with the hand grip of the walker.

For canes, the hand grip of the walking stick must correspond with the greater trochanter of femur.

It is essential to **DEMONSTRATE** to the person the gait pattern he/she must use.

Instructions given to them must be clear, simple and easily comprehended. They must be instructed on how to use the stairs, door knobs, and how to get in and out of a chair and bed.

Ambulation is a motor skill. Hence, it is essential that they **PRACTICE** it in order to reduce their fear and anxiety.

CAREGIVERS play a very important role in this teaching process as she will assist, guide or supervise the elderly while the individual is walking with the aid.

It is essential to ensure that the following points of **SAFETY** are reinforced

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The Singapore Family Physician

- Proper foot wear is an essential component, loose slippers are not advisable.

- Wear and tear: The walking aid should be regularly inspected for wear or tear eg, the rubber ferrules do not have cracks, the lock mechanism of the folding frame is secure.

- Hazards: The area where the elderly is supposed to walk must be clear of barriers like electric wires, loose carpets and rugs and make sure the floor is dry.

- Caregiver's position: The position of the caregiver is of utmost importance. The caregiver should always stand behind and to the slightly weak or affected side as they will be the guard in the event of fall.

Recommending and teaching the elderly how to use walking aids will ensure them of safety, security and stability and allow them greater independence.

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A Cardiac Emergency?

Tan HC

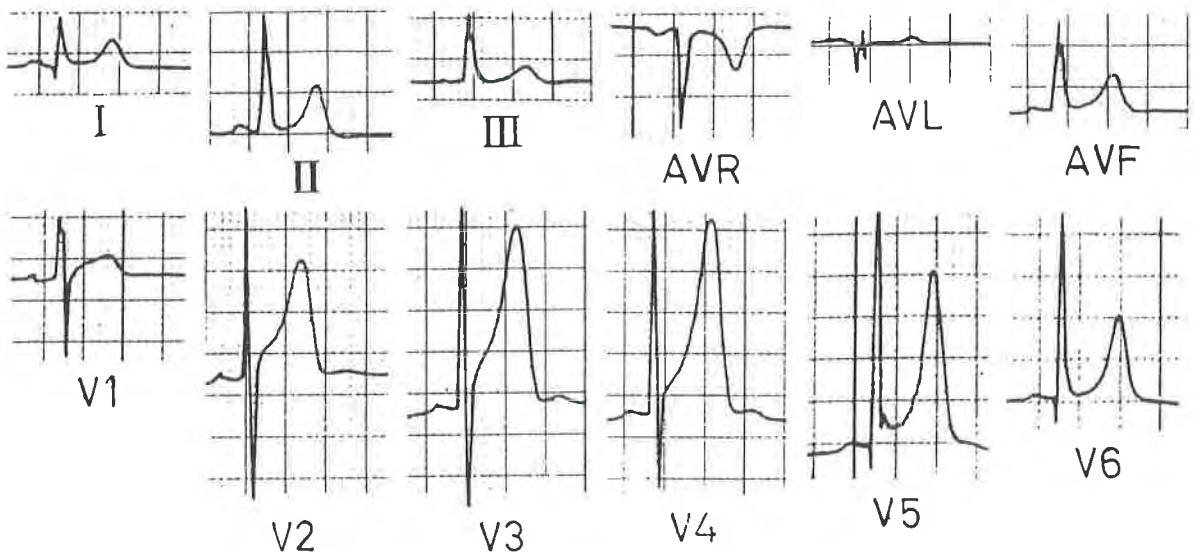
A 20-year old man came to see you for the problem of chest pain which you diagnosed to be musculo-skeletal in origin. However, to allay the fears of his mother accompanying him, you performed an electrocardiogram (Fig 1). To your surprise, you noted abnormal changes which at first glance resemble an "acute myocardial infarction".

As you examined the electrocardiogram in a systematic fashion, you noted that it might not be so ominous after all. The ECG showed sinus rhythm with a normal axis. The main 'abnormality' was an elevated ST-segment and tall T-wave in the precordial leads V2 to V4. The ST-segment assumed an upward concave morphology, and an elevated take-off from the J-point 4 mm above the isoelectric T-P line which was best seen in lead V4 and V5. The T wave is tall and asymmetrical with a gently sloped ascending limb and a sharp descending limb ending in a prominent U wave. You now recall that the described morphology belongs to Grusin pattern II normal variant, a form of early repolarisation pattern.

Early repolarisation syndrome or patern is commonly seen among young healthy males, in as much as 30% of the local population. The common differential diagnoses will be the hyperacute phase of acute myocardial infarction, acute pericarditis and hyperkalemia. Further recollection of the ECG changes in transmural myocardial infarction reminds you that while the ST-segments may also be elevated, there ought to be reciprocal changes in the leads overlying normal myocardium opposite the infarct site. Serial ECGs of an ongoing infarct will exhibit evolving changes such as the development of pathological Q-wave, convex ST-segment elevation and T-wave inversion. In acute pericarditis, the maximal elevation of ST-segments is in lead II, together with widespread ST-segment elevation in the precordial leads. The height of the T-wave is usually normal and PR-segment depression may be seen. In hyperkalemia, the T-wave is similarly tall compared to that of Grusin pattern II. However, the morphology is different in that the T-wave is symmetrical, slender and scooped inwards. The clinical situation should be considered during the interpretation of ECGs.

You can now heave a sigh of relief and reassure the patient and his mother that all is well and good. No further cardiac investigations or referral to a cardiologist is required.

Fig 1 Resting 12-lead Electrocardiogram (ECG)



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The author wishes to thank Prof Chia Boon Lock for his assistance

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A Housecall for a Psychiatric Emergency

Ng CLL

SUBJECT: **JMA, 21 year Malay male**
DATE: **7.00 PM 1995**

Introduction

J was well until his father passed away six months prior to the house call. Over that time of bereavement, he under went a change in personality and became more withdrawn. His relatives recounted that he was quite close to his late father, who at the time of death looked at him straight in the eyes before breathing his last breath.

J began to blame himself for his father's death and began talking to himself, laughing to himself and crying to himself. Occasionally, he would sing to himself as well. As time went by, J neglected self-care, refusing to bathe or shave. He held a job as storekeeper but refused to go work. One day, he presented a knife to his mother with both hands and asked her to end his life. On the day prior to the house-call, he asked his brother in law to use the "rotan" to cane him. J had not been sleeping for many many nights. He has a close knit Malay community who took turns to take care of him round the clock at home, believing that this was a passing phase.

I was called to do a house call when J became suddenly very aggressive and "gila" (Malay: insane).

Mental state examination

Upon arriving at the three-room Housing Estate apartment, I noted that he was of small build but had to be held down by seven male relatives. He was struggling and shouting incoherently. He said he sees ghosts.

A quick history as stated above was ascertained and BP was read as normal.

Provisional diagnosis was that of acute psychosis and depression.

He was given stat dose:
1. i/m Dormicum 10 mg
2. i/m Fluanxol 10 mg

Fifteen minutes later, he was still as restless and violent. He was given another dose of i/m Dormicum 5mg and an ambulance was called.

I waited until the ambulance officer arrived at the scene to take him to the National University Hospital since the family did not want the stigma of admission to the local mental institution, Woodbridge Hospital.

Inpatient progress:

J was difficult to manage in NUH and had to be tranferred to Woodbridge Hospital. His mental state stabilized after four weeks of medication and electro-convulsive therapy.

Outpatient progress:

He was discharged to my care after several months of follow up with the psychiatrist in the community psychiatric clinic.

Final diagnosis: Schizophrenia

His medication regime consisted of:
1. Artane 4 mg om
2. Carbamazepine 400 on
3. Thioridazine 100 mg om
4. Vitamin B complex ii/ii om
5. Vitamin C 1/1 om

He developed severe extra pyramidal side effects after i/m Clopixol 200 mg (protuding tongue) which had to be discontinued.

Concluding progress notes:

I am happy to report that J has not had a relapse for the past four years. He is gainfully employed at a supermarket working as a store hand and enjoys his working and family life. Looking at his disposition, one can hardly believe that this was the patient I saw five years ago one fateful night in 1995.

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Quiz

Test Your ECG Knowledge

Koo CC

42 year old female complained of increasing breathlessness for the last six months. However, she has no chest pain. Two weeks ago, she was referred to the ENT surgeon for hoarseness of voice. This revealed paralysis of her left vocal cord. Clinical examination revealed blood pressure of 130/80 mm Hg and a soft short mid diastolic murmur. Chest X-ray revealed cardiomegaly and pulmonary congestion.

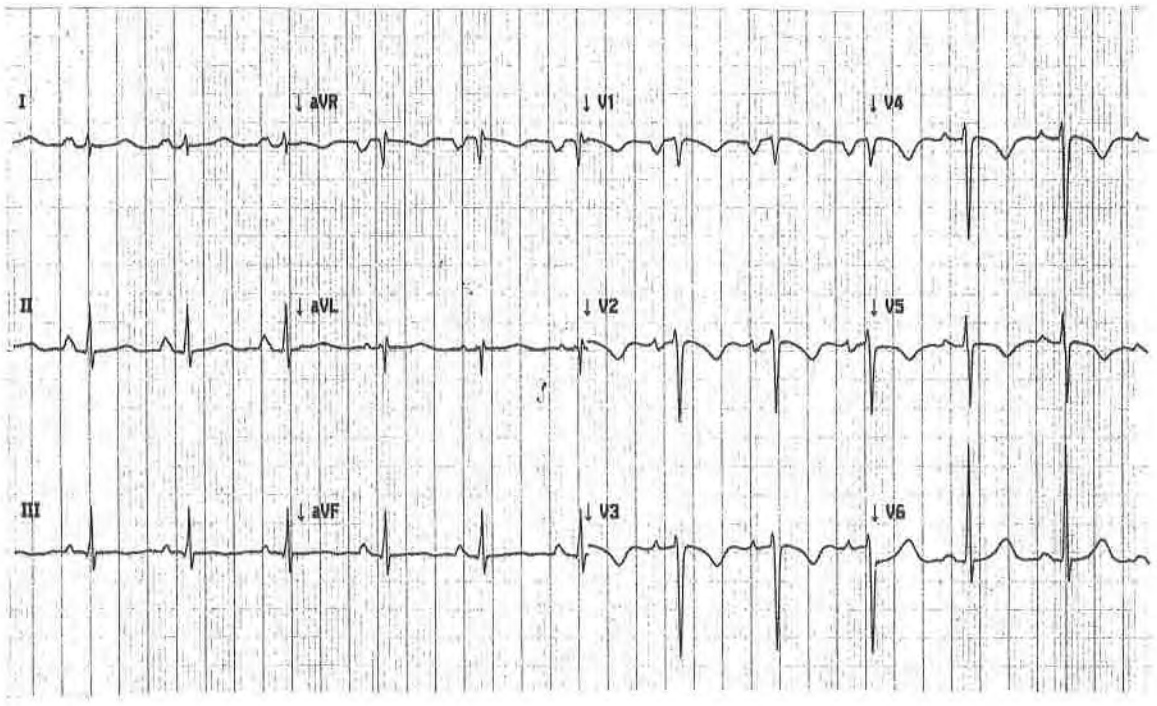
1. Comment on the ECG abnormalities.
2. What are your differential diagnoses?
3. What further investigation would you arrange to confirm your diagnosis?

Answer:

1. ECG revealed sinus rhythm with right axis deviation. There is symmetrical T-wave inversion from V1-5. In addition, there is poor R wave progression.

2. Differential diagnoses include *(i) viral myocarditis with congestive cardiomyopathy, (ii) rheumatic heart disease with congestive cardiac failure* and *(iii) ischaemic heart disease.*

3. Viral myocarditis complicated by heart failure is possible at this age. This can explain the abnormal ECG changes and X-ray signs of pulmonary congestion. Fever is not always present. However, the presence of a short mid diastolic murmur excludes this pathology. The differential diagnosis is rheumatic mitral stenosis complicated by heart failure. However, the short duration of the mid diastolic murmur excludes severe mitral stenosis. This cannot explain the degree of pulmonary congestion! Coronary artery disease is very unlikely in premenopausal women especially in the absence of coronary risk factors.



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4. As the patient was in cardiac failure, the only relevant test is the **echocardiogram**. This revealed normal cardiac valves including the mitral valve. However, it revealed ***a large echogenic mass*** involving nearly the whole of the left atrium! Part of this mass move across the mitral valve into the left ventricle during diastole. This is consistent with the diagnosis of **left atrial myxoma**.

She was referred to the cardiothoracic surgeon and had **surgical excision of a large left atrial myxoma**. Her peri-operative course was uneventful. Two weeks follow-up revealed no cardiac murmur and her chest X-ray, ECG and echocardiogram were normal.

Points to remember:

1. ***Breathlessness*** is a common complaint in our clinical practice. It is very important to elicit a detailed medical history. Do not treat such patients as "neurotic".

2. Go back to basic clinical examination. Listen hard for abnormal heart sounds including extra heart sounds and murmurs. Do not ignore clinical signs as this can narrow down your differential diagnosis. Her mid diastolic murmur indicates outflow obstruction across the mitral valve. Classically this is found in patients with rheumatic mitral stenosis. This is best heard on the left lateral posture using the bell portion of the stethoscope. As in this case, this is also heard in patients with large atrial myxoma. The left atrial mass impedes diastolic blood flow from the left atrium to the left ventricle. However, this is often soft and of short duration.

3. If the diagnosis is still in doubt after your clinical assessment and chest X-ray, send the patient for echocardiogram. As in this case, the echocardiogram is diagnostic and cost effective.

4. ***Atrial myxoma is rare*** and any cardiologist is considered "fortunate" to make such diagnosis in their clinical practice. Nevertheless, it is imperative to make this diagnosis, as this is curable!

Quiz

Test Your ECG Knowledge *(Corrigendum)*

Koo CC

A 42 year old female complained of palpitations. She was told that she had an "abnormal" ECG two years age when she had surgery for acoustic neuroma **(Figure 1)**.

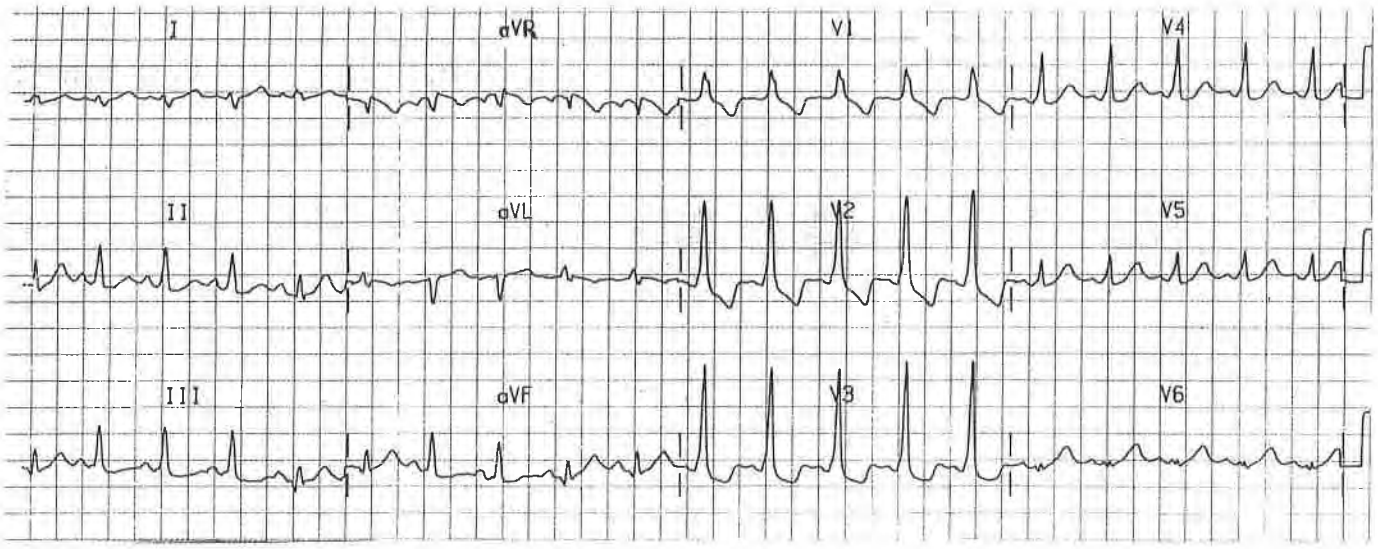
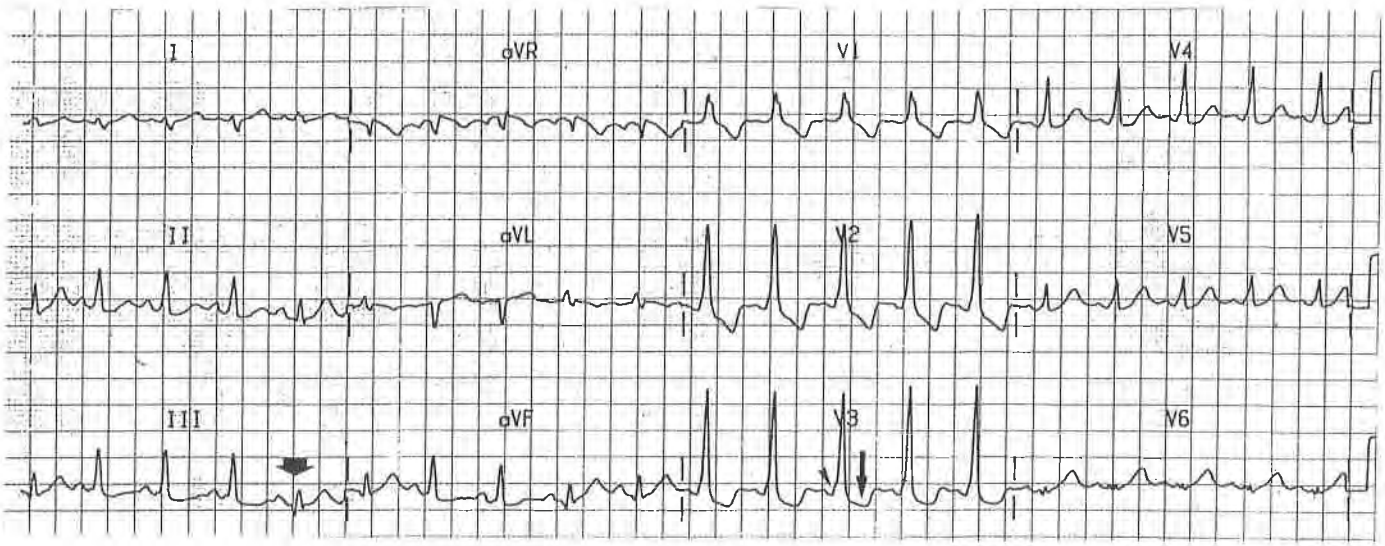


Figure 1

- What is the cardiac rhythm?
- List the ECG abnormalities.
- How would you further investigate this patient?
- What are the probable causes for her palpitations?

- The cardiac rhythm is *sinus tachycardia* at 120 bpm. Note the P waves before each QRS complexes **(figures 2)**.



- There are several ECG features of *pre-excitation* or *Wolff-Parkinson-White* ECG *abnormalities* (lead V3). Firstly the *PR interval is short* i.e. less 120 milliseconds (less than three small squares). Note, how the P wave merges with QRS complex. Secondly, the small *delta wave* (arrow◀) at the

onset of the QRS complexes. This is the result of early activation of the ventricle by the accessory pathway. Thirdly, the *QRS complexes are broaden* (more than 120 miliseconds) and the *ST segments are abnormal* with ST depression and T wave inversion (arrow ↓). The abnormal QRS complex is the result of early and abnormal ventricular pre-excitation by the accessory pathway (figure 3a). Remember the accessory pathway is an "extra" nerve that connects the atrium to the ventricle.

- Note the *normal QRS complex* i.e. the fifth QRS complex in lead III (broad arrow ➨). A normal PR interval and QRS complex follow the P wave. This indicates normal conduction via the AV nodal and His-Purkinje system (Figure 3b). Occasionally, patients with accessory pathways have intermittent antegrade conduction via its accessory pathway and at times via the normal AV nodal His-Purkinje system only.

- On further questioning, the patient has lost a few kilograms in weight, complained of tremors of extremities and has poor appetite.

- Her thyroid function is abnormal and is consistent with *thyrotoxicosis*. She was referred to the endocrinologist for further treatment.

- Remember, *to take a detailed medical history* before referring the patient for further investigations of Wolff-Parkinson-White ECG. Sometimes, the patient can have other causes of palpitations unrelated to the accessory pathway i.e. thyrotoxicosis.

- Patient with Wolff-Parkinson-White syndrome can have an abnormal ECG relating to early ventricular activation by the accessory pathway. This may be no more than a "cosmetic" effect without any pathological significance. They can lead a perfectly healthy and normal life without any troublesome arrhythmias. Furthermore, they do not require anti-arrhythmic drug therapy. On the other hand, the accessory pathway may predispose the patient to re-entrant tachyarrhythmias i.e. *supraventricular tachycardia* (SVT). Less commonly, these patients have *atrial fibrillation*. If the accessory pathway has short refrectory period i.e. able to conduct impulses from the atrium fast, the patient is likely to have atrial fibrillation with very fast very ventricular rate and risk of sudden cardiac death!

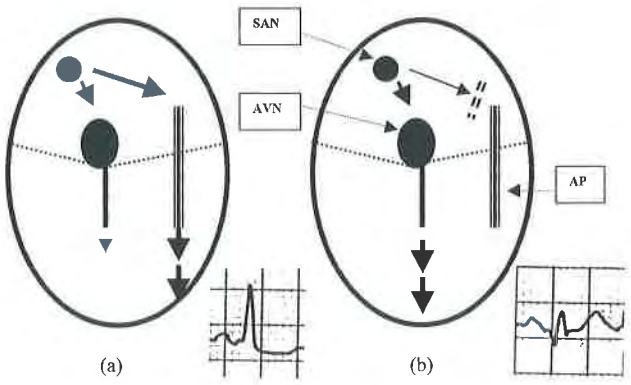


Figure 3: Diagram of sinoatrial impulse conduction via the AVN-His-Purkinje system and the accessory pathway. SAN: sinoatrial node, AVN: atrioventricular node, AP: accessory pathway. Figure 3a: antegrade conduction of the sinus impulse is faster via AP than AVN. Hence, the classical ECG pattern of pre-excitation. Figure 3b: antegrade conduction via the normal AVN system only. Hence, the normal PR interval and QRST complex.

Editor's Note:
Due to errors in the printed version of this article in Vol 25, No 1 1999, this article is printed again in Vol 25, No 3 1999. Our apologies to Dr Koo Chee Choong.

The Singapore Family Physician

Test Your Eye-Q (No. 8)
A Painful Red Eye in a Patient with Systemic Lupus Erythematosus

Au Eong KG, Yip CC***

A 32-year-old woman with a history of systemic lupus erythematosus for 3 years complained of severe pain and redness in her right eye for two weeks. There was no associated visual disturbance. She did not have any history of injury or surgery to her eye. Figure 1 shows her right eye looking downwards and inwards. The cornea, anterior chamber, crystalline lens and the posterior segment were normal in both eyes.

Questions

1. What does Figure 1 show?
2. What is the diagnosis?
3. What is the main differential diagnosis?
4. What systemic diseases are associated with this condition?
5. What is the treatment for this condition?

Answers

1. Figure 1 shows an inflamed right eye with tortuous and dilated conjunctival, episcleral and scleral vessels. The scleral vessels are large, deep vessels that cannot be moved with a cotton swab and do not blanch with instillation of topical 2.5% phenylephrine onto the eye. A small nodular swelling of the superotemporal part of the sclera is also present.

2. The patient has nodular anterior scleritis.

3. The main differential diagnosis is episcleritis. In *episcleritis*, the sclera is not inflamed and only the episcleral and conjunctival vessels are tortuous and dilated. Episcleral vessels are large and run in a radial direction beneath the conjunctiva. The inflamed episcleral and conjunctival vessels can be moved with a cotton swab and they blanch with topical phenylephrine. Pain, if present, is often mild in episcleritis. In contrast, pain in *scleritis* is severe, deep and often radiates to the ipsilateral side of the head or face. The sclera in *scleritis* may also have a bluish hue that is best seen in natural light by gross inspection. In *conjunctivitis*, only the conjunctival vessels are inflamed and both the bulbar and tarsal conjunctiva are often affected.

4. Fifty percent of patients with scleritis have an associated systemic disorder. The more common systemic associations include connective tissue disease (e.g. systemic lupus erythematosus, rheumatoid arthritis, Wegener's granulomatosis, relapsing polychondritis, polyarteritis nodosa, Reiter's syndrome, ankylosing spondylitis), herpes zoster ophthalmicus and syphilis. Scleritis may also occur following ocular surgery.

5. Treatment for scleritis includes systemic and topical corticosteroids and non-steroidal anti-inflammatory drugs (NSAIDs) such as flurbiprofen. In severe cases, immunosuppressive therapy (e.g. cyclophosphamide, methotrexate, cyclosporine, azathioprine) may be necessary.

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A Point of Digression

The Waterfall Series – Part I

Tan NC

Ton Nga Chang Waterfall

Speaking in Malay, Rahman, our Thai guide cum driver, predicted a crowd at the Ton Nga Chang waterfall as we drove out of Hatyai towards the countryside. This was not unexpected as it was Sunday. Located about 34 km west of Hatyai, this waterfall is a popular retreat for the local residents who wish to have a cool respite from the heat and humidity.

Rahman's prediction was correct. Many families gathered at the base of the waterfall, enjoying their picnics under the cool shade of the forest. The youngsters' chuckles and laughters vied with the rumbles of the waterfall whose water slid down the moss-laden boulders. A more powerful torrent juxtaposed itself at one corner. Many visitors enjoyed a cool immersion in the natural pool. A few stoics clammed their eyes tightly and stood unperturbed under the tumbling showers. Others had fun splashing water vigorously at one another.

Rahman led us to the next level of this multi-tiered waterfall. It is here where the name of this waterfall is derived. "Ton Nga Chang" means "elephant's tusks" in Thai. With a little imagination, the twin falls appeared to glide down the steep granite promontory like two tusks of an elephant (or perhaps a mammoth is more apt!). What impressed me were the trees that clinged tenaciously to the rocky islets in spite of the strong spray from the torrents. Here the powerful currents would deter any one from swimming. Instead, a rope suspended between two trees right in front of the waterfall tempted some restless youngsters to use it as a swing.

Rahman prompted us to view the "elephant tusks" from the top. Never did we expect the ascent to be a jolt to our jaded muscles. The stairway was a series of ill-defined steps consisting of gravel nestled in between the intertwining tree roots. With our minds rapidly planning the next move, we concentrated on putting a firm footing on each step, grabbing whatever nearby branch or vine for support. The leaves and mud from the recent rain occasionally deceived our perception, leading to a few slips. We were unable to match Rahman's stamina and had to stop every now and then to clear our fogged spectacles and to ventilate our lungs.

Our efforts paid off eventually as we were rewarded with an entralling view of yet another magnificent waterfall at the summit. A breeze created an aqueous gossamer that appeared to be blown off from the curtain of water. We discovered that we were not alone; a Thai couple was deeply engrossed in their conversation amidst the waterfall's aquatic symphony. What a perfect venue for a romantic rendezvous, if not for our intrusion!

Mae Ya Waterfall

My wife and I craned our necks out of the Suzuki wagon windows, looking for signs leading to the Mae Ya Waterfall. Our good friend, How Hong, too kept a look-out whilst steering carefully on the winding road. At a tiny town on the periphery of the Doi Ithanon National Park earlier on, a

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"kopitiam" vendor whom we bought our fried rice lunch, was very confident when she pointed out the direction of the waterfall. Despite the language barrier, she appeared to understand when we mentioned "Nam Tok Mae Ya" and the way she gestured was rather reassuring. Never did we expect to encounter a bifurcation at a road junction barely fifteen minutes after we thanked and bid good-bye to the helpful vendor. The road sign was in Thai alphabets and there was nobody in sight. After a short deliberation, we chose the left path. Afterall, we had fifty percent of being right!

The next half hour, we were skirting the hilly terrain within the vast national park. Still, there was no sign that we were heading in the right direction. The surrounding deciduous forests, with foliage in rusty blotches due to the cool dry season in January, appeared endless.

Our hearts began to sink, infiltrated gradually by the sap of regrets. Perhaps we should have chosen the road on the right .....perhaps we should have hired a Thai driver instead of opting for a 4-wheel self-drive. The previous three days, a local driver fetched us straight to the many waterfalls in the hills betweeen Chiangmai and Mae Hong Son. Even the tumultous journey to the Phua Suak Fall was completed without any hassle in the hands of the experienced Thai driver. Now on a good bitumen road, we seemed lost. We were contemplating a "belakang pusing" when How Hong spotted a white sliver slicing down two distant hilllocks above the forest canopy. My immediate intuition was the Mae Ya waterfall. The guide-book which I regarded like a bible throughout this trip, noted that this waterfall was amongst the highest in Thailand, falling from a height of 250 meters. The sense of uneasiness was rapidly replaced by one of relief and anticipation.

Another twenty minutes later, we arrived at the foot of the Mae Ya waterfall. How Hong parked the wagon in front of a long row of hawker stalls. The air was thick with the aroma of barbecued chicken and meat kebabs, which were popular with the Thais. I was a little apprehensive of the close commercial congregation to the waterfall, with fears of pollution. My worry was fortunately unfounded. The environs were kept clean, thanks to the close scrutiny of the park officers.

An open-air platform allowed a panoramic vista of the waterfall. Looking up the Mae Ya Fall from this platform to the Prussian blue sky above, it resembled a tapering colossal amphitheatre ingeniously sculptured by nature. One could easily mistake it for a grand stairway leading to the heavenly residence of the deity, guarded by a lonely tree deeply rooted at the 'entrance'. The "pathway" to heaven had never been straightforward. The waterfall was indeed an intricate and bewildering network of rocky steps.

Several visitors were seen scurrying up the rocky steps to get closer photo shots of the waterfall but none attempted to scale this maze-like stairway. Mere mortals like us on the platform were more than contented to capture this splendour on film. It proved to be a challenge as my 35mm camera was unable to accomodate the magnitude of the waterfall.

A group of Thai undergraduates from the University of Chiangmai shared our sentiments. They approached How Hong to take their group photo. Keen to practice their spoken English, they enquired about our trip, which we filled them with details. We expressed keen interests in their education system, as we planned to visit their campus later. It did not take long before we too joined in their group photo.

Sai Yok Waterfalls

The platform creaked as we tried to catch up with the Thai lad who was showing us the various rooms on the boathouse. Forming a central vein linking the various boathouses and barges moored by the Kwai Noi River, it was wet after the rain.

Through the numerous gaps in between the planks, we caught glimpses of the swift currents beneath, accelerated by the downpour earlier.

Chwee Bee and I had arrived at the Sai Yok National Park late in the afternoon, partly due to the rain. We were concerned about finding accomodation at the park at this hour. As we tread cautiously on the platform, we peered into the dark empty rooms as the doors were left open. The boathouses were virtually deserted. The rainy season in mid August could have kept the locals away from the park. We settled for the last room right at the end. The choice was obvious; it was a room with a view. The Sai Yok Lek waterfall was visible from the side window and the sound of the splashing water provided continuous piped-in music. Apart from that, the room was sparsely furnished with two beds and an old table. The attached bathroom was nothing more than four bamboo partitions with a conspicuous cavity at one corner. Being used to instant lighting at the flick of the switch in Singapore, it took us a while to become accustomed to the oil-lamp whose flame cast our faltering shadows on to the bare walls.

The rains rejuvenated the waterfalls with renewed vigour and vitality. Barely ten meters away from our room, the torrents of the Sai Yok Lek waterfall were swollen by the rain. A limestone promontory bifurcated the waterfall into two before it plunged into the tawny Kwai Noi River. The twin falls churned up mini whirlpools in the river as the latter made a perpendicular right turn further downstream.

The same night, the moon turned up to grace the occasion of our visit. It highlighted the waterfall with moonbeams and added glitter to the foamy water. Even the cicadas teamed up to serenade in the moonlight. It was a pity that there were only two spectators to soak up this wonderful sight.

The next morning, we made our way to the more renowned member of the Sai Yok waterfalls, the Sai Yok Yai Fall. The word "Sai" referred to the fig tree in Pali language and "Yok" was "rock" in Thai. "Sai Yok" is supposed to derive its name from the giant fig tree that had fallen into the Kwai Noi River more than hundred years ago. Today the legendary fig tree is long forgotten. Instead, the local residents vividly remember the song, "Khmer Sai Yok" written by Prince Narisranuvativong in praise of the waterfall's loveliness and tranquility. The waterfall also received another eminent guest, the revered King Chulalongkorn (RamaV) who travelled all the way from his royal palace in Bangkok to bathe in the cascades. A plaque of his majesty was erected near the waterfall to commemorate his visit.

Long before these royal visitors, the area was inhabited by our primitive ancestors, as evidenced by excavated skeletal remains and stone tools dating back to the Paleolithic or old stone age (40 000B.C.) in the park. It seemed our forebears too were smart to choose this serene waterfall as their abode.

A canopy of lush foliage crowned the Sai Yok Yai waterfall. The water split into threads as it fell over the intricate stack of mammoth cobblestones. From afar, the waterfall appeared to be draped in a lacey apparel.

We hired a long tail boat and requested the boatman to station the vessel right in front of the waterfall, a small price to pay for such a magnificent view. Another option would be to view the waterfall from the opposite shore after crossing the suspension bridge.

In the afternoon, the repressive heat and humidity forced us to seek respite in the natural pools on the upstream of the Sai Yok Yai waterfall. The water flowing down from the Bilauktaung and Tesserim mountains was exceptionally clear and refreshing. The sunrays that percolated through the foliage lit up the pools in various shades of green. Little wonder that it was once a favourite retreat for the royals.

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