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The SINGAPORE FAMILY PHYSICIAN

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Vol. XVIII NO. 3
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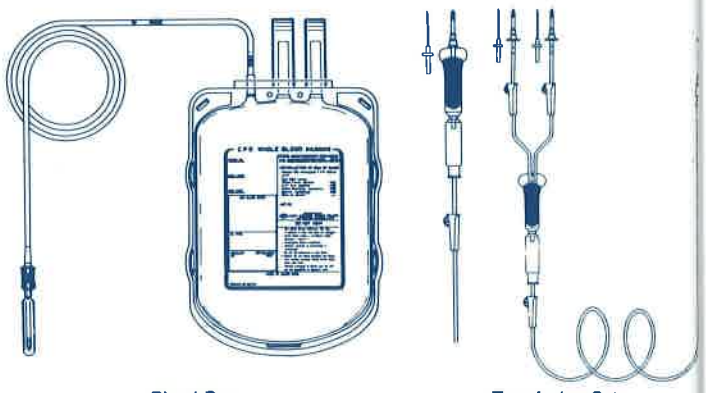

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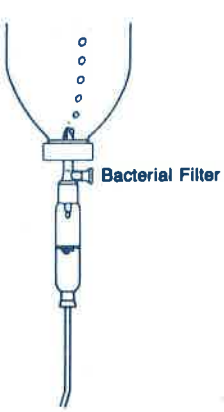

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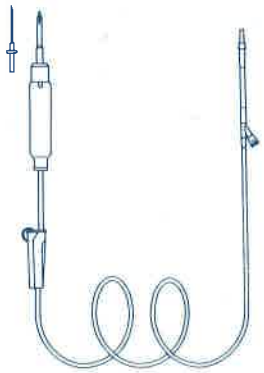

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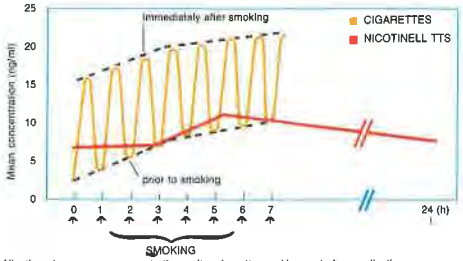

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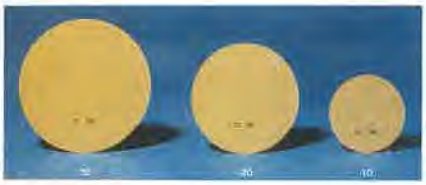

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BE MALARIA WISE

INTRODUCTION

People still die of malaria (falciparum malaria) because the doctor has missed the diagnosis. Part of the reason is that many doctors do not consider the diagnosis of malaria unless the symptoms – teeth chattering, chills, shakes and high fever (what used to be called "the ague") – occur in cycles. This is a fallacy.

DECEPTIVE

The picture of typical symptoms is true for P vivax, malariae and ovale – malaria types that are rarely fatal. What may not be known and is not stated clearly in many standard textbooks is that falciparum malaria – which is quickly fatal if left untreated – often does not behave in this way: all that the patient brings along is a severe headache and a nondescript fever (which may be mild and not accompanied by shakes)[1].

Also, he or she may appear relatively well when he or she is, in fact, seriously ill. This is another deception. A parasite rate of 5% of erythrocytes indicates a serious infection. When this reaches 10%, the fatality rate of non-immune patients (those who have not been exposed to falciparum malaria infection before) is over 50% in spite of treatment[2].

SUSPICION

The only way that deaths from falciparum malaria can be avoided is to be malaria wise. A high index of suspicion and a working knowledge of malaria prevention, detection and management are essential. The increase of drug resistant malaria in many parts of the world means that no drug regimen currently available guarantees complete protection against malaria. Some countries with both urban and rural malaria may not have any malaria in major cities most frequently visited by tourists. Kenya, for example has no malaria in Nairobi, and Brazil has none in Rio de Janeiro. However, unknown to their doctors, many patients do wander off the beaten track into jungles and remote places. Unless a thorough travel history is taken, such information may be left undisclosed.

Chloroquine resistance has been reported in all areas where malaria occurs, except for Central America west of th Panama Canal Zone, Mexico, Haiti, the Dominican Republic, and most of the Mideast (including Egypt).

ANTI-MALARIA STRATEGY

There are two main aspects in the anti-malaria strategy namely, Prevention; Detection and Treatment. A framework of the 3As of prevention and the 3Ts of detection and management of malaria described by Bradley[3] in a symposium on malaria held in 1989 jointly by the London School of Tropical Medicine and Hygiene and the Royal Society of Medicine in the United Kingdom is practical and easy to remember. This framework is described in this editorial.

PREVENTION: *AWARENESS*

The first A in prevention stands for awareness. This applies not to just doctors but also the patient. Both should be aware of the possibility that if the patient has a fever, it might be malaria. The patient should be educated to remind the doctor of the possibility of malaria and volunteer a detailed travel history.

PREVENTION: *AVOIDANCE*

The second A in prevention is avoidance. This is not avoidance of travel but avoidance of being bitten by mosquitoes. With increasing problems over chemotherapy and drug resistance, methods

of avoiding mosquito bites become important. There is a need actually to spend tome with the patient who is travelling to get this vital point across. Rolling shirt sleeves down at dusk, use of mosquito repellants and use of mosquito nets are important tasks.

PREVENTION: *ANTIMALARIALS*

The third A in prevention is of course, the antimalarials. The decision whether it is better to use routine chemoprophylaxis or to depend on avoidance measures alone and getting oneself treated if one becomes sick depends on the combination of two factors: transmission (the chance of being bitten by an infected mosquito) and chloroquine resistance. There are four possibilities.

(a) Low transmission and no chloroquine resistance:
A choice can be made between no prophylaxis or prophylaxis which will be effective, easy and straightforward.

(b) High transmission and no chloroquine resistance:
Antimalarials should be taken, and they will be highly effective. The problem is that these areas are becoming smaller.

(c) Low transmission and chloroquine resistance is common:
An example of such areas is most of Thailand. The current thinking is towards people taking a reserve treatment drug with them – e.g., mefloquine - 15 mg/kg (maximum 1 gm or 4 tablets) taken as one dose – and perhaps not using chemoprophylaxis. If it is decided that the patient is knowledgeable enough, such a strategy could be carefully explained to him and in particular what action is expected of him when he has a fever. The other alternative is to put him on a prophylactic drug that will be useful in a chloroquine resistant area; examples are mefloquine 250mg once a week or doxycycline 100mg every day.

(d) High transmission and high chloroquine resistance:
An example of such areas is East Africa. The options are either to take nothing and relying on getting treated if one becomes sick – as described in (c) – or to take an appropriate chemoprophylactic and seek medical attention if one still becomes sick.

With regard to choice of chemoprophylaxis, it is important to note that this is a constantly changing picture and regular updating is necessary. Fansidar is now of doubtful use in chloroquine resistant areas of Thailand, for instance.

DETECTION AND TREATMENT: *THINK* MALARIA

Thinking of the possibility of malaria is the first T in detection and treatment. In every febrile patient, a detailed travel history should be obtained to rule out malaria as the cause of the symptom.

DETECTION AND TREATMENT: *TEST* FOR MALARIA

The second T in detection and treatment is testing for malaria. When in doubt it is wiser to test for malaria. It is important to remember that false negative results commonly occur, particularly if the patient is partially treated or on some chemoprophylaxis. Repeated tests may need to be done. Also, this should not be left to the next day: a person suspected of malaria should be treated as an emergency. This disease does not forgive the complacent doctor.

DETECTION AND TREATMENT: *TREAT* FOR MALARIA

The third T is to treat early and treat correctly. This will require the patient to be admitted if the possibility of falciparum malaria exists.

EDUCATION

Finally, it is important to reiterate that health education is all important. It must reach out not only to practising doctors but also practising pharmacists and of course, the patient. Practising pharmacists are an important group because often they sell antimalarial drugs for prophylaxis di-

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rectly over the counter. Upon their shoulders must rest the responsibility of educating the patient on how to use the drugs effectively, to be impressed that antimalarials do not provide cast iron protection, to remember to visit his doctor if unwell on return and to give a complete travel history to the attending doctor. Together, we should be malaria wise.

References

1. Nicholas White. "Watch the symptoms", World Health, Sep-Oct, 1991: 8-9
2. Bruce-Chwatt. Chemotherapy of Malaria. 2nd ed revised. Geneva: WHO, 1986, 121.

GLG

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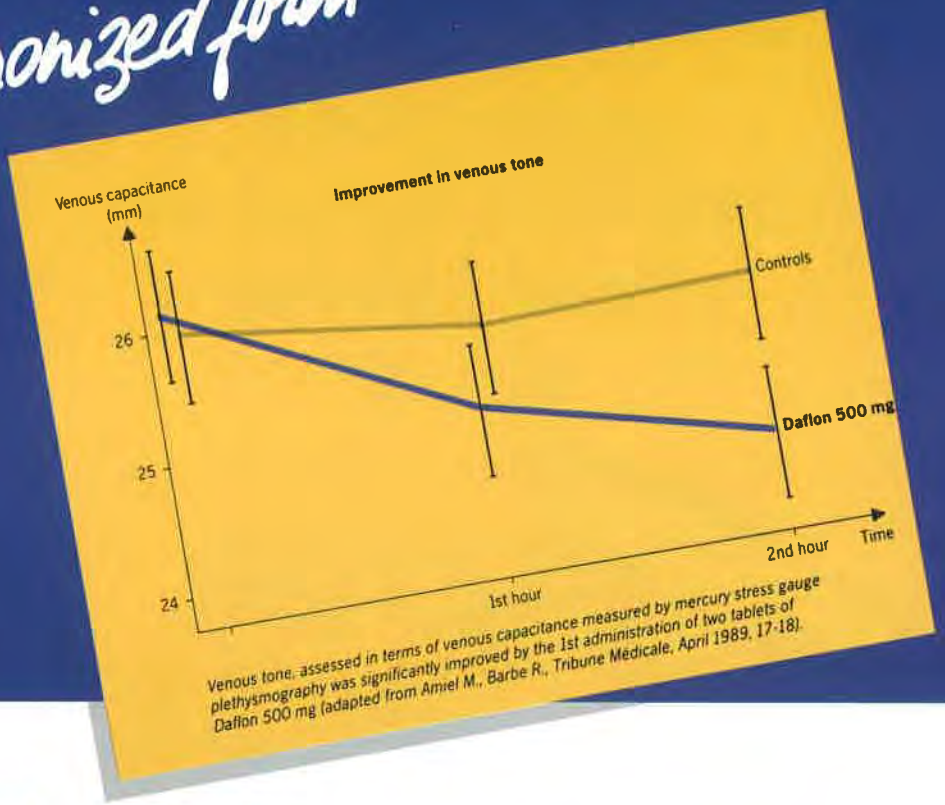

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EDITORIAL

NIDDM: AN ICEBERG DISEASE

Whilst the majority of patients with insulin dependent diabetes mellitus (IDDM) are under the care of specialists, the majority of non-insulin dependent diabetes mellitus (NIDDM) patients are looked after by general practitioners. In the 1988 one-day morbidity survey, diabetes mellitus accounted for 8% of all outpatient attendances in the Government polyclinic and 5% in the private clinic; most of these were NIDDM patients.

Current knowledge is to regard NIDDM as an iceberg disease, meaning that the disease appears more benign than it actually is. Not many patients are troubled by the symptoms of polydipsia and polyuria. Not many suffer from acute complications of cellulitis, neuropathy or pruritus vulvae. Most of the NIDDM patients with uncontrolled blood sugars are relatively asymptomatic. Unfortunately, NIDDM is also characterised by the insidious development of damage to many organs of the body (eye, kidney, heart and nerves); damage that does not occur in non-diabetic people and is clearly related to the metabolic disturbance. The degree of metabolic disturbance required for the development of the late complications is highly variable, but it is clear from those non-insulin dependent patients who present with complications that such damage can occur in the absence of symptoms.

Some idea of complication rates in newly diagnosed NIDDM patients is provided by Cheah et al's survey of diabetes in Singapore in 1985[1]. In their study of 196 newly-diagnosed patients, there were 17 (8.7%) who had nephropathy, 14 (7.1%) who had retinopathy, 12 (6.1%) who had ischaemic heart disease, and 4 (2.0%) who had neuropathy. Fifty three (27%) had hypertension.

Diabetes mellitus care requires several paradigm shifts on the part of the patient. The patient is used to the remedial model of care[2]. The doctor treats the disease and the patient recovers. This is not so in diabetes. The strategy of care is in the prevention of complications in the future whilst the patient is relatively well today, the so-called preventive model of care. Then, there is the self-care model instead of the doctor-directed model. Patients are used to the doctor taking the active role in health care. In diabetes mellitus, the doctor advises the patient. It is the patient that makes decisions about the daily actions that will affect his diabetic control.

The message is clear. Delivery of diabetes care must be directed towards prevention of tissue damage, and this care has to be delivered in and by an asymptomatic patient. Organisation of care has to be largely focussed on preventive medicine rather than on classical remedial medicine upon which medical care had achieved much of its success.

The doctor needs to review his management strategies with the aim of providing effective preventive care. The patient needs to be properly inducted into the meaning of diabetes and how he can contribute to the total care of his medical condition. This begins with the initial visit to the doctor. It is during the first few visits that the stage is set for success or failure for a particular patient. It is difficult to convince a patient who has been accustomed to the lax control for a few years that he needs to pay attention to his diet, to reduce weight, to exercise and to maintain a near normal blood sugar at all times.

This issue of the Singapore Family Physician focuses on the fundamental aspects of care of the NIDDM patient: initial management, dietary management, weight control, drug therapy, exercise and continuing care. We must, as a profession, make concerted efforts to educate the patient on the importance of these in the control of diabetes and its metabolic complications.

MV

References

1. Cheah et al. Epidemiology of diabetes mellitus in Singapore: Comparison with other ASEAN countries. Ann Acad Med 1985; 14:2:232-239.
2. Turnbridge WM and Home PD. Diabetes and Endocrinology in Clinical Practice. London: Edward Arnold, 1991:83.

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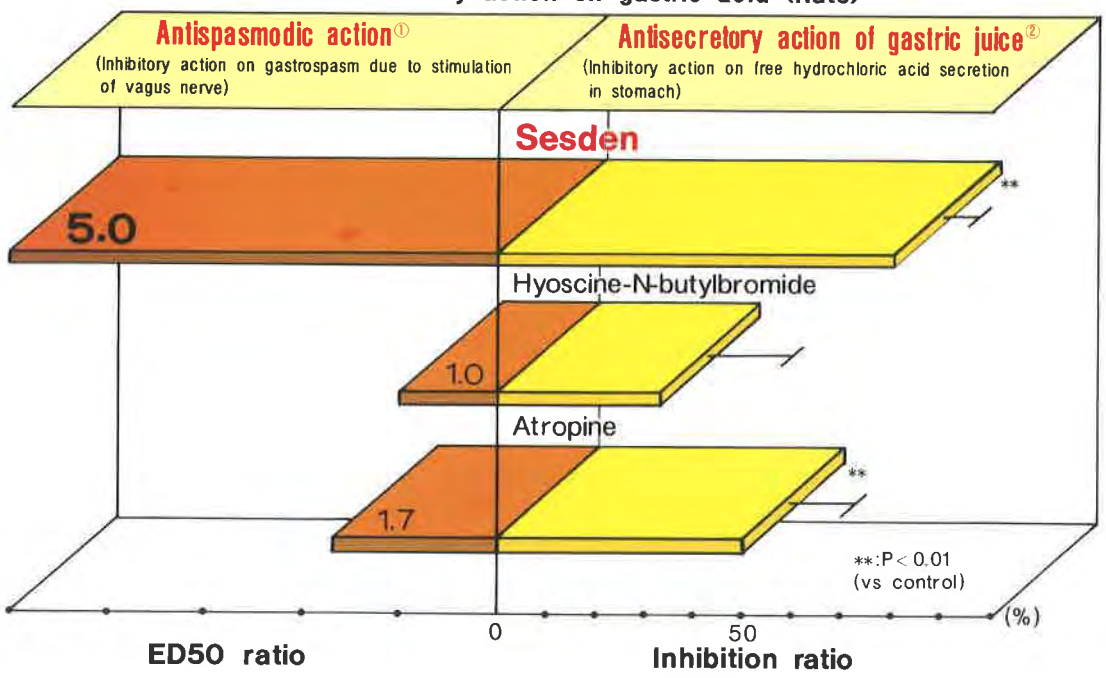

① H. Tamaki : Jpn. J. Pharmacol. 22 685(1972)
② S. Harigaya : Folia Pharmacol. Japon. 78 71(1981)

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DIABETES — THE SINGAPORE EXPERIENCE

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INTRODUCTION

Diabetes mellitus, as a disease, demands much respect. It has a long history. Ancient Chinese medical writings described a syndrome of polyphagia, polydipsia and polyuria. The importance of diabetes is further demonstrated by its high prevalence throughout the world.

However, despite the magnitude of its impact and extent, diabetes has often not been viewed by patients and doctors alike, as "threatening". People with diabetes talk of "living with the disease", and not "dying from it". This is because the diabetic is often able to live a "normal" life for about a decade or more after being diagnosed. Only then does the patient discover that this insidious disease has, in the meantime, run its smouldering course and progressed to complications which are incapacitating or life-threatening. Diabetes, we know today, doubles the risk of disability or death from coronary heart disease or stroke. It is the leading cause of blindness and amputations among adults and accounts for one-third of all causes of kidney failure. The public health importance of diabetes is undisputed.

In Singapore, we find that diabetes exerts the same impact as in other developed countries. This has been the consequence of our rapid socio-economic development and the affluent lifestyles which followed.

The national prevalence of diabetes in Singapore doubled from 1.7% in the population aged 15 years and above in 1975 to 4.7% in 1984. The level now is 8.6%, once again a near doubling of the 1984 level. This is a disturbing trend.

When narrowed to the population aged 40 to 69 years, which is the target population for mature-onset diabetes, the level of diabetes in the Singapore population rises to 18% in 1992. By contrast, the rate for Chinese is 1% in China and 6% in Taiwan. The rate among the Chinese in Mauritius, on the other hand, is 14%. This is a country where there is a high level of obesity and inactivity. Among White populations, diabetes prevalence rates vary from 3% in Australia to 2% in Britain and 5 to 6% in the US.

Coronary heart disease mortality in Singapore for diabetes has also not declined as diabetes is a major risk factor for this condition. When compared with similar communities, Singapore's coronary heart disease death rates are very high and have reached the levels of developed countries who have experienced a close to 50% decrease.

Admissions for diabetes currently average 12 days of stay in our hospitals for each episode of admission. Overall, diabetes is the leading single disease condition for patient utilisation in acute hospitals today. Diabetes was responsible for 47,000 patient days in 1990, comprising 14% of all patient days in our hospitals. This has been steadily rising in recent years. Mature-onset diabetes accounts for over 90% of all cases of diabetes in Singapore.

However we all know that relatively few patients are admitted to hospitals and even fewer patients die with the straightforward diagnosis of diabetes. Most admissions and deaths are due to the complications of this disease. Even so, diabetes

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was the sixth leading cause of death in 1991.

Medical knowledge tells us that diabetes is not a simple disease but is part of a complex of lifestyle-related disorders such as obesity, physical inactivity, unbalanced diet, hypertension and a general unhealthy state of health. There is a definite familial predisposition to diabetes but lifestyle-modified behaviour powerfully reduces the onset of this disease.

The majority of diabetics are managed by primary health physicians for the greater part of their illness. A survey in 1988 of patients seeking care from primary health physicians showed that general practitioners provide three-quarters of outpatient care in Singapore. The survey also showed that government clinics see proportionately more diabetics (8% vs 2% of other cases). Well-treated and well-managed diabetics, during the early period of their disease, can lead normal and productive lives and can be prevented from progressing to complications. This is when the patients are under the care of their primary health physicians. This is obviously where the best strategy for the control of diabetes must lie. Then only can we see a reduction in the prevalence of the disease among Singaporeans, a reduction in the economic loss of work days from diabetes and its many complications such as coronary heart disease, blindness, amputation and end-stage kidney failure.

Good diabetes control during the care by the primary health physician requires close supervision and monitoring of the patient, with continuous support in terms of education and motivation. This is to help the diabetic adopt and maintain the appropriate healthy lifestyles to keep his condition in good control. This forms the basis for the Diabetes Training Programme, aimed at Primary Health Physicians and the Diabetes Care Support Team. The Support Team has an important role to play and comprises Nurses, Nutritionists and Podiatrists, all of whom are essential to the complete care of the diabetic. The training programme will be a comprehensive one, providing up-to-date state-of-the-art information in all aspects of proper diabetes control and management at primary health level. Doctors who have undergone the training will be well informed and will be able to motivate patients to change their lifestyles in order to prevent the disease from degenerating. These doctors can refer their patients to a Diabetes Education Centre where they will receive complete and individualised advice on all aspects of their care, covering self-care, self-responsibility and self-coping. The patient should then be well-motivated to change his lifestyle appropriately and better control his disease. The Diabetes Training Programme for Primary Health Physicians and the Diabetes Care Support Team is therefore a strategic and crucial programme for the control of diabetes in Singapore.

To set up this programme, the Ministry of Health has worked closely with the College of General Practitioners and the Diabetic Society of Singapore, the three relevant organisations for a venture in this area. It has been shown to be a very successful effort. This illustrates yet another example of successful public and private sector collaboration. Such a collabarative effort should be commended and serve as a model for other similar undertakings.

INITIAL MANAGEMENT OF NIDDM

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INTRODUCTION

The key to optimal care of the patient with Non-insulin-Dependent Diabetes Mellitus (NIDDM) is jointly held by the patient and his family doctor. There is a need for both to recognise that NIDDM is not a mild disease even though there may be few or no symptoms. Indeed, the disability in the early stages may be remarkably absent. Once the patient learns to accept the tiredness of uncontrolled diabetes, life may continue with little or no dietary control and weight control until some serious complication sets in. The patient may well regard the taking of oral hypoglycaemics that he often obtains without seeing the doctor as enough care. It is found in several studies, including the one conducted by Chan and Fong[1], that only one-third of patients have good glycaemic control. It is sad but true that many patients begin to take their diabetic control seriously only after some catastrophe has occurred. As family doctors, we need to be more pro-active in getting important messages across, both to ourselves and to our patients, right from the initial visit. What is visible in the early stages of presentation may only be the tip of the iceberg. It is good investment to spend enough time at the first consultation. It may well require 15-20 minutes, but it is time well spent.

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PRESENTATION

There are three ways that a new NIDDM patient may present to his family doctor.

1. **No symptoms**
   The patient is picked up during pre-employment examination or by a diabetic relative; very often the person picked up is the result of curiosity: he decides to get his urine or blood tested, just in case, and it turns out that the person being tested is diabetic. In the female patient, the patient may be discovered during routine testing during pregnancy.

2. **Subacute symptoms**
   The patient may experience tiredness, malaise, polyuria, polydipsia or weight loss for several months before he finally decides to seek a consultation.

3. **Acute symptoms**
   An acute infection of the foot, skin or genitalia or peripheral neuropathy presenting as painful legs may be the first indication of diabetes.

AIMS OF MANAGEMENT

In today's management of NIDDM, there are four main aims:

1. **Relieve symptoms**
   The subacute symptoms of polydipsia, polyuria, polyphagia and weight loss that prompt some patients to seek care require relief. In those who present with acute complications like pruritus vulvae and cellulitis, it is not enough to address only the management of these without attention to possible underly-

ing diabetes. Even if the patient is not symptomatic, it is important to impress on the patient that moderately raised blood sugar causes tiredness and he is at risk of infections.

2. **Critical control in certain situations**
Critical glycaemic control is important in pregnancy for fetal well-being and the reduction of fetal wastage.

3. **Prevent or delay long-term complications**
Whilst there is no hard evidence that tight control prevents complications, the available results are suggestive. The results of the Diabetes Control and Complications Trial (DCCT) study, a multicentre trial being carried out in USA and Canada to confirm the relationship between long-term complications and plasma glucose concentrations, are being awaited with interest. These will be available in 1993.

4. **Psychological adjustment, quality of life and longevity**
NIDDM is a chronic disease. The patient needs to understand and accept the disease, to acquire appropriate attitudes and willingness to make lifestyle changes in order to bring the disease under control. There is a need for tenacity to keep working at the disease day after day, month after month and year after year. The encouraging words of family members and particularly the family doctor are important in empowering the patient to carry on.

CURRENT CONCEPTS

1. **Multifaceted disease**
It is no longer enough to regard NIDDM as a blood sugar disease. NIDDM is associated with resistance to insulin-stimulated glucose uptake, hyperinsulinemia, hyperlipidemia and hypertension. Together they form the components of Syndrome X, described by Reaven in 1988[2]. Obesity is an important factor in insulin resistance. The importance of weight and diet control is to reduce the peripheral insulin resistance, reduce excessive secretion of insulin and prevent further islet cell damage from glucose toxicity.

2. **Importance of lifestyle changes**
Diet control, weight control and exercise remain the cornerstones of management. Smoking is an additional risk factor in diabetes for ischaemic heart disease.

3. **Use of oral hypoglycaemics agents (OHAs) and insulin**
Drug treatment should only be started if diet and weight control have been tried and failed to relieve symptoms or control blood glucose adequately, and not before. The therapeutic approaches start with the relief of the over-loaded ß-cells by dietary manipulation and exercise. If this is not entirely effective, the next step is to promote peripheral action of insulin (with a biguanide) or stimulate exhausted ß-cells with sulphonylureas. Finally, the switch to insulin or insulin-OHA combination may be required in the NIDDM patient whose hyerglycaemia has not been reduced by maximal dosage of both biguanide and sulphonylurea.

4. **Teaching patient skills**
There is a need to spend efforts to empower the patient to provide self care. These include learning self monitoring of blood glucose and general care of skin, feet and blood pressure, to quit smoking and to learn food choices at home and when eating out. Sick-days care should also be learnt.

5. **Eye disease and Blood Pressure control**
Strict blood pressure control prevents the acceleration of diabetic retinopathy as the result of hypertension.

6. **Need for yearly review**
The yearly review is an important feature of continuing care in NIDDM. It is an opportunity to review in detail all drug therapy, smoking and drinking habits, problems with treatment and occurrence of complications. It is also an opportunity to redefine the targets of care. Annual review is only useful if the results are adequately recorded on well designed forms, if appropriate action is subsequently taken on identified problems, and if appropriate recall procedures are in place to ensure the annual appointments are issued and attended[3].

STEPS IN THE INITIAL CARE OF THE NIDDM PATIENT

There are nine tasks to complete.

1. **Confirm diagnosis with blood glucose level**
   Diabetes mellitus is a long term medical problem. Hence, there must be a firm basis for labelling the patient as such. Diagnosis in non-pregnant adults is established if ONE of the following three criteria is fulfilled:

Criterion 1: Presence of symptoms and **unequivocal** elevation of either the *random* or *fasting* glucose concentration as shown in Table 1.

Table 1. Abnormal blood sugars

Test blood	Venous plasma mmol/l (mg/dl)	Venous whole blood mmol/l (mg/dl)
Random glucose	≥ 11.1 (200)	≥ 10.0 (180)
Fasting glucose	≥ 7.8 (140)	≥ 6.7 (120)

Criterion 2: In the absence of symptoms, at least TWO random or fasting glucose concentrations exceeding the cut-off values stated in criterion 1, obtained on separate occasions.

Criterion 3: When either criterion 1 or 2 has not been met, only then will an oral glucose tolerance (OGTT) be necessary. The only other indication for OGTT is for the diagnosis of gestational mellitus.

Table 2. Diagnostic Values for OGTT (1980 WHO Criteria)

	Venous plasma mmol/l (mg/dl)	Venous whole blood mmol/l (mg/dl)
FASTING glucose	≥ 7.8 (140)	≥ 6.7 (120)
and / or 2-hour glucose	≥ 11.1 (200)	≥ 10.0 (180)

2. **Initial evaluation – Is it secondary? Is it NIDDM?**

   It is necessary to decide whether the diabetic state is secondary to drug causes (steroid therapy and thiazide therapy), pancreatic disease, or hormonal disturbances like Cushing's syndrome or acromegaly. Genetic and other syndromes such as glycogen storage disease, Prader-Willi syndrome, ataxia telangiectasia or lipodistrophy are quite rare. Also, it is necessary to confirm that the patient is non-insulin dependent. History of diabetic ketoacidosis, age of onset under 30 years, severe weight loss and short history of symptoms are points that suggest insulin-dependent diabetes.

3. **Conduct initial assessment**

   A checklist is given in the Appendix to this paper on the information to collect at the initial visit. Broadly, the checklist covers medical history: specific symptoms, additional risk factors; a systemic review to check for complications; a dietary history to provide a basis for dietary advice; a psychological assessment; a socio-economic assessment; a physical examination; and baseline laboratory investigations.

4. **Decide place of initial management – outpatient or inpatient**

   With the exception of cellulitis as the presenting problem, the NIDDM patient can be managed as an outpatient.

5. **Decide initial therapy – tablets or not?**

   It is important not to fall into the trap of prescribing oral hypoglycaemics to all diabetic patients. Weight control and dietary management may be enough. It is important to spend time to explain to the patient this non-pharmacological approach in the first instance.

6. Have a referral policy

The core of any effective diabetic service consists of two elements, namely, medical staff with support facilities, and an educational service with appropriate teaching aids. A routine referral policy should be worked out depending on how much the family doctor would be able to do at the clinic. Referral to the dietitian may be necessary if the doctor has no time to educate the patient or if the case is problematic. Initial referral to the ophthalmologist for assessment of retinopathy will be necessary, as some 7% of patients will have retinopathy at first examination[4].

7. Deliver initial health education

It is important to avoid an information overload during the first visit. Finding out the patient's ideas, concerns and expectations enables the family doctor to customise the health education to meet the patient's needs. It may be necessary to correct misconceptions that the patient has. The health education messages to be imparted at the first visit are shown in Table 3.

8. Plan subsequent health education

The newly diagnosed NIDDM patient has to learn self-care and survival skills. The family doctor may teach some of these himself and refer the patient for further health education to the dietitian and health education bodies. The Singapore Diabetic Society runs courses for diabetic patients.

Table 3. Initial health education of the NIDDM patient

* The importance of lifestyle change
  – weight control (especially, if overweight)
  – healthy diet (may wish to refer to dietitian as well)
  – exercise (start with simple ones)
* The initial management is non-pharmacological, namely weight control, healthy diet
* The need for a regular follow-up plan (initially more frequently; quarterly or less often when controlled; and an annual check-up).
* The need for an eye check-up – initially and annually.

Table 4. Self-care and survival skills

Self-monitoring blood glucose
General care: skin, foot, BP, quit smoking
Food choice, eating out
Sick-days care

9. Establish follow-up plan

The patient should be briefed on the follow-up plan. Attempts should be made to involve family members as well. Setting metabolic targets helps the patient to have objective goals to achieve (Table 5).

Table 5. Metabolic Targets in the management of diabetes

	Good	Acceptable	Poor	High risk
HbA1c (%)	6 -	8 -	10 -	12 -
Glucose (venous plasma, mmol/l)				
Fasting	< 5.5	5.5 -	6.7 -	> 9.0
Post-prandial	< 6.7	9.0 -	10.0 -	> 11.0
Cholesterol (serum, mmol/l)	< 5.2	5.2 -	6.5 -	> 7.8
Triglycerides (serum, mmol/l)				
Fasting	< 1.7	1.7 -	2.2 -	> 3.5
Body mass index (kg/m2)				
Male	< 25.0	25.0 -	27.0 -	> 30.0
Female	< 24.0	24.0 -	26.0 -	> 29.0

CONCLUSIONS

There are four take-home messages relevant to the initial management of the NIDDM patient. First, NIDDM is a multifaceted disease and it is not enough to focus only on hyperglycaemia. Second, there is a need to be patient-oriented and involve the patient; attention should be paid to explain the meaning of the disease in terms of what dividends can be had in maintaining weight and dietary control; time should be spent in initiating self-care skills. Third, modern diabetic care requires the involvement of the health care team, such as the services of the dietitian, the endocrinologist and the ophthalmologist in providing the necessary care and surveillance. Lastly, there is a need to have a continuing care plan.

References

1. Chan PSM & Fong NP. Management of diabetes mellitus in general practice: a study of 499 cases in Singapore. Sing Fam Physician 1989; 15:1:131–139.
2. Reaven GM. Banting Lecture 1988. Role of insulin resistance in human disease. Diabetes 1988;37:1595–1607.
3. Turnbridge WM and Home PD. Diabetes and Endocrinology in Clinical Practice. London: Edward Arnold,1991:83.
4. Cheah et al. Epidemiology of diabetes mellitus in Singapore: Comparison with other ASEAN countries. Ann Acad Med 1985; 14:2:232–239.

APPENDIX: INITIAL ASSESSMENT CHECKLIST OF THE NIDDM PATIENT

MEDICAL HISTORY

* Specific symptoms
  - weight loss
  - polyuria
  - polydipsia
  - enuresis/nocturia
  - fatigue

* Additional risk factors
  - family history of diabetes, ischaemic heart disease and cerebrovascular accidents
  - hypertension
  - hyperlipidemia
  - smoking
  - lack of exercise
  - drugs e.g., steroids
  - alcohol

* Systemic Review
  - neurological symptoms (paraesthesia, numbness, dizzy spells, weakness of lower limbs, bladder and sexual dysfunction)
  - foot and toe problems
  - infection (e.g., skin infections, urinary tract infection, balanitis, pruritus vulvae)
  - visual acuity
  - ischaemic heart disease
  - vascular problems (cold peripheries, claudication, chronic ulcers)
  - obstetric history (e.g., gestational diabetes, big babies, recurrent abortions, infertility)

DIETARY HISTORY

* patterns of diet
  - number of meals per day
  - timing of meals
  - home meals and non-home meals

* types of diet:
  - high carbohydrate
  - high protein
  - high fat
  - vegetarian

PSYCHOLOGICAL ASSESSMENT

* personality type, outlook on life
* View of diabetes as a disease
* Misconceptions of diabetes
* Presence of denial, guilt and depression
* Level of education and intelligence

SOCIO-ECONOMIC ASSESSMENT

* Work/occupation (type of work, hours of work, shift work, environment)
* Hazards at work (heights, machinery)
* Financial status, position in society
* Social support (home/family, worksite/employment, food source)

PHYSICAL EXAMINATION

(to include basic measurements and target organ assessment)

* Weight
* Height
* Body mass index (BMI)
* Blood pressure – standing and lying
* Cardiovascular system including peripheral pulses (dorsalis pedis, femoral, carotid, radial)
* Eyes:
  - visual acuity
  - premature cataracts
  - fundoscopy/retinal photography
    - microaneurysms
    - haemorrhages
    - exudates
    - neovascularisation

  Feet:
  - skin condition (including cold peripheral skin, loss of hair, chronic ulcers)
  - diminished pulses
  - pressure areas
  - interdigital problems
  - abnormal bony structure
  - nail hygiene

Neurological system:
- light touch
- pin-prick sense
- vibration sense
- position sense
- tendon reflexes (knee, ankle)
- Babinski reflexes
- motor power of limbs
- Renal system – dependent oedema

LABORATORY INVESTIGATIONS

* Fasting blood glucose
* Urinanalysis: glucose, ketones, protein; blood, nitrates and microscopy
* Serum creatinine
* Fasting triglycerides/cholesterol/HDL/LDL
* Glycosylated haemoglobin (e.g., HbA1c, HbA1)
* ECG
* CXR

DIETARY MANAGEMENT IN DIABETES

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HISTORICAL DEVELOPMENT OF DIETARY MANAGEMENT

The need for dietary restrictions was first recognised in insulin-dependent diabetics. Prior to the development of insulin for injection, such persons were not expected to live past childhood. Control then consisted of restricting dietary carbohydrate severely in an attempt to maintain normal glucose levels. The advent of mass-produced insulin brought a new era in diabetes management. A more liberal intake of carbohydrates was allowed. This was the era of the 40% carbohydrate diet and 10g carbohydrate exchanges. Today, the carbohydrate content has been further liberalised to 50-60% of total energy requirements with a preference for high-fibre complex carbohydrates.

It is now recognised that the two types of diabetes require almost opposite kinds of dietary management. Type II diabetes calls for self-restraint in the consumption of food, but there is little need for a rigid meal schedule. For the overweight person with Type II diabetes, fewer calories and more exercise to achieve weight loss are critical to diabetes management. Type I diabetes calls for more predictable, regular access to meals with the awareness that extra food may be needed at times to compensate for physical activity.

Many of the dietary guidelines for healthy people are also specific management strategies for the control of diabetes: maintenance of ideal body weight, emphasis on complex carbohydrate and fibre, avoidance of sugar, fat and sodium. It is important to note that in principle, the diet for a diabetic person is no different from a healthy diet for a non-diabetic person. Body weight and carbodydrate intake contribute to the control of glucose levels. It is especially important for the individual with diabetes to avoid excess fat and sodium because of the increased risk of developing late complications: atherosclerosis, hypertension, renal failure and blindness.

GOALS

Dietary management is the cornerstone of management for every diabetic. It has several goals. These are to:

* control blood glucose levels to prevent hyperglycaemia, hypoglycaemia and complications of diabetes mellitus.
* achieve and maintain desirable body weight.
* provide adequate nutrition to improve overall health.
* maintain blood lipids within normal range.
* achieve normal growth in children and adolescents.

RECOMMENDED GUIDELINES FOR DIABETICS

Many countries have produced national dietary guidelines for people with diabetes. Table 1

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Table 1. Recommended dietary guidelines for individuals with diabetes

* A high intake of starchy fibre-rich foods which make up about 50 to 60% of total calories.
* A fat intake of 25-30% of total kilocalories to help control unnecessary weight gain and to protect against heart problems. *Saturated fat should be less than 10% of the total kilocalories, the rest should be unsaturated fat.*
* An intake of protein foods which makes up to about 15 to 20% of total kilocalories. Proteins from both animal and plant sources are recommended.
* A cholesterol intake of less than 300mg a day (100mg per 1000 kilocalories).
* A total salt intake of less than 5g a day (2000 mg sodium).
* Alcohol is not recommended for overweight diabetic individuals.

summarises the recommended dietary guidelines of the Singapore Dietitians' Association (September 1988).

TEACHING THE DIABETIC PATIENT TO MAKE CHANGES

To make changes, the diabetic patient needs to know basic nutritional facts, meal planning and food selection skills and apply these in his daily life.

BASIC NUTRITIONAL FACTS

Kilocalories and weight control

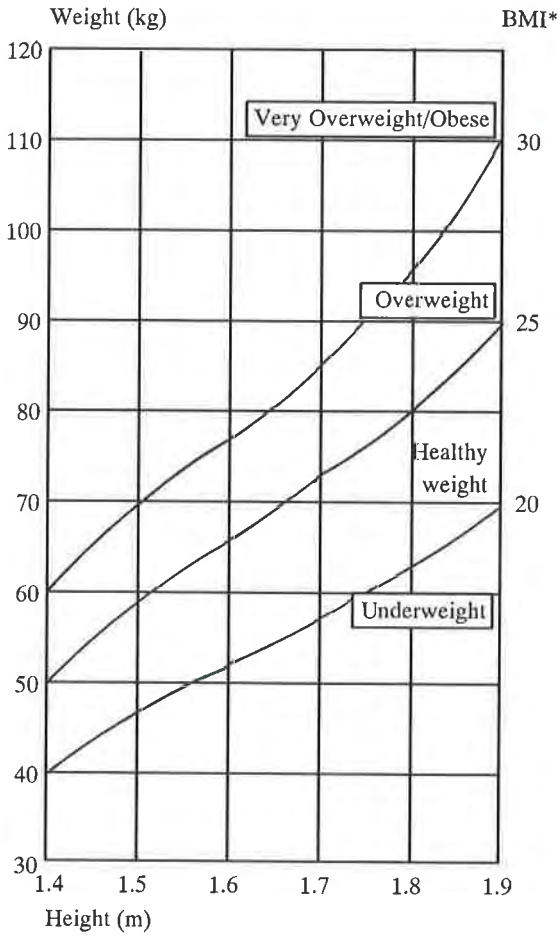
Keeping body weight within the healthy weight range is an essential part of the total diabetic management. Being overweight makes diabetes difficult to control. Table 2 shows the use of the body mass index (BMI) as a measure of weight status. Small framed persons should maintain their weight at the lower end of the weight range. The weight can also be read off from the weight for height chart (Figure 1).

Table 2. BMI and weight status

$$BMI = \frac{\text{Weight in kg}}{\text{Height in metres X Height in metres}}$$

BMI	STATUS
Less than 20	Underweight
20 to below 25	Healthy weight
25 to below 30	Mild overweight
30 to below 40	Moderate overweight
40 and above	Obesity

WEIGHT FOR HEIGHT



The majority of overweight persons – about 90 percent – fall in the category of mild overweight. For these individuals, only a moderate kilocalorie restriction is indicated, along with increased physical exercise. Such a programme can also be provided by nonphysician health professionals or community groups. Table 3 shows the instructions for weight reduction.

Table 3. Instructions for weight reduction

The principle is to lose weight gradually. NEVER reduce weight through a crash course unless supervised in a hospital.

* The target is to lose between 1/2 to 1 kg per week
* To lose 1/2 kg of weight a week, you need to burn off 500 kilocalories a day.
* This can be achieved by reducing your calorie intake and increasing your calorie expenditure.
  For example, if you eat 250 kilocalories per day and exercise to burn off 250 kilocalories per day, you will create a deficit of 3500 kilocalories per week, which is equivalent to 1/2 kg of fat.

Carbohydrates

Carbohydrates are the main source of energy for the body. One gram of it yields 4 kilocalories. Carbohydrates are no longer restricted so much as they once were. The recommended allowance is 50-60% of total calories from carbohydrates. A distinction is made between simple sugars ("quick carbohydrates") and complex carbohydrates which are found in starches ("slow carbohydrates"). The latter is preferred.

Simple Sugars

Besides glucose and table sugar, simple sugars are found in sweetened condensed milk, kaya, jams, ice-cream, ice-sticks and sweetened soft drinks. As they are high in kilocalories, they contribute to weight gain. As they are rapidly absorbed into the blood stream when ingested, simple sugars result in poor control of diabetes. For these two reasons, simple sugars should therefore be avoided by people with diabetes.

Ripe fruits and milk also contain simple sugars (fructose and lactose respectively). Fruits are also rich in fibre, minerals and vitamins; milk is rich in protein and calcium. They are therefore allowed as part of the meal plan for people with diabetes.

Complex Carbohydrates

Starches are complex carbohydrates. They are more slowly absorbed from the gut and therefore allow better control of blood sugar levels. Some examples of starches are rice, oats, wheat flour, pulses or legumes.

Fibre

It is important to mention fibre in the diet. Fibre refers to a group of foods of plant origin which the human gastrointestinal system (stomach and intestines) cannot digest. Fibre can slow carbohydrate absorption and consequently the rise of blood sugar is more gradual.

Fibre may be soluble or insoluble.

* **Water soluble** e.g., gums and pectins.
  Water soluble fibre is found in beans, pulses, oats, barley, apples, citrus fruits and vegetables. This type of fibre slows down gastric emptying. This slowing down can affect the glycaemic response and thus can be useful for people with diabetes. A high intake of soluble fibre particularly gel-forming fibre (found in beans and pulses) also leads to a reduction in blood cholesterol by decreasing intestinal absorption and increasing faecal excretion of dietary cholesterol.

Table 4. Dietary Fibre in Nuts and Seeds

Food Per 100g edible portion	Total Dietary Fibre (g)
Chestnut, dried	41.80
Candle nut – dried	8.57
Cashew nut dried	7.91
Lotus seed, dried	33.60
Jack fruit seed, raw	18.00
Sunflower seed, dried	8.97
Pumpkin seed, dried	8.11
Water melon seed, dried – black	8.02
Water melon seed, dried – red	7.41
Gingko seed, raw	6.56

Source. Gourley L. Lee HP and Lee SM. Dietary Fibre – its Components in Some Southeast Asian Foods. The Singapore Dietitian, 1987;3;1:10-11.

Table 5. Dietary Fibre in Cereals and Beans

Food Per 100g edible portion	Measure	Total Dietary Fibre (g)
Wholemeal bread**	1 slice (30g)	2.0
White bread**	1 slice (30g)	0.9
Unpolished rice, cooked	1 mb (150g)	2.6
White rice, cooked	1 mb (150g)	1.2
Oats, uncooked	15g	1.0
Green gram*	50g uncooked	7.5
Black bean*	50g uncooked	7.5
Dhall (lentils)*	50g uncooked	6.8
Red bean*	30g uncooked	7.5

Sources: **Australian Composition of Foods, 1981.*
***Sunshine bread*

Table 6. Dietary Fibre in Local Vegetables

Food Per 100g edible portion	Total Dietary Fibre (g)
Stink bean (petai)	5.55
Lady's fingers	4.95
Snow peas – edible-podded	4.59
Water chestnut	3.56
Chinese lettuces (local)	3.37
French beans (local)	3.23
Kangkong	3.13
Sweet potato leaves	2.77
Bitter gourd	2.75
Egg plant – purple	2.55
Spinach	2.25
Lotus tuber	2.04
Chinese cabbages, small	2.00
Hairy gourd	1.92
Wax gourd	1.73
Radish (oriental)	1.64
Chinese cabbages, big	1.43
Cucumber (locally grown)	1.24
Old/brown cucumber	0.97

Source. Gourley L. Lee HP and Lee SM. Dietary Fibre – its Components in Some Southeast Asian Foods. The Singapore Dietitian, 1987;3;1:10-11.

Table 7. Dietary Fibre in Local Fruits

Food Per 100g edible portion	Total Dietary Fibre (g)
Chiku (Sapodilla)	8.60
Jackfruit (Chempedak)	5.47
Guava	5.18
Durian	4.41
Banana (average)	4.10
Soursop	3.36
Jackfruit (Nangka)	2.78
Orange – Thai, thick skin	2.70
Orange – Thai	2.70
Papaya	2.50
Starfruit	1.88
Mango – Filipino	1.83
Pineapple	1.69
Lychee	1.64
Pear Korean, brown skin	1.54
Rambutan	1.46
Mangosteen	1.44
Longan	1.06
Watermelon	0.39

Source. Gourley L. Lee HP and Lee SM. Dietary Fibre – its Components in Some Southeast Asian Foods. The Singapore Dietitian, 1987;3;1:10-11.

* **Water insoluble** e.g., cellulose and hemicellulose.
A typical food that is high in insoluble fibre is wheat bran. Other examples are whole-wheat bread and vegetables. Insoluble fibre holds onto, but does not dissolve in, water, making foodstuffs move faster through the intestinal tract. The fibre is not digested and contributes to faecal bulk, occasionally acting as a laxative. Such fibre is useful in a weight reduction programme as it provides bulk and satiety. Insufficient intake of fibre is associated with various disorders of the bowel, including cancers, piles and diverticulitis.

The recommended amount per day is 25-30g. An increase in dietary fibre should be done gradually and accompanied by an increase in consumption of water. Local vegetables and fruits which are found to be rich in fibre are: stink bean, snow pea, lady's fingers, sapodilla, jackfruit, guava, nuts, seeds, fungi and seaweeds. See Tables 4 and 5 for fibre content of nuts, seeds, cereals and beans,

Table 8. Dietary Fibre in Local Cooked Food

Food Per serving	Dietary fibre (mg)	Food Per serving	Dietary fibre (mg)
POULTRY		NOODLES	
Chicken, duck	–	Satay beehoon	18.1
		Beehoon with cuttlefish and kangkong	11.4
MEAT		Mee siam	9.8
Beef, Mutton, Pork	–	Mee rebus	9.3
		Wanton noodles, dried	5.3
SOUP		Beef noodles, soup	4.2
Peanut with pork ribs	7.3	Fried hor fun	3.9
Carrot with potato and pork bones	4.5		
Herbal chicken	0.4	BREAD BUNS	
		Chicken curry bun	1.6
SEAFOOD		Otar bun	1.3
Fish curry with okra	5.5	Sausage bun	1.1
Fish masak pedas	4.0	Cheese bun	1.0
Prawn fritters	1.5	Bun susi	0.8
VEGETABLES		MISCELLANEOUS	
Mixed vegetables: carrots, cauliflower, mushroom	7.7	Chinese rojak	7.8
Lady's fingers fried with shrimp paste	6.1	Carrot cake mashed and fried	5.0
Kailan fried with beef slices	5.6	Yong tau hoo (mixed)	4.9
Kangkong fried with shrimp paste	5.4	Bak pow	3.9
Brinjal curry	4.9	Chwee kway	3.3
Chap chye	4.1	Yam cake (1 piece)	2.1
French beans fried with onions and dried prawns	4.0	Rice dumpling, nonya type	2.0
		Carrot cake piece, deep fried	1.4
		Sweet potato fritters (1 piece)	1.3
		Char siew pow (steamed)	1.0

Source: Food & Nutrition Department. Proximate Food Composition. MOH, 1992.

and Tables 6, 7 and 8 for fibre content of locally eaten foods.

To meet dietary fibre requirements include the following in the diet daily:

Bread, wholemeal -
2 slices or 1 medium bowl oats

Fruits -
2 serves e.g., apple, guava, pear (include fruit skin), banana, papaya, orange

Vegetables -
2 serves e.g., snow peas, lady's finger, french beans, carrot, cabbage, spinach

Unpolished rice -
mix with white rice (3 to 4 times per week and gradually increase to daily, if possible)

Beans, pulses -
red bean or green gram soup (as snacks) frequently.

Fats

Fats are concentrated sources of energy. One gram of it yields 9 calories. Approximately 20-30% of the total kilocalories should be derived from fat. There are three types of fats, namely, saturated, mono-unsaturated and polyunsaturated fats. All fats yield the same amount of calories. Some fats are also rich in cholesterol (e.g., lard or meat fat).

* **Saturated fats** raise blood cholesterol. They are generally found in foods of animal origin such as milk and meat. Saturated fats are also found in coconut and palm oils.

* **Polyunsaturated fats** help to lower blood cholesterol. These fats are found mainly in plants such as soya bean, corn, sunflower and safflower seeds. Other sources are soft margarine, walnuts and fish oils.

* **Mono-unsaturated fats** are believed to have a favourable effect on blood cholesterol. They are found in olive oil, peanut oil and nuts except walnuts.

As fats are high in energy, they should be used

sparingly especially for those who are overweight. Low-fat foods, lean meats and polyunsaturated fats are emphasised to prevent cardiovascular disease. Cholesterol restrictions are imposed if ordered by the physician. See Table 9 on reducing fat intake.

Table 9. Reducing fat intake

* Trim off all visible fats and skin from meat and poultry.
* Use fish, chicken and lean cuts of meat more often than red meat.
* Use skimmed or low fat milk and low fat dairy products.
* Skim fat from stock or soup.
* Replace coconut milk with skimmed or low fat milk or low fat yoghurt.
* Try steaming, boiling, grilling, microwave or baking instead of frying. When frying use minimal oil. Stir-fry instead of deep-frying.

Proteins

Proteins are important to the body for growth and repair. They also provide energy: the percentage of kilocalories derived from protein is usually 15-20%. One gram yields 4 calories. Proteins are derived from animal and plant sources. Animal proteins can be high in saturated fat and/or cholesterol. The dietary advice is to include one vegetable protein and one animal protein of low saturated fat and low cholesterol content every day. For animal proteins choose lean cuts of meat, skinned chicken, fish, skimmed milk and low fat dairy products.

Avoid proteins high in saturated fats, such as fatty meat, tinned meats, sausages, full cream milk, full cream dairy products as well as proteins high in cholesterol, such as, internal organs, egg yolk, prawns, crabs, squid and cuttlefish. Proteins from plant sources are *low in saturated fats and have no cholesterol.* Examples are: nuts, pulses and legumes and soya products.

Free foods

Free foods are foods that can be eaten freely without being counted in the meal plan (except in diabetics with kidney complications):

a) Fluids: Plain tea/coffee, Water, Sugar-free soft drinks, Mineral water, Unsweetened lime/lemon juice, Clear soup.

b) Flavourings and condiments: Herbs and spices, Ginger, Pepper, Essences, Garlic, Vinegar, Lemon, Gelatin.

c) Vegetables: *The following are generally low in carbohydrates and can be taken without restriction in the diet* – Cabbage, Winter-melon, Cucumber, Bitter gourd, Celery, Hairy gourd, Chillies, Green pepper, Green leafy vegetables. Vegetables such as carrots, and beetroot need to be taken in restricted amount – each serve of 1/2 medium bowl (cooked) contains 5g carbohydrates and 25 calories.

Cholesterol

The liver can produce all the cholesterol that the body needs. Thus, we do not need much cholesterol from dietary sources to stay healthy: less than 300 mg of cholesterol per day from the diet. (100mg per 1,000 kilocalories). The level of cholesterol in the blood is influenced not so much by the amount of cholesterol we ingest but more by the amount of total fats in our diet, especially **saturated** fats. A high intake of soluble fibre also leads to a reduction in blood cholesterol. Table 10 shows the dietary measures to reduce cholesterol intake. See Table 11 for cholesterol content of common foods, and Table 12 for that in common local cooked food.

Table 10. Meaures to reduce blood cholesterol

* Restrict foods high in saturated fats such as fatty meats, meat skin, cream, full cream milk and its products, coconut and its products.
* Increase dietary fibre by including foods such as whole grain cereals, beans and peas, vegetables and fruits.
* Avoid or eat less of foods high in cholesterol such as all internal organs, egg yolk, prawns, squid and crabs.

Salt

Salt in food comes from three sources: 50%

comes from commercial foods; 25% comes from salt added during cooking, and 25% comes naturally from foods such as meat, fish, milk, fruit and vegetables. There is actually no need to add an excessive amount of salt in cooking. A pinch of it in cooking is all that we need. Processed and preserved foods should be avoided. Salt appears under different names in commercial foods e.g., monosodium glutamate (MSG, Ve-tsin, Mei-jing), sodium phosphate, sodium ascorbate and sodium bicarbonate.

To reduce salt intake

* Avoid highly salted foods e.g., preserved fish, prawns, eggs and vegetables.
* Reduce intake of processed foods such as bacon, ham, sausages and cheese.
* Avoid the use of monosodium glutamate. It is not necessary.
* Avoid adding salt or sauces at the table.
* Avoid eating out too often.

Sweeteners

Sugar substitutes are not always preferable. Sugar alcohols, such as sorbitol and xylitol, are often found in dietetic foods; they contain 4 kilocalories/gm and thus should not be considered "free foods". In addition, excess intake may lead to diarrhoea.

Saccharin and aspartame have no appreciable kilocalorie content in amounts commonly consumed but may be used in foods contributing other sources of kilocalories, such as fat. These foods may give a person with diabetes a false perception that if the food is sugar free it is also kilocalorie free. Fructose offers no advantage over sucrose for the person with diabetes, although both sweeteners may be tolerated in small amounts.

Label Reading

The individual with diabetes should learn to be a label reader. In this way he will know more about what he is eating. Sugar, fat, salt and fibre often appear under different names. Ingredients on labels are listed in order of quantity, namely, from the largest to the smallest amount. If a product which one is to avoid/restrict is high on the list, this item is most likely unsuitable for him. On the other hand, if it is low down or at the end of the list, it is safe to eat a little of it. Various names for sugars, fats and salt may appear on food labels (Table 13).

Table 11. Cholesterol Content of Foods

Food		Cholesterol mg per 100g edible portion
Brain		2200
Kidney		400
Ikan billis	whole, dried with entrails	383
Liver	- chicken	380
	- pig	260
Egg	- one yolk	250
Butter		230
Prawns	- 6 medium	200
Squid		190
Lobster		150
Duck	- meat only	160
Chicken	- dark meat (leg)	110
Crab		100
Sardines	- canned in oil	100
Cheese	- parmesan	90
Lamb	- lean	79
Fish	- Salmon (smoked)	70
Cheese	- cheddar	70
Fats	- lard, beef dripping	70
Chicken	- light meat	69
Pork	- lean	69
Tuna	- canned in oil	65
Fish	- various	60
Beef	- lean	59
Ikan bilis	- dried minus head and entrails	58
Oysters	- 10 medium	50
Scallops	- 10 medium	40
Fish	- mackerel	30
Milk	- full cream, liquid	14
Cheese	- cottage	13
Milk	- skimmed, liquid	2
Vegetables		0
Margarine	- polyunsaturated	0

Source: McCance & Widdowson's. The composition of foods, 1988; Tee ES, Ng TKW, Chong YH. Cholesterol contents and Fatty acid composition of some Malaysian Foods. MMJ.

MEAL PLANNING

Meal Plan

A meal plan is a guide to show what the patient can eat and how much. By using the food exchange list, the patient can have a greater variety of foods.

Table 12. Cholesterol in local cooked food

Food Per serving	Cholesterol (mg)	Food Per serving	Cholesterol (mg)
NOODLES		SOUP	
Beef noodles, soup	30	Carrot with potato and pork bones	74
Wanton noodles, dried	36	Herbal chicken	91
Satay beehon	95	Peanut with pork ribs	93
Beehoon with cuttle fish and kangkong	75	Mutton	150
Mee siam	127		
Mee rebus	181	VEGETABLES	
Fried hor fun	211	Mixed vegetables: carrots, cauliflower, mushroom	-
BREAD BUNS		Chap chye	18
Chicken curry bun	4.4	Lady's fingers fried with shrimp paste	21
Cheese bun	8.6	Kangkong fried with shrimp paste	22
Otar bun	26	dried prawns	95
Bun susi	28	French beans fried with onions and	
Sausage bun	48	Kailan fried with beef slices	108
POULTRY		MISCELLANEOUS	
Roast duck	116	Sweet potato fritters, 1 piece	trace
Chicken rendang	117	Carrot cake, piece, deep fried	2
Chicken curry	169	Chwee kway	3
Chicken chop	169	Yam cake	8
		Char siew pow (steamed)	13
MEAT		Rice dumpling, nonya type	20
Lean pork fried with szechuan chye	20	Bak pow	26
Minced beef with kiam chye	94	Chinese rojak	28
Mutton rendang	98	Carrot cake mashed and fried	41
		Yong tau hoo (mixed)	49
SEAFOOD			
Prawn fritters	83		
Fish masak pedas	170		
Fish curry with okra	173		

Source: Food & Nutrition Department. Proximate Food Composition. MOH, 1993.

Table 13. Names for sugars, fats and salt on food labels

Sugar	Fat	Salt
corn syrup	animal fat	baking powder
dextrose	butter oil	bouillon
disaccharides	cocoa butter	brine
fructose	coconut oil	broth
glucose	copha	monosodium glutamate (MSG)
glucose syrup	corn oil	sodium alginate
golden syrup	cottonseed oil	sodium ascorbate
invert sugar	diglycerides	sodium benzoate
lactose	hydrogenated fat	sodium bicarbonate
maltose	lard	sodium phosphate
mannitol	monoglycerides	soy sauce
monosaccharides	shortening	
polysaccharides	vegetable fat/oil	
sorbitol	whole milk solids	
sucrose		
treacle		
xylitol		

Table 14. Nutrient content of food exchanges

Food	Nutrient content			
Exchange	CHO g	PRO g	FAT g	KCal
Starch/bread	15	3	-	80
Meat				
Lean	-	7	3	55
Medium fat	-	7	5	75
High fat	-	7	8	100
Vegetable	5	2	-	25
Fruit	15	-	-	60
Milk				
Skim	12	8	trace	90
Low fat	12	8	5	120
Full cream	12	8	8	150
Fat	-	-	5	45

Table 15. Household measures

Measurements (abbreviation)	Volume ml	CHO Exchange (Rice/noodles)	Diameter cm	Depth cm
Small bowl (sb)	150	2	9	4
medium bowl* (mb)	250	3	11	4.5
big bowl (bb)	350	5	12	5.5
large bowl (lb)	500	8	15	5.5
teaspoon (tsp)	5			
tablespoon (tbsp)	15			
Chinese tablespoon	20			

The bowls mentioned above are melamine ware. These are available in most Chinese crockery and cutlery shops. They are also widely used in hawker centres. ** equivalent to 1 cup.*

Table 16. Food exchanges for varying daily energy intakes

Food Exchange	Total daily energy intake (Cals)				
	1200	1500	1800	2000	2200
Starch/bread	6	8	10	12	14
Meat	6	6	7	8	8
Vegetables	2	2	2	2	2
Fruits	3	3	3	3	3
Milk	1	1	1	1	1
Fat	3	5	5	5	7

Standard meal patterns for calorie variations (approximate nutrition value; carbohydrate 55-60%, protein 15-20% and fat less than 30%)

Table 17a. Distribution of food exchanges for 1500 kcals

MEAL	NUMBER OF FOOD EXCHANGES					
	Starch/ bread	Meat	Vegetables	Fruit	Milk	Fat
Breakfast	2	-	-	-	-	1
Mid-morning snack	-	-	-	-	-	-
Lunch	3	3	1	1	-	2
Mid-afternoon snack	-	-	-	1	-	-
Dinner	3	3	1	1	-	2
Bedtime snack	-	-	-	-	1	-
Total	8	6	2	3	1	5

Table 17b. Menu plan for 1500 kcals

Exchange	No of exchanges	Food portion
Breakfast		
Starch/bread	2	2 slices wholemeal bread or alternative
Meat	-	
Milk	-	60 ml skim milk
Fat	1	1 tsp margarine or alternative
Coffee/tea	-	1 cup
Mid-morning snack		
Starch/bread	-	
Milk	-	60 ml skim milk
Coffee/tea	-	1 cup
Lunch		
Starch/bread	3	1 medium bowl rice or alternative
Meat	3	90g fish/lean meat/chicken*
Vegetable	1	1 exchange or more of leafy vegetables
Fruit	1	1 apple or alternative
Fat	2	2 tsp oil for cooking
Mid-afternoon snack		
Starch/bread	-	
Fruit	1	1 slice papaya or alternative
Coffee/tea	-	1 cup
Milk		
Dinner		
Clear soup		as desired
Starch/bread	3	1 medium bowl rice or alternative
Meat	3	90g fish/lean meat/chicken*
Vegetable	1	1 exchange or more of leafy vegetables
Fruit	1	1 orange or alternative
Fat	2	2 tsp oil for cooking
Bedtime snack		
Starch/bread	-	
Milk	1	240ml (1 cup) skim milk

Note:

*90g meat is size of 1 small taukua.
*90g chicken is size of 1 chicken drumstick.
*90g fish is size of 1 medium selar/kembong

Table 18a. Distribution of food exchanges for 2000 kcals

MEAL	NUMBER OF FOOD EXCHANGES					
	Starch/ bread	Meat	Vegetables	Fruit	Milk	Fat
Breakfast	3	1	-	-	-	1
Mid-morning snack	-	-	-	-	-	-
Lunch	4	3	1	1	-	2
Mid-afternoon snack	-	-	-	1	-	-
Dinner	4	4	1	1	-	2
Bedtime snack	1	-	-	-	1	-
Total	12	8	2	3	1	5

Table 18b. Menu plan for 2000 kcals

Exchange	No of exchanges	Food portion
Breakfast		
Starch/bread	3	3 slices wholemeal bread or alternative
Meat	1	1 slice lean ham or alternative
Milk	-	60 ml skim milk
Fat	1	1 tsp margarine or alternative
Coffee/tea	-	1 cup
Mid-morning snack		
Starch/bread	-	
Milk	-	60 ml skim milk
Coffee/tea	-	1 cup
Lunch		
Starch/bread	4	3/4 big bowl rice or alternative
Meat	3	90g fish/lean meat/chicken
Vegetable	1	1 exchange or more of leafy vegetables
Fruit	1	1 apple or alternative
Fat	2	2 tsp oil for cooking
Mid-afternoon snack		
Starch/bread	-	
Fruit	1	1 slice papaya or alternative
Coffee/tea	-	1 cup
Milk		
Dinner		
Clear soup		as desired
Starch/bread	4	3/4 big bowl rice or alternative
Meat	4	120g fish/lean meat/chicken
Vegetable	1	1 exchange or more of leafy vegetables
Fruit	1	1 orange or alternative
Fat	2	2 tsp oil for cooking
Bedtime snack		
Starch/bread	1	
Milk	1	240ml (1 cup) skim milk

Note:

*90g meat is size of 1 small taukua.
*90g chicken is size of 1 chicken drumstick.
*90g fish is size of 1 medium selar/kembong

Food exchanges

A food exchange consists of a list of foods within the group which have the same nutrient of similar quantity. Every food on a food exchange list has roughly the same amount of carbohydrate, protein, fat and calories. For example, each item on the starch/bread exchange list has 15g carbohydrate, 3g protein, trace fat and provides 80 calories of energy. Any food on the list can therefore be exchanged or "traded" for any other food on the same list.

The American Diabetic Association and the American Dietetic Association have jointly developed a system of six food exchanges. Table 14 shows the amount of carbohydrate (CHO), protein (PRO), fat (FAT) and kilocalories (kCal) in one exchange from each exchange list. Household measures simplify the measurement of food quantities. Table 15 shows the quantities of commonly used household measures used locally. Locally eaten food items in the six food exchange lists are shown in the Appendix to this paper. The number of exchanges may be calculated once the total daily energy requirements of the individual is known. Table 16 shows the food exchanges for total daily energy requirements of 1200 kcal, 1500 kcal, 1800 kcal, 2000 kcal and 2200 kcal, while examples of meal plans for total daily energy requirements of 1500 kcal and 2000 kcal are shown in Tables 17 and 18, respectively.

References

Singapore Dietitian Association. Position Statement. Dietary Recommendations for Individuals with Diabetes Mellitus. The Singapore Dietitian 1988, 3;3:4-8

Gourley, HP Lee. Dietary Fibre - its Components in some Southeast Asian Foods

McCance & Widdowsons. The composition of foods, 1988. London: Her Majesty's Stationery Office

Tee ES. Nutrient composition of Malaysian foods. Kuala Lumpur: Nutrient Sub-committee on protein food habits research and development, Malaysia, 1988

American Diabetic Association & American Dietitic Association. Exchange Lists. (Table *on exchange lists*)

Appendix – Food Exchange Lists

I. STARCH/BREAD EXCHANGE LIST
Each item below contains 15 gm carbohydrate and 80 Calories

Food Item	*Description*	*Portion*
BREAD AND ALTERNATIVES		
Bread	white/wholemeal	1 slice
	rye/french	1 slice
	pita	1/2 no
Bread sticks		2 no
Bun	Burger/Frankfurt	1/2 no
Roll	plain, small	1 no
Muffin	English	1/2 no
Biscuits	Ryvita	3 pc
	cream cracker	3 pc
	krackermeal	3 pc
	small cracker	6 pc
	digestive	1 pc
	marie	3 pc
Bran cereals	flaked/all bran	1/2 mb
Cornflakes		3/4 mb
Oatmeal	rolled/instant, cooked	1/2 mb
Pancake	plain	2 no
Popcorn	popped, no sugar/fat added	3 mb
Rice Krispies		3/4 mb
Waffle		1 no
Wheatgerm		2 tbsp
RICE AND ALTERNATIVES		
Rice	white/unpolished, cooked	1/3 mb
Rice-porridge	thin	1 mb
	thick	1/2 mb
Noodles		1/3 mb
Macaroni/sphaghetti	cooked	1/3 mb
Chappati	no fat	1 sm
Idli		1 sm
Dosai		1 pc
STARCHY VEGETABLES		
Corn		1/2 mb
Corn-on-cob	small	1 no
Green peas	canned/frozen	1/2 mb
Potato	small – 100 g	1 no
Sweet potato	small – 120 g	1/2 no
Pumpkin	cooked	1/2 mb
Water chestnut		4 no
Yam	cooked	1/3 mb
LEGUMES/PULSES		
Beans/peas/dhall	cooked	1/3 mb
Baked beans	tinned	1/4 mb
FLOUR		
Glutinous rice flour		2 tbsp
Rice flour		2 tbsp
Corn flour		2 tbsp
Tapioca flour/arrowroot		2 tbsp
Wheat flour		2 tbsp

Source: Tee ES. Nutrient composition of Malaysian foods, 1988. Direct weighing by TSE.
Abbreviation: no = number; pc = piece; tbsp = tablespoon; mb = medium bowl

II. MEAT EXCHANGE LIST

Food Item	*Description*	*Portion*
Lean meat: Each item below contains 7 gm protein and 55 Calories		
Beef	Eye round, sirloin, flank, tenderloin	30g
Pork	Lean pork, lean ham, tenderloin	30g
Mutton	Lean meat	30g
Poultry	Chicken/turkey (no skin and fat)	30g
Fish		30g
Fish	Tuna in water	1/4 mb
Frog's legs		2 medium
Oyster		6 no
River snails		10 no
Prawns, crabs, lobster, scallop		60g
Egg	White	3 no
Bean product	Taukua	1 pc
	Tofu big	1/2 pc
Cheese	Cottage cheese	1/4 mb
Meat with medium fat: Each item below contains 7 gm protein and 75 Calories		
Beef	Ground, rib, chuck, rump, steak	30g
Pork	Ground, spare ribs, chop	30g
Lamb	Chop, leg or roast	30g
Poultry	Chicken with skin	30g
	Duck with no skin/fat	30g
Organ meat	Heart, liver, kidney	30g
	Intestine, brain, tongue	30g
Fish	Tuna in oil	30g
Egg	Whole, duck/hen	1 no
Cheese	Mozarella (low fat) – in pizza	30g
Meat with high fat: Each item below contains 7 gm protein and 100 Calories These items should be used only occasionally		
Beef	Corned beef, ribs	30g
Pork	ground pork, spare ribs	45g
Sausages		30g
Meat	canned e.g., pork luncheon meat	30g
Fish	fish cakes, balls, fried	30g
Cheese	Cheddar, Swiss	30g
Peanut	Peanut butter	1 tbsp
Bean	Soya bean curd, fried 2 pc	30g

Sources: Tee ES. Nutrient composition of Malaysian foods, 1988;
American Diabetic Association & American Dietetic Association. Vegetable List. in: Exchange Lists, 1992.
Notes: 30 gm meat or fish is about one-third piece of small bean curd (Taukua) or size of 1 match box.
Abbreviation: no = number; pc = piece; tbsp = tablespoon; mb = medium bowl.

III. VEGETABLE EXCHANGE LIST

VEGETABLES WITH SOME CALORIES

Each vegetable serving on this list contains about 5g carbohydrate and 25 Calories
Unless otherwise stated, the serving size for vegetables (one vegetable exchange) is:
1/2 cup of cooked vegetable or vegetable juice; 1 cup of raw vegetables

Asparagus
Beans
Bean sprouts
Beet
Broccoli spears
Carrot
Cauliflower
Egg plant
Lady's fingers
Leeks
Onions
Tomato
Turnip

FREE VEGETABLES

Each vegetable serving on this list contains less than 20 Calories per serving (raw, 1 cup)

Cabbage
Celery
Chilli – raw
Chinese cabbage
Green onion
Mushroom
Radish
Chives (*koo chai*)
Cucumber
Lettuce
Spinach
Pepper – green, raw
Water cress (*si yang chai*)

STARCHY VEGETABLES

Starchy vegetables such as corn, peas and potatoes are found on the Starch/bread list.

Sources: Tee ES. Nutrient composition of Malaysian foods, 1988;
American Diabetic Association & American Dietetic Association. Vegetable List. in: Exchange Lists, 1992.

IV. FRUIT EXCHANGE LIST

Food Item	*Description*	*Portion*
Each item below contains 15gm carbohydrate and 60 Calories		
Apple	medium	1 no
Apricot	small	4 no
Banana	5" long	1 no
Cempedak		2 no
Cherry		12 no
Chiku (Sapodilla)		1 no
Custard apple		1 no
Grapefruit		1/2 no
Date	fresh	2 no
Duku	small	8 no
Durian	Thai	1 seed
Grape	small	15 no
Grape	big	5 no
Guava	medium	1/2 no
Jackfruit		3 seeds
Kiwi	big	1 no
Honey melon	medium	1/8 slice
Orange, mandarin	medium	1 no
Mango	small	1/2 no
Longan	small	8 no
Lychee	medium	4 no
Mangosteen		3 no
Papaya		1 slice
Peach		1 no
Pear	small	1 no
Persimmon		1/2 no
Pineapple		1 slice
Plum		2 no
Prune	fresh	2 no
Prune	dried	3 no
Pomegranate		1/2 no
Rambutan		4 no
Raisins		2 tbsp
Strawberry	fresh	8 no
Soursop		1 slice
Starfruit	small	1 no
Watermelon		1 slice
Apple/Grapefruit/pineapple/orange juice		1/2 mb
Grape/prune juice		1/3 mb
Pomelo		2 seg

Sources: Tee ES. Nutrient composition of Malaysian foods, 1988; Direct weighing by TSE.
American Diabetic Association & American Dietetic Association. Vegetable List. in: Exchange Lists, 1992.
Abbreviation: no = number; pc = piece; tbsp = tablespoon; mb = medium bowl.

V. MILK EXCHANGE LIST

There are 3 types of milk, namely, full cream, low fat and skimmed milk. The Calories they yield depend on their fat content. *Include 1 to 2 serves of milk per day. Choose skim or low fat version.*

Food Item	*Serving*	*% Fat*	*Fat (g)*	*Calories*
Skim milk	240ml	0.1	trace	90
Skim milk powder	3 tbsp	0.1	trace	90
Low fat	240ml	2.0	5	120
Full cream	240ml	3.4	8	150

Source: Tee ES. Nutrient composition of Malaysian foods, 1988. Direct weighing by TSE.

VI. FAT EXCHANGE LIST

Each of the below contains 5 gm fat and 45 Calories

Fat exchange foods are usually allowed for cooking purposes e.g., with fish or meat and vegetables. *They should be limited to 2 to 5 serves per day.*

Food Item	*Description*	*Portion*
UNSATURATED FATS		
Margarine	polyunsaturated	1 tsp
Margarine	low calorie	1 tbsp
Mayonnaise	regular	1 tsp
Mayonnaise	low calorie	1 tbsp
Oil	corn, sesame, peanut, soybean, sunflower	1 tsp
Salad dressing	regular	1 tbsp
Salad dressing	low calorie	2 tbsp
Almonds	roasted	6 no
Cashews	roasted	1 tbsp
Peanuts	small	20 no
Peanuts	big	10 no
Peanut butter		2 tsp
Walnuts	whole	2 no
Pumpkin seeds		2 tsp
Other nuts/seeds		1 tbsp
SATURATED FATS		
Butter		1 tsp
Coconut shredded		2 tbsp
Cream	light	2 tbsp
Cream	heavy	1 tbsp
Coffee creamer		4 tsp

Source: Tee ES. Nutrient composition of Malaysian foods, 1988. Direct weighing by TSE.
Abbreviation: no = number; pc = piece; tbsp = tablespoon; mb = medium bowl.

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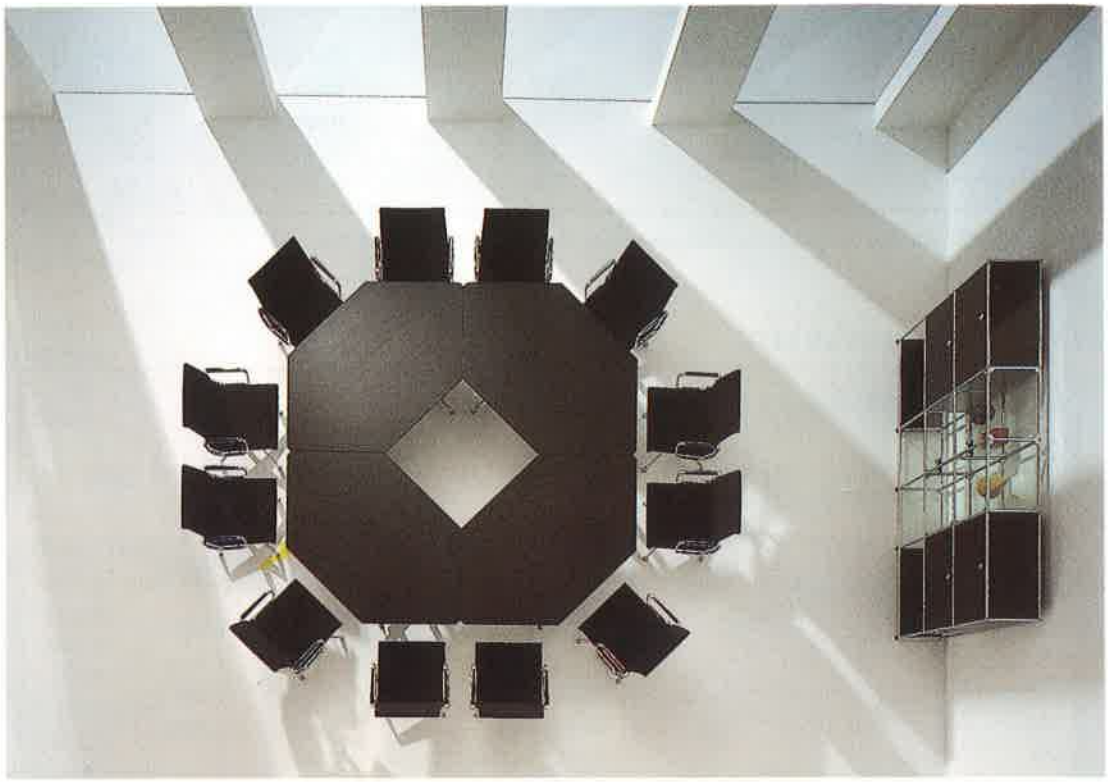

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DIABETES AND EXERCISE

Huan M W, MBBS (S), MCGP (S)

INTRODUCTION

Besides dietary control, regular physical activity and exercise form the other most important therapeutic modality in diabetes management.

PHYSIOLOGICAL EFFECTS OF EXERCISE

With exercise, the muscle takes up more glucose from the circulation. This glucose is replenished by increased production by the liver, and the plasma glucose level remains fairly constant. However, with short bouts of strenuous exercise, the blood glucose concentration may actually rise, while prolonged exercise can lead to a slight decrease (with prolonged exercise, free fatty acids mobilised from adipose tissue become another important source of energy for the muscle).

The increased hepatic glucose output and the mobilisation of free fatty acids both require a sufficient fall in circulating insulin with exercise. But if the insulin level drops too low, the increased glucose uptake by exercising muscle is not possible. Therefore, in patients with insulin-dependent diabetes mellitus (IDDM), the relative insulin deficiency can cause accelerated hyperglycaemia, lipolysis, and the rapid development of ketosis.

POTENTIAL BENEFITS (Table 1)

Regular physical exercise results in lower insulin levels (both basal and postprandial) and increased insulin sensitivity, thus improving long-term glycaemic control in patients with non-insulin-dependent diabetes mellitus (NIDDM), and lowered insulin requirements in patients with IDDM.

Exercise helps decrease some risk factors for cardiovascular disease (even in patients without diabetes), besides improving overall cardiovascular function. It can help reduce blood pressure in patients with mild to moderate hypertension, even without weight loss. Improvement in the lipid profile is another benefit – levels of serum triglycerides and very low density lipoprotein (VLDL) cholesterol are significantly decreased, low density lipoprotein (LDL) cholesterol is decreased, and high density lipoprotein (HDL) cholesterol is increased with intense regular physical training.

When combined with appropriate diet planning and moderate calorie reduction, regular exercise can make weight loss or weight maintenance easier, and help improve body composition, decreasing body fat while preserving or even increasing lean body tissue. Together with the increased physical work capacity and improved

Table 1. Potential Benefits of Exercise

1.	Lower plasma insulin levels
2.	Improved insulin sensitivity
3.	Decreased insulin resistance
4.	Cardiovascular conditioning
5.	Reduced blood pressure
6.	Improved lipid profile
7.	Weight reduction and control
8.	Improved physical fitness
9.	Improved sense of well-being

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physical fitness, the weight loss adds to an improved psychological state and quality of life, besides helping to reverse the insulin resistance that occurs with obesity.

RISKS

Exercise in patients with diabetes is not without risk. In particular, hypoglycaemia can occur in patients on insulin or oral hypoglycaemic drugs. Moreover, in IDDM patients, late-onset hypoglycaemia can also occur 6-15 hours after prolonged exercise is completed, especially if the exercise is sporadic. Conversely, exercise can result in hyperglycaemia if the exercise is very strenuous, but in patients with IDDM, even moderate-intensity exercise can cause hyperglycaemia and ketosis or ketoacidosis.

Cardiovascular disease – angina pectoris, myocardial infarction and arrhythmias – can be exacerbated (remember that ischaemic heart disease in diabetics is often silent) or precipitated by exercise. Blood pressure can rise excessively during exercise; orthostatic hypotension can occur after exercise (especially with impaired responses to dehydration caused by autonomic neuropathy). Also, weight-bearing exercise can worsen degenerative joint disease, which is itself more common in obese patients. Orthopaedic injuries and foot ulcers can occur, especially in the presence of neuropathy in the diabetic patient.

Exercise can also aggravate several long-term complications of diabetes. Retinal haemorrhage or detachment can be precipitated by jarring or rapid head movements. Exercises that increase blood pressure, e.g. heavy lifting and Valsalva-like manoeuvres, can be especially dangerous in patients with active proliferative retinopathy (retinal or vitreous haemorrhage can result), hypertension and ischaemic heart disease.

PRESCRIPTIONS FOR EXERCISE

1. **Screening**
   All patients with diabetes, before starting on any exercise program, must have a full history taken and undergo a complete physical (especially ophthalmological and neurological) examination, especially to search for cardiac, vascular and neurological complications. Exercise-stress ECG is recommended in patients over 35 years old, to help identify silent ischaemic heart disease, exaggerated hypertensive response to exercise or post-exercise orthostatic hypotension. Urine-testing for microalbuminuria should be included in the assessment of renal function.

2. **Selection of exercise types**
   If any complications or abnormalities are discovered in this process, the patient must be advised to avoid any exercises that have potential risk of worsening these complications. Otherwise, the patient can select any exercise type and intensity he personally prefers. Generally, the best regimen for these patients is low-impact, moderate-intensity, aerobic exercises, such as walking, cycling and swimming.

3. **Exercise sessions**
   Each session must commence with warm-up and stretching exercises for 5-10 minutes, proceed on to moderate-intensity exercises (50-75% of maximum aerobic capacity), for 20 to 45 minutes, and end with a cool-down period of at least 5-10 minutes of exercise at about 25-30% of maximum aerobic capacity.

   Diabetics need to exercise three to five times a week on a long-term basis to achieve all the potential benefits. Benefits of exercise are lost when no exercise is performed for a three-day period. Those attempting to lose weight may need to exercise five days or more a week.

4. **Patient education**
   All patients should be educated, particularly about:
   - carrying/wearing a card or bracelet identifying them as diabetics
   - the risk of post-exercise hypoglycaemia (both immediate and late-onset), and the need to carry glucose tablets or some other short-acting carbohydrate with them
   - avoiding dehydration during exercise by taking adequate fluids, and
   - regular and frequent blood glucose self-monitoring to document responses to exercise and

to plan safe exercise sessions, especially in patients with IDDM.

Special instructions for patients with either IDDM or NIDDM are given below.

Compliance with a prescription for an exercise program is usually increased if the patient is allowed to choose activities he enjoys and timing convenient to him. It can be further enhanced by participation in group exercise activities and by reinforcement by the patient's family and colleagues. Measurement of progress and positive feedback by the patient, his family and his doctor are all plus points for better motivation and compliance.

EXERCISE INFORMATION FOR PATIENTS WITH IDDM

1. Blood glucose levels will fall with exercise of moderate intensity and long duration, while vigorous exercise of short duration can cause blood glucose to rise.

2. It is important to have overall good blood glucose control before exercise. If blood glucose is above 250 mg/dl and ketones are present, exercise will probably worsen control, and is best avoided until ketones are no longer present, or when blood glucose is between 100 and 250 mg/dl.

3. Eat a meal one to three hours before exercise.

4. It is essential to monitor blood glucose levels before, during and after exercise to document and learn your own response patterns to different types of exercise and to prevent hypoglycaemia and hyperglycaemia with exercise.

5. If pre-exercise blood glucose is less than 100 mg/dl, eat a small snack just before exercise.

6. During vigorous exercise of long duration, take supplemental snacks containing 15-25 gm rapidly-absorbed carbohydrate every 30 to 60 minutes to maintain normal blood glucose levels.

7. Take insulin at least one hour before exercise. If it has to be taken less than one hour before exercise, inject into a non-exercising area of the body.

8. Reduce insulin dosages before exercise. The short-acting insulin dose in those on multiple-dose regime may be reduced by 20-50% before prolonged exercise of more than 45 to 60 minutes, and the post-exercise doses may be adjusted based on results of blood glucose monitoring.

9. Alter daily insulin schedule:

(a) Those on a single morning dose of intermediate-acting insulin may decrease the dose by 30-35%, or take 65% of the usual dose in the morning and the other 35% of the dose before the evening meal.

(b) Those on intermediate- and short-acting insulin combinations may reduce the pre-exercise dose of short-acting insulin by 50% or omit it altogether; they may also decrease the pre-exercise dose of intermediate-acting insulin and take supplement doses of short-acting insulin later if required.

EXERCISE INFORMATION FOR PATIENTS WITH NIDDM

1. Exercise can result in an increased appetite. Care must be taken not to increase food or calorie intake.

2. Patients with blood glucose values of less than 200 mg/dl have a greater glycaemic benefit from exercise than those with higher blood glucose levels.

3. Patients taking sulphonylureas and who have good glycaemic control may need to reduce or omit the dose of medication before exercising, depending on blood glucose monitoring results.

4. In patients on diet control alone, supplemental snacks before, during or after exercise are not necessary unless the exercise is unusually strenuous or prolonged.

5. In patients treated with very-low-calorie diets (600-800 kcal/day), the diet should contain at least 35% of calories as carbohydrate to maintain normal muscle glycogen stores so as to prevent hypoglycaemia during high-intensity or prolonged exercise sessions.

CONCLUSION

Regular physical exercise is recommended as an important component of the treatment of all persons with diabetes. Patients themselves tend to underestimate its beneficial effects, and busy doctors spend scant time pushing its importance or discussing it in detail.

Exercise should be fun and be enjoyed. Increasing normal daily activities, (e.g. walking, climbing stairs instead of taking elevators, walking longer distances to and from work or home) is an excellent start. More regular programs are useful and safe prescriptions if they are individualised, depending on the patient's personal preferences, motivation, age and physical condition. Eventually the rewards of exercise will present themselves, and not exercising will become unthinkable.

ORAL HYPOGLYCAEMIC AGENTS

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INTRODUCTION

The majority of the Asian diabetic population has *Type II* diabetes (Non-Insulin-Dependent Diabetes or NIDDM). Patients with Type II diabetes produce sufficient endogenous circulating insulin to prevent ketoacidosis, but their insulin response is either lower than normal or insufficient to meet the physiological demand produced by reduced insulin sensitivity.

The aim of diabetes management is the maintenance of good glycaemic control, thus preventing or minimising the likelihood of acute and long-term end-organ complications. While diet control remains a cornerstone of successful management, and weight loss the primary therapy for obese Type II diabetics, oral hypoglycaemic agents (OHAs) are indicated where:

- at diagnosis, symptoms are marked and blood glucose readings are 20 mmol/lit (360 mg/dl) or higher, and
- despite genuine attempts with a diet and exercise program, hyperglycaemia persists.

Contra-indications to oral hypoglycaemic therapy include:

- type I diabetes
- diabetes in pregnancy and lactation
- hepatic and renal dysfunction
- cardiovascular insufficiency, and
- severe concurrent illness (e.g. sepsis, trauma, etc)

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GUIDELINES FOR THE FAMILY PHYSICIAN

1. OHAs are most likely to be effective in type II diabetic patients with:

- a disease onset later than age 40,
- a disease duration of 5 years or less,
- above ideal body weight,
- fasting blood sugar levels of below 8 mmol/lit (145 mg/dl) or 2-hour post-prandial blood sugar levels of less than 12 mmol/lit (215 mg/dl), despite an adequate trial of diet alone,
- no history of ketoacidosis, and
- those who have either never received insulin or have required a regimen of less than 20-30 units per day for good control.

2. Before starting with OHAs, it must be emphasised to the patient that:

- drugs are only adjuncts to diet
- there is a 10-20% chance of primary failure (i.e. adequate control is never achieved) with their use
- there is a cumulative 5-10% per year secondary failure rate (i.e. loss of control after initial successful treatment for at least one month), and
- insulin may be required in the future, either permanently (with or without the OHA) or temporarily (under conditions of stress).

3. Characteristics of OHAs to be considered when choosing one for a given patient:

- spectrum of actions
- duration of action/number of doses per day
- potency in the therapeutic range

- side-effects
- metabolism
- drug interactions
- cost

4 The minimum effective dose should be used initially, and this gradually increased on a weekly basis until satisfactory glycaemic control is achieved.

5 If good blood glucose control cannot be achieved and maintained by treatment with OHAs alone, then insulin **must** be used.

TYPES OF OHAs

1. Sulphonylureas

Sulphonylureas act mainly by stimulating secretion of insulin from pancreatic ß-cells. However, they also increase the sensitivity of these ß-cells to glucose, increase the number of insulin receptors, increase the sensitivy of insulin receptors in liver, fat and muscles, and reduce insulin resistance at post-receptor sites.

All sulphonylureas should be given prior to meals. If one of these agents is ineffective at full dosage, changing to another may be tried but is usually not successful. There is no point in using two sulphonylureas, but addition of metformin (a bignanide) to a sulphonylurea may be useful at this stage.

Table 1. Adverse effects of sulphonylureas

Hypoglycaemic
Weight gain
Gastrointestinal disturbance
Cholestasis
Pruritus
Erythema multiforme
Blood dyscrasia
Hyponatraemia (Chlorpropamide)
Drug interactions

Increased appetite and hypoglycaemia are common side-effects. Hypoglycaemia is common and, in the elderly often unrecognised, especially with the stronger or longer-acting sulphonylureas. Increased appetite and tendency to hypoglycaemia may result in moderate weight gain. Another common problem is the use of sulphonylureas to achieve normoglycaemia in the face of dietary non-compliance, resulting in increasing obesity. Other side-effects are rare but gastrointestinal and cutaneous reactions may occur occasionally. *(Table 1)*.

Recommended sulphonylureas (Table 2) are:

- Glibenclamide – Long-acting potent, second generation OHA. Dose range of 1.25 to 15 mg daily; can be given once or twice daily.

- Gliclazide – Short-acting, potent, third generation OHA. There are some reports of beneficial effect on fibrinolysis and platelet adhesiveness. Dose range of 40 to 320 mg daily given once or twice daily.

- Glipizide – Short-acting, potent, third generation OHA. Dose range of 2.5 to 40 mg daily; given once or twice daily. Excretion chiefly through the urine.

Older, less used sulphonylureas are:

- Tolbutamide – Weaker, short-acting OHA. Dose range of 0.5 to 3 gm; given two to three times daily. Useful in older patients, especially in those with renal impairment.

- Chlorpropamide – Potent OHA with a prolonged duration of action. Dose range of 125 to 500 mg; given once daily. In fact may last several days; hypoglycaemia may occur, and it should be used with caution in the elderly and in the presence of cardiac, renal or hepatic disease. Alcohol-flushing also occurs and appears to be genetically determined. Side-effects as for other sulphonylureas, but in addition cholestatic.

Table 2. The Sulphonylureas

Chemical Name	Tolbutamide	Chlorpropamide	Glibenclamide	Gliclazide	Glipizide
Brand name	Rastinon	Diabenese	Danoil, Euglucon	Diamicron	Minidiab
Tablet size	500mg	250mg	5mg	80mg	5mg
Duration of action	6-12 hrs	24-72 hrs	12-24 hrs	12-24 hrs	12-24 hrs
Daily dosage range	0.5-3.0mg	125-500mg	1.25-20mg	40-320mg	2.5-40mg
Daily dose frequency	2-3	1	1-2	1-2	1-2
Metabolites	Inactive	Some activity	Inactive or weakly active	Little activity	Inactive
Excretion	Kidney	Kidney	Kidney 50% Bile 50%	Kidney	Kidney 80% Bile 20%
Comments	Shortest-acting	Side-effects more frequent	-	Dose > 160mg daily should be given in 2 divided doses according to the main meals of the day	Daily dose of > 15mg should be divided into 2 doses according to the main meals of the day

Drug Interactions with sulphonylureas

A number of drug interactions may occur between sulphonylureas and other agents *(Table 3)*.

II Biguanides

Metformin, the only biguanide currently in use, acts by:

- delaying intestinal glucose absorption
- decreasing insulin resistance at post-receptor sites
- increasing peripheral glucose uptake
- suppressing basal hepatic glucose output, and
- decreasing gluconeogenesis

It is used either alone (especially in the obese, where it is often the drug of choice) or in combination with a sulphonylurea. The usual dose range is 0.25 to 0.5g twice daily, with a maximum of 3g daily, administered in three divided doses, given with or after meals to minimise side-effects.

Gastro-intestinal disturbance like anorexia, nausea and diarrhoea are fairly common side-effects, and are usually dose-dependent. (The mild anorectic effect is useful in the obese patient). Vitamin B12 malabsorption and a metallic taste in the mouth occur rarely. Lactic acidosis is an uncommon but potentially dangerous side-effect, particularly in the elderly or in those with renal or hepatic disease or in the presence of hypoxia from cardiac or respiratory failure. Long-term use of metformin should be accompanied by periodic check on renal and hepatic function.

SECONDARY OHA FAILURE

Causes: Initially achieved glycaemic control may be subsequently lost, even with maximal doses of OHAs, due to many factors, which may be related to the disease, the therapy or the patient.

Table 3. Interactions of Sulphonylureas

Agent	Action	Nett reaction
Aspirin, fibrates, trimethoprim	Displace sulphonylurea from albumin binding sites	Increase hypoglycaemia
Alcohol, H2 blockers, anti-coagulants	Competitive inhibitors of sulphonylurea metabolism	
Probenicid, allopurinol	Inhibit urinary excretion of sulphonylureas	
Alcohol, aspirin	Drugs with intrinsic hypoglycaemic properties	
ß-blockers, sympatholytic drugs	Antagonists of endogenous counter-regulatory hormones	
Barbiturates, rifampicin	Increase sulphonylurea metabolism	Worsen glycaemic control
ß-blockers	Antagonise action of sulphonylureas	
Thiazides and loop diuretics, ß-blockers, corticosteroids, oestrogen, phenytoin	Inhibit insulin secretion or action	

1 *Disease-related factors:*

- Decreasing ß-cell function
- Increasing insulin resistance

2 *Therapy-related factors:*

- Poor or inadequate patient education
- Inadequate drug dosage
- De-sensitisation of ß-cells to chronic continuous sulphonylurea exposure ("sulphonylurea resistance"). It has been shown that ß-cell sensitivity can be restored by discontinuation of sulphonylurea medication for a period, implying that intermittent administration of these agents when secondary failure is first detected may be a therapeutic option worthy of consideration.
- Impaired absorption of drugs due to hyperglycaemia
- Concommitant therapy with other drugs – those that directly cause hyperglycaemia (e.g. steroids, thiazides) and those that decrease levels or actions of sulphonylureas (e.g. ß-blockers, barbiturates).

3 *Patient-related factors:*

- Dietary non-adherence and weight gain
- Failure to increase the level of physical activity
- Poor patient compliance with regular medications
- Emotional stress. This may cause poor control by reducing self-care and dietary adherence, and also a direct metabolic effect.
- Intercurrent illnesses, such as infections and thyrotoxicosis.

Insulin treatment

Insulin treatment remains the mainstay of management of secondary failure. The insulin is either administered alone, or in combination with OHAs. It reverses many of the metabolic derangements present in secondary failure, and improves glycaemic control.

Insulin alone. Often, a small dose of up to 40 units of once-daily injection of intermediate- or long-acting insulin is sufficient to achieve glycaemic control. However, where endogenous insulin reserves (as measured by C-peptide levels) are low or absent, twice daily or multiple injections regimens similar to those recommended for type I patients will be necessary. Reversion to oral therapy may be possible after a trial of insulin for a few months; in other cases insulin may be

required on a permanent basis.

Combination therapy. The rationale for the use of combined insulin-sulphonylurea therapy lies in the different and possibly synergistic effects of the two drugs – the insulin corrects the basal hypoinsulinaemia, while the sulphonylurea simultaneously stimulates endogenous insulin secretion and improves peripheral insulin sensitivity; in this way, smaller doses of insulin are required and used, and the development of hyperinsulinaemia (and consequent insulin resistance and atherosclerosis) avoided. Combining metformin and insulin does not appear to have the same effect.

Residual ß-cell function and endogenous insulin reserve is a pre-requisite for the success of this form of therapy, and the type II patients most likely to benefit from combination therapy are the obese ones requiring over one unit of insulin per kilogram of body weight for glycaemic control.

Addition of a low night-time dose (to reduce gluconeogenesis) of NPH (isophane insulin suspension) or IZS Lente to a maximal dose of sulphonylurea is often advised. In practice, however, a daily morning injection works just as well and appears more convenient and accepted.

CONTINUING CARE IN DIABETES

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INTRODUCTION

Long-term management of diabetes has as its aims good biochemical control, prevention and early diagnosis of acute and long-term complications and hence reduction of mortality, and treatment of accompanying disorders. This requires the cooperative efforts of the doctor, the patient and the patient's family, and can only be achieved by proper education and motivation of the patient and his/her family, monitoring of control, and regular counselling and reviews.

EDUCATION

This is a vital part of the management of diabetes. At the time of diagnosis of diabetes, the usual accompanying emotional shock makes it difficult for effective learning to occur. In-depth education of the patient and the patient's family may have to be delayed a little, till after they have accepted the fact that they have to live with diabetes, and are more receptive of the "survival skills" required.

Diet, exercise and weight control are the most important factors in the management of non-insulin dependent diabetes mellitus (NIDDM). Besides these, the patient will have to be educated on the treatment regime (tablets and/or insulin injections), adaptation to travelling and illness (sick days), footcare, and the possible complications of diabetes or treatment.

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Diet in Diabetes

Dietary compliance remains the biggest problem in management, and proper dietary advice is essential, often requiring the services of a dietitian. Basically the diabetic diet is a normal healthy diet and recommends three regular meals (meals should not be missed), with complex carbohydrate foods high in fibre forming the basis of each meal, and avoidance or minimal intake of simple carbohydrates and sugars, fats and alcohol.

The individual patient will require his own diet prescription according to his activity pattern, ethnic group, culture etc. Total calorie intake will have to be restricted, more so for the overweight or obese diabetic, for whom achieving weight reduction is a very important goal. Flexibility of diet in NIDDM is possible to a much greater extent than with IDDM; patients with IDDM, especially young diabetics who snack between meals will require more thorough dietary education. The goal in nutrition education is to promote long-term behaviour change in a patient with established eating patterns. As such, this education must be a continuing process.

Diabetics and Exercise

Regular physical exercise leads to improved physical fitness and psychological state, and loss of body fat and weight, with resultant improved general health. In the diabetic, physical exercise has additional benefits of increased insulin sensitivity, improved glucose tolerance, lower blood pressure, and favourable changes in the lipid pattern with decreased risk of atherosclerosis, which occurs with increased frequency in the diabetic population. Regular

physical exercise can also allow a reduction in dosage of injected insulin, and can help reverse the resistance to insulin that occurs as a result of obesity.

The diabetic patient, especially if he has never exercised before, must be screened for vascular and neurological illness or complications, including silent ischaemic heart disease (stress ECG recommended in patients over 35 years of age), and must be carefully advised on the type, intensity, frequency and duration of recommended exercise and how to monitor his blood sugar and avoid complications, especially in those with IDDM.

Target Weight

Diet and exercise will help the diabetic lose weight. Goals for weight loss (both short-term and long-term) must be individualised for each patient, and the target weight may be higher than the published "desirable" weight range for that patient's sex, height and body frame. This "reasonable" target weight is one which can be achieved and maintained by the patient with diabetes. Losing even 10-20 pounds may dramatically improve glucose tolerance, and seeing his blood sugar levels drop will encourage the patient to shed further weight by diet and exercise.

Psycho-social aspects of Diabetes Management

The family doctor will be best able to reassure and help the patient with his reactions to the illness and with his daily problems of living as a diabetic, who will also need guidance on necessary life-style changes (including cessation of smoking) and advice on planning for pregnancy etc. Getting the patient to join the Diabetic Society will help him with another resource for information and support.

MOTIVATION

The process of making life-style changes, taking tablets or insulin injections regularly, and monitoring biochemical control requires active participation by the patient. This can be ensured only by the establishment of a supportive working relationship between the doctor and the patient, and will be enhanced by support from the patient's family.

The management plan has to be individualised. The patient has to be educated on his illness so as to understand and assist in the setting of goals and to adhere to the management plan, keep records of blood sugar levels etc., provide the doctor with feedback and attend regular follow-up. One method to consider is the use of contract, setting as a goal one or two objectives that the patient wants to achieve.

Factors that naturally influence adherence are age, education, socio-economic level, race, religion and the belief system of the patient. The type of treatment regimen instituted must be carefully chosen – its frequency and complexity of schedule, cost, possible side-effects and interference with the patient's life-style – so that it can be adhered to by the patient. Patient compliance with medications requires careful explanation on the need for medications and the choice of medication; also, the patient's concerns about taking medications long term must be addressed by the doctor.

MONITORING CONTROL

Regular monitoring of metabolic control is required to relieve symptoms and to make necessary adjustments to treatment so as to prevent or delay complications and improve quality of life. Good control is critical in situations of rapid growth e.g., puberty and pregnancy.

Urine glucose tests

Although not as reliable as blood glucose tests, these are cheap, convenient and easy to perform. They can be carried out either several times a day or once a day but at different times each day. Disadvantages include failure to indicate hypoglycaemia and their total unreliability in patients (especially the elderly) with high renal thresholds.

Blood glucose monitoring

The frequent estimation of blood glucose is the most important parameter for monitoring diabetic

control. This is easily done by the patient himself with blood glucose meters (the recommended mode of self-monitoring) or with visually-read test strips, and is much more accurate than urine sugar testing. Although more expensive than urine testing, Self Blood Glucose Monitoring (SBGM) with the patient in his normal work or home environment is the best method of assessing glucose control, the results being used to improve control, to adjust food intake and also to adjust insulin doses.

Indications for SBGM are listed in Table 1. Advantages of SBGM (Table 2) far outweigh the disadvantages of being expensive, time-consuming and painful.

Table 1. Indications for SBGM

- Poorly controlled diabetes
- Frequent hypoglycaemic attacks
- Young children
- Pregnancy
- Altered renal threshold (unreliable urine tests)
- Obesity in NIDDM
- Complications of diabetes
- On dialysis
- Colour blindness

The frequency of blood glucose tests is determined by stability of control, method of insulin delivery, the patient's own preference and financial considerations. For NIDDM on diet or oral hypoglycaemic agents, three to four tests a day (fasting and post-prandial) twice a week is recommended. For IDDM or NIDDM requiring insulin, four tests a day (before meals and at bedtime), two to three days a week with an occasional test at 3 A.M., and during episodes of "Hypos" is advised, with increased frequency of testing in case of change in routine, change of insulin requirements, or illness. Accuracy of test results depends on testing technique and proper handling and maintenance of all the equipment.

Table 2. Advantages of SBGM

- Provides correct measurement of blood glucose level
- Establishes renal threshold
- Allows adjustment of treatment while maintaining normal lifestyle (away from hospital)
- Facilitates detection of "hypos"
- Facilitates detection of symptoms not associated with "hypos"
- Offers direct feedback when "Hyper" can be traced to increased eating pattern.
- Use as an educational tool providing visual proof of "Hyper"
- In renal diseases where urine samples are difficult or cannot be obtained
- A way of gaining knowledge of the action, duration and peak effect of insulin

Targets for Control

The targets to be set will be decided between the doctor and the patient, and will vary with the individual, depending on his age and other factors. A useful guide is given in Table 3.

Table 3. Targets for control of diabetes

	Ideal	Good	Fair	Poor
Fasting blood glucose	3.5-6.7mmol/L (60-120mg/dl)	<7.8mmol/L (<140mg/dl)	<11mmol/L (<200mg/dl)	>11mmol/L (>200mg/dl)
Post-prandial blood glucose	<7.8mmol/L (<140mg/dl)	<11mmol/L (<200mg/dl)	<12.8mmol/L (<230mg/dl)	>12.8mmol/L (>230mg/dl)
Urine glucose	0	0	trace to +	> +
HbA1c (glycohaemoglobin)	<7%	7.8%	8-9.5%	>9.5%

(Reference ranges vary between laboratories, depending on the method used)

"Ideal control" should be the ultimate aim, especially during pregnancy. But it may be difficult, impossible or unnecessary in certain patients e.g. in the elderly, due to risk of hypoglycaemia and limited life expectancy. "Good control" are those levels at which the risk of diabetic complications is thought to be minimised.

MANAGEMENT PLAN

Regular reviews by the attending doctor (usually every three months – more frequent if diabetes is not well controlled) will help determine progress, or lack of it, in meeting the set goals and to identify problems. The following areas will need special review:

History

- smoking habits, adequacy of exercise, alcohol use, weight control, nutritional evaluation, adherence to all aspects of self-care, and psychosocial adjustment.
- symptoms of hypoglycaemia and/or hyperglycaemia
- symptoms relating to eyes, circulation, feet and infections
- current medications, any adjustments of therapeutic regime by the patient

Examination

- weight (and height and sexual maturation staging in children), blood pressure, heart, carotid and peripheral pulses
- visual acuity, eyes, and optic fundi (at least once a year)
- tendon reflexes and sensation (at least once a year)
- condition of feet (skin, pulses, neuropathy)
- insulin injection sites

Laboratory evaluation

- SBGM data provided by the patient
- Blood glucose, HbA1c
- Urine protein
- *Fasting Lipid profile
- *Routine urinanalysis, including microalbumin
- *Renal function
- *ECG

* at least once a year

The patient should have an annual comprehensive physical examination to screen for complications, including impotence. Regular ocular review is very important as the early, easily treatable stages of vision-threatening retinopathy are often without symptoms and have to be looked for.

These regular reviews should be followed by continuation of patient education, review of therapeutic regime, any necessary health recommendation and referral to appropriate specialists, including the dietitian and podiatrist for a team approach to continuing care in diabetes.

WHY RESEARCH AND WHAT RESEARCH

Emmanuel S C , MBBS (S'pore), MSc (Public Health), AM

I WHY RESEARCH AND WHAT RESEARCH?

Research is a quest for knowledge, through diligent search or investigation or experimentation, aimed at the discovery and interpretation of new knowledge. Research and scientific methods in health may be considered a course of critical inquiry, leading to the discovery of fact or information which increases our undertstanding of health and disease.

Research should be an important component of care provided by General Practitioners for the following reasons:

(i) Many years ago, medical knowledge was more general and was based on good sound basic principles of medicine. We have come a long way since, in terms of new knowledge, advances in care and medical technology which have revolutionised the management of diseases.

Absence of research brings about a lethargy in practice methods and perpetuates outdated information and knowledge and diminishes interest in one's career and profession.

(ii) With a better informed population and advances in medical knowledge and treatment, research will provide you with the information to meet the rising expectations of your patients. With increasing awareness and litigation by patients, it is important that general practitioners try to remain in the forefront of medicine.

(iii) Furthermore, the physician's healing touch today needs to be tempered by a consideration of other important dimensions pertaining to the patient, such as spiralling healthcare costs, quality of life for the patient after treatment, available resources, responsibility to the patient, his family and to society.

(iv) To maintain an edge and competitiveness, we need to move on to innovative approaches in the care we deliver and participate in activities which have a creative and inquiring slant to derive better and more cost-effective methods of healthcare and medical management, relevant to our patients, to improve their health and the quality of medical care provided.

(v) Your practice supports research by providiing a body of good and relevant information such as patients, facilities etc.

(vi) Research into, say, the leading conditions for which ambulatory care is being sought in your practice and biographic characteristics of these parties will enable you to

- focus the investigation and management of the conditions to the highest level of sophistication possible at outpatient level.

This will ensure that your patients re-

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ceive the best and most complete care possible at the ambulatory level and in so doing cut down on referrals to secondary and tertiary institutions for management. This is in line with the preferred wishes of most patients.

- Prioritise your resources and facilities to develop care for these patients to the fullest.

- Study the risk factors to these conditions so that you can give appropriate health education and treatment for these conditions to help prevent and control them.

(vi) Although you may feel your knowledge and skills in research are limited, you are uniquely qualified to select questions related to your practice. Answers to these questions will add bit by bit to medical knowledge, particularly pertaining to medical care as delivered by General Practitioners, strengthen the specialty of family medicine and enhance the quality of medical care provided by family physicians.

II THE RESEARCH QUESTION AND HOW TO FORMULATE IT

We need to demystify research and realise that there are several areas in which you, as family physicians, are eminently qualified and have a unique perspective of your practice and can carry out research which is interesting and relevant to your practice and your patients.

General Practitioners who aspire to conduct research in practice may suffer from the problem of having many research interests arising out of natural and active curiosity. Also, many of your areas of interest may not be sufficiently focussed. You should therefore articulate your needs and arrange your research such that it meets your needs. At the end, the findings of your research should be translated into a better service for your patients.

RESEARCH MUST START WITH A QUESTION

The practitioner should think creatively, begin planning and designing his research idea as well as discussing potential research projects with colleagues.

A CATERGORIES OF RESEARCH

1) Empirical and Theoretical Research

The philosophical approach to research is basically to two kinds: empirical and theoretical.

Health research mostly follows the empirical approach, i.e. it is based upon observation and experience more than theory and abstraction.

Empirical research in the health sciences necessarily involves quantifications for the most part, through 3 related numerical procedures :

a) measurement of variables

b) estimation of population parameters (the determination and comparision of rates, ratios, proportions, etc).

c) statistical testing of hypothesis or estimating the extent to which chance alone may account for our findings.

2) Basic and Applied Research

Research can be functionally divided into basic (pure) research and applied research.

a) Basic research is usually considered to involve a search for knowledge without a defined goal of utility or specific purpose.

b) Applied research is problem-orientated and is directed towards a defined and purposeful end; it is frequently generated by a perceived need and is directed towards the solution of an existing problem.

APPLIED RESEARCH to be of maximum benefit, should be directed at priority areas in healthcare comprising the following:

(i) Areas of maximum patient load, i.e. related

to leading causes of ill-health among your patients.

This will involve clinical or epidemiological research into the key diseases and their risk factors so that you can draw up more effective methods of disease prevention and control, improved diagnosis, treatment and rehabilitation of these patients. Examples could include clinical trials to identify better methods of treatment for these diseases or to answer such a question: can coronary attacks be better managed at home?

The role of adjuvant or alternative medicine, eg traditional Chinese medicine, in minimising the side-effects of treatment or in improving treatment through an adjuvant action, should also be explored.

(ii) Research into the elderly in view of their increasing numbers in the future, their special health needs and how best these needs can be met.

(iii) Studies of outcome assessments of new technologies and procedures, i.e. quality assurance.

Here you can study different methods and procedures to identify the most cost-effective methods and those with the best outcomes or effectiveness.

(iv) Health Systems Research i.e. research into your medical practice styles against the standard practice style e.g. referrals to hospitals and length of stay of these patients, issue of MCs. The aim is to evolve improved approach to service delivery, e.g. improved quality of service such as waiting time for medical care, appointment scheduling.

Other examples of Health Systems Research include fresh approaches in examining the role of the various healthcare workers in your practice and how to maximise each one's role so as to provide the most cost effective healthcare. The important aim here is to maximise the care provided at all levels of healthcare by all grades of health workers. Health service indicators can be derived from comparative studies.

B RESEARCH STRATEGIES AND DESIGN

The selection of a research strategy is the core of a research design and is probably the single most important decision the investigator has to make. The strategy must include definition of variables, their levels and their relationship to one another.

(i) Experimental versus Observational Strategies

Experimental studies are also called interventional studies.

Although an experiment is an important step in establishing causality, it is often neither feasible nor ethical to subject human beings to risk factors in etiological studies, Instead epidemiologists make use of "natural experiments" when available and they resort to analytical studies.

(ii) Descriptive Studies

Epidemiological description entails the description and comparison of disease occurrence and its distribution in population groups according to

- characteristics of persons;
- characteristics of place;
- characteristics of time; and
- characteristics of family.

When an epidemiological study is not structured formally as an analytical or experimental study, i.e. when it is not aimed specifically to test an etiological diagnosis, it is called a "descriptive" study and belongs to the observational category of studies.

Descriptive studies entail the collection, analysis and interpretation of data. Both qualitative and quantitative techniques may be used, including questionnaire, interviews, observation of participants, all service statistics and documents describing communities, groups, situations, programmes and other individual or etiological units.

The distinctive feature of this approach is that its primary concern is with description rather than with the testing of hypothesis or proving causality.

1 *Case Series*

This kind of study is based on reports of a series of cases of a specific condition or a series of treated cases, with no specifically allocated control group. They represent the numerator of disease occurrence and should not be used to estimate risks.

In an attempt to make such series more impressive, clinicians may calculate the proportional distribution, which consists simply of the percentages of the total number of cases that belong to a specific category of age, sex, ethnic group or other characteristic. These numbers are still not rates because the denominator is still the cases and not the population at risk. This calculation is called "numerator analysis" and is a common source of confusion and misrepresentation in medical literature. It can be accepted as a measure of risk only if the subgroups are identical in size.

2 *Community Diagnosis or Needs Assessment*

This kind of study entails collection of data on existing health problems, programmes, achievements, constraints, social stratification, leadership patterns, focal points of resistance or high prevalence, or groups at highest risk. Their purpose is to identify existing needs and to provide base-line data for the design of further studies or action.

3 *Epidemiological Description Of Disease Occurrence*

This common use of the descriptive approach entails the collection of data on the occurrence and distribution of disease in populations according to specific characteristics or individuals (age, sex, education, smoking habits, religion, occupation, social class, marital status, health status, personality, etc.), place (rural/urban, local, subnational, national, international), and time (epidemic, seasonal, cyclic, secular). A description may also be given by familial characteristics, such as birth order, parity, family size, maternal age, birth interval or family type. This information is also the descriptive part of every study.

4. *Descriptive Cross-sectional Studies or Community (Population) Surveys*

Cross-sectional studies entail the collection of data on, as the term implies, a cross-section of the population, which may comprise the whole population or a proportion (a sample). Many cross-sectional studies do not aim at testing a hypothesis about an association and are thus descriptive. They provide a prevalence rate at a point in time (point prevalence) or over a period of time (period prevalence). The study population at risk is the denominator for these prevalence rates.

Included in this type of descriptive study are surveys, in which the distribution of a disease, disability, pathological condition, immunological condition, nutritional status, fitness (weight and height), intelligence, etc., is assessed. This design may also be used in health systems research to describe "prevalence" by certain characteristics – the pattern of health service utilisation and compliance – or in opinion surveys. A common procedure used in family planning and, now, in other services, is the KAP survey (survey of knowledge, attitude and practice).

(iii) Analytical Studies

Types of analytical studies are:

1 Case Control studies
2 Prospective Cohort or Longitudinal studies

III PLANNING OF RESEARCH

In planning research, the following basic steps are required:

1) Literature review
- What is already known about the proposed theory and methods of investigation?

2) Primary Research Question

3) Methods
   - Variables - what will you be measuring and how?
   - Sources of Data - who or what will provide the data?

4) Study design and sample
   - Target population, sampling strategy, sample size.

5) Data Collection
   - What methods should be used to collect data?

6) Data handling and analysis
   - How will the data be coded and analyzed?

7) Costs
   - What are the costs of the materials, equipment, and staff needed to complete the research?
   - What is the timetable?

8) Ethical Issues

9) Presentation of results
   - Will the findings be presented as a thesis, a journal publication, or a talk at a meeting?

10) References

When the plan for the whole investigation is clear, it should be written down in the form of a protocol. The protocol will serve as an invaluable guide to you and others of your work and its intentions.

IV LITERATURE SEARCH

No idea is completely new.

It is almost certain that others would have carried out studies related to your interest.

A review of literature is therefore very practical. A good literature review need not be lengthy, tedious or daunting.

The following outlines the scope of Literature Search resources available at our Medical Libraries.

Literature Search using the Medline

- The Medline database is one of the most useful and popular databases for acquiring medical information.

- It embodies a comprehensive range of biomedical literature including research, clinical medicine, administration, policy issues and health care services.

- The Medline corresponds to three printed indexes: INDEX MEDICUS; INDEX TO DENTAL LITERATURE; and INTERNATIONAL NURSING INDEX.

- Its coverage includes over 3,200 journals and 300,000 citations, and each year's information is available on an individual CD ROM.

- Search for information from the Medline can be done via subject heading (MeSH), author, key words, title of article or name of journal.

- Other databases for medical information are PDQ, Cancer Lit, AIDS, etc.

- Currently Medline facilities are available in the NUS Medical Library, Postgraduate Medical Library, SGH, KKH and Mount Elizabeth Hospital. General practitioners have access to only the first two libraries.

- GPs can dial up to the NUS and PGML Library Medline from home, if they have a computer, modem and telephone line.

Others

In addition to Medline, the NUS Medical Library also contains:-

- Excerpta Medica: covers monographs, reviews, yearbooks, annuals
- Micromedex: provides information on drugs and poisons
- Current Contents : condenses the contents page of current journals onto one floppy disk monthly
- OSH ROM : gives information on Occupational Safety and Health.

NEW STRATEGIES IN ASTHMA CARE: AN UPDATE FOR THE PRIMARY CARE DOCTOR

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INTRODUCTION

Until recently, bronchial asthma was regarded as only a bronchospastic disease, and treatment was aimed largely at bronchodilatation. The current thinking is that in addition to the increased response of airway smooth muscles to various stimuli, there is an inflammatory response that is present even in patients with very mild asthma[1,2]. The new rationale of asthma therapy is therefore directed not only at the resolution of bronchospasm but also on the decrease of airway inflammation.

The focus of management has also shifted to regard asthma as a disease of different grades of severity (mild, moderate and severe), each requiring different strategies of care. Prevention of asthma attacks and maintenance of good control through appropriate use of medications and self-care are seen as strategies towards decreasing the risk of mortality and the development of irreversible damage to the airways and permanent bronchial obstruction.

The work of several panels of experts on asthma treatment, namely those of Canada[3], the United Kingdom[4,5] and an International Committee[6], have served to clarify the treatment appropriate to each clinical situation. Treatment of bronchial asthma is no longer ad hoc or empirical.

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APPROACH TO MANAGEMENT

The approach to management depends on the clinical presentation of asthma. Acute episodes require episodic and short term treatment. Chronic persistent asthma requires long-term follow-up and regular assessment. Those who have mild asthma will need minimal monitoring, whereas patients with severe asthma will probably require the expertise of a chest physician.

Management strategy includes the establishment of an accurate diagnosis, the assessment of the degree of severity, avoidance or control of asthma triggers, the institution of therapeutic regimens both acute and chronic, patient education, self care and follow-up.

Diagnosis of Asthma

The diagnosis of asthma is suggested if a patient has symptoms of cough productive of sputum, associated with wheezing, breathlessness and chest tightness. These symptoms occur episodically, especially during the night or early hours of the morning, and can be triggered by allergens or physical stimuli such as cold or exercise[3].

Objective criteria for establishment of diagnosis include the measurement of airflow rates such as the forced expiratory volume in 1 second (FEV1) or the peak expiratory flow rate (PEFR). Demonstrable reversible airways obstruction, such as an improvement of PEFR of > 15% after bronchodilator treatment, or, alternatively, a decrease in PEFR of > 15% following exposure to a known stimulus, confirms the diagnosis of asthma[7].

Assessment of Asthma Severity

An assessment of the patient's degree of severity is important as it helps in deciding the treatment regimen to be used. Asthma can be classified broadly into mild, moderate and severe as indicated in Table 1.

Asthma Triggers

Trigger-factors that can induce asthma include allergens, chemical substances used in the industries such as colophony, medications, physical changes such as cold and exercise, and possibly emotional upset.

Avoidance and control of these will decrease the episodes of acute exacerbation and, in the long term, prevent irreversible bronchial damage.

Skin-testing for allergic reaction to suspected asthma triggers or demonstration of circulating IgE antibodies are only of value for identifying specific antigens, for the purposes of future avoidance[6]. They are not helpful in diagnosing asthma or in the assessment of severity[9].

MANAGEMENT OF CHRONIC PERSISTENT ASTHMA

New strategies in pharmacological therapy of chronic persistent asthma focus on the following:

1. Use of anti-inflammatory agents (such as corticosteriods and cromolyn sodium) as the cornerstone of treatment, instead of bronchodilators;

2. Inhalation, rather than oral, as the preferred route of administration of medications.

Step-Wise Approach

This was proposed in the recent International Consensus Report on Diagnosis and Treatment of Asthma[6]. A four-step plan based on the degree of severity of asthma aims to achieve and maintain control.

Control of asthma is defined as :

1. minimal symptoms, ideally none;
2. infrequent exacerbations;

Table 1 **CLASSIFICATION OF ASTHMA SEVERITY[6]**

ASTHMA SEVERITY	MILD	MODERATE	SEVERE
Clinical Features Before Treatment			
Frequency of Exacerbation	1-2 times/week	> 2 times/week	- Frequent - Previous life-threatening episode - Hospitalisation past year
Symptoms	Asymptomatic between exacerbations	Requires inhaled beta$_2$ agonist almost daily	Continuous
Nocturnal asthma	< 2 times/month	> 2 times/month	Frequent
LUNG FUNCTION			
PEFR predicted at baseline daily variability after bronchodilator	> 80% < 20% normal	60 - 80% 20 - 30% normal	< 60% > 30% below normal

Note: 1. Baseline PEFR established by predicted PEFR for age and sex, or personal best over a 2-3 week observation.
2. Diurnal variation established by twice-daily measurements on waking and before bed. Variability calculated from mean highest and mean lowest values recorded over 2-3 weeks[6].

3. minimal need for inhaled beta2 agonist, ideally none;

4. no limitation on activities, including exercise;

5. (near) normal PEFR, with circadian variation of < 20%;

6. minimal (or no) adverse effects from medications.

The achievement of control is possible in patients with mild or moderate asthma. In patients with severe asthma, this may be difficult, and one has to be content with 'best possible result', which consists of least symptoms, least limitation of activities, least need for inhaled beta2 agonists when necessary, and best achievable lung function tests.

The Step-Wise Approach is shown in Table 2. Progression from one step to a higher step is considered when control cannot be achieved. Conversely, step-down can be considered if symptoms are very well controlled. However, this should only be done slowly and cautiously to avoid a rebound.

Follow-up Care

The above regime should be implemented with regular supervision by the family doctor. At follow-up consultations, the patient's compliance should be checked, together with the degree of control, using criteria listed above. The patient should be encouraged to buy a peak flow meter for use at home. Any problems the patient faces should be discussed and advice given. Modifications to treatment regimes may be necessary depending on control of asthma. The aim, as always, is to enable the patient to lead as normal a lifestyle as possible, including the ability to take part in leisure and sporting activities.

Indications for Specialist Referral

It has been said that in practices where doctors have an interest in asthma, there will be very few referrals [10]. This is because, increasingly, asthma is recognised as a chronic disease requiring long-term monitoring, just like hypertension and diabetes. Nevertheless, referral to a chest physician should be considered under the following circumstances :

Table 2 **STEP-WISE APPROACH IN ASTHMA MANAGEMENT**

STEP	ASTHMA SEVERITY	THERAPY
1	Mild	* Short-acting inhaled $beta_2$ agonist prn <3 times/week * $Beta_2$ agonist or cromolyn sodium before exercise
2	Moderate	* Anti-inflammatory daily (cromolyn in children. Inhaled corticosteroids 200-500 ug increasing up to 750 ug) * Inhaled $beta_2$ agonist prn <4 times/day
3	Moderate	* Inhaled corticosteroids 800-1,000ug daily (>1000ug under specialist supervision), and * sustained release theophylline, oral $beta_2$ agonist, or long-acting inhaled $beta_2$ agonist, especially for control of nocturnal symptoms * Short-acting inhaled $beta_2$ agonist prn <4 times/day
4	Severe	* Inhaled steroids, theophylline and $beta_2$ agonists as above, with or without inhaled anticholinergic, and * oral corticosteroids alternate day or daily single dose

1. When the diagnosis is in doubt, such as when PEFR does not improve after bronchodilator use in a patient who wheezes, or in patients in whom multiple pathology is suspected.

2. To establish a possible aetiological cause, such as in patients with suspected occupational asthma, or allergic tendency.

3. Patients who present some problem in management, such as severe asthmatics who require high doses of steriods, those who are 'brittle' or 'unstable', and the fatality-prone.

MANAGEMENT OF ACUTE EXACERBATIONS

Acute exacerbations or 'asthma attacks' are largely preventable. They usually develop over several days, but may sometimes deteriorate very quickly in a matter of hours. These episodes may range from mild to life-threatening, and are usually as a result of failed maintenance treatment or exposure to asthma triggers[6]. In managing the patient with exacerbation, the general practitioner needs to assess the severity, institute initial treatment, reassess and decide whether to send the patient home or to refer to the hospital for further observation and management.

Assessment of Severity[5, 6, 11]

Physical signs of a severe attack include inability to talk in complete sentences, breathlessness while sitting up, visible use of accessory muscles, agitation, loud wheeze, tachypnoea (respiratory rate $\geqslant$25/min), tachycardia (pulse rate 100-120/min) and pulsus paradoxus, with inspiratory fall in systolic blood pressure of 10-25 mmHg. PEFR after initial bronchodilator treatment (usually taken by patient before consultation) is usually between 50 to 70% of predicted value or personal best.

It is important to pick up the patient at risk of dying. Features of imminently life-threatening asthma include drowiness or confusion, cyanosis, bradycardia, absence of wheeze and silent chest on auscultation.

Immediate Management[5, 6, 11]

Oxygen should be given if available in the clinic, as death from asthma is usually due to hypoxia. The highest concentration available should be given, at a high flow rate, as the relief of hypoxia does not lead to carbon dioxide retention in this situation.

Bronchodilators, preferably beta2 agonists (e.g. salbutamol 2.5-5 mg; terbutaline 5-10mg), should be given via a nebuliser if available, or by multiple actuations from metered dose inhalers into attached spacers. This may be repeated in 20 minutes if there is not much improvement with the first dose. Ipratropium bromide 0.5mg may be added to the nebuliser solution. Intravenous bronchodilators should be given in life-threatening situations. If the patient is not already on oral theophylline, then i/v aminophylline (250 mg over 30 min) can be administered. Alternatively i/v beta2 agonists (e.g. salbutamol 200ug or terbutaline 250ug over 10 min) may be given slowly.

Systemic corticosteroids should be started immediately by the first doctor attending to the patient with an acute severe attack, as studies on asthma deaths have found the under-use of steroids to be an important contributory factor[11]. Oral prednisolone 30-60 mg and/or i/v hydrocortisone 200mg are recommended[5].

Reassessment

This is based on clinical findings such as the degree of breathlessness, the respiratory and pulse rates, as well as PEFR level. If there is sustained improvement in symptoms and PEFR of $\geqslant$ 80% predicted/personal best value, then the patient can be sent home with monitoring by the general practitioner. As full recovery from an acute exacerbation may take several days, the patient should be prescribed inhaled bronchodilators every 3-4 hours for at least 24-48 hours[6]. Oral corticosteriods, if started, should be continued for one to two weeks (e.g. prednisolone 30 mg each morning), and can be stopped without the need to taper down as the duration of use is short[12]. Where relevant, any trigger that precipitated the acute episode should be identified and the patient advised to avoid further contact with it.

Criteria for Emergency Referral to Hospital

These are listed as below[5]:

1. Any life-threatening features
2. Features of severe attack persisting after initial treatment
3. PEFR <40% predicted/personal best (or <200L/min) 15-30 min after nebulisation
4. A lower threshold for admission in the following:
   - patients seen in the afternoon or evening, as opposed to patients seen in the morning,
   - recent onset of nocturnal symptoms / worsening of symptoms,
   - history of previous severe attacks,
   - concern over assessment of severity,
   - adverse social circumstances.

Follow-up on Discharge from Hospital

Upon discharge from the hospital, the patient should ideally be followed-up within a week by his general practitioner, and be continued for a short period of time on the medications he is discharged with. He should also be reviewed by the hospital specialist until such time that the best achievable lung function is reached. It may be necessary, as a consequence of this acute episode, to modify the medications he was previously on, in order to achieve control of asthma.

The Fatality-prone Asthmatic Patient

The increase in the number of asthma deaths reported in many countries is a cause of real concern to doctors looking after asthmatic patients. Recognition of contributory factors will help physicians prevent unnecessary deaths.

Why do asthmatic patients die? Listed below are some possible reasons[13, 14]:

1. Severity of attack not recogised, both by the patient as well as by the physician;
2. Severity recognised but not the risk of death; and these are related to patient education and physician knowledge.
3. Inadequate treatment, such as insufficient doses of bronchodilators given, or given at too far apart intervals.
4. Inappropriate treatment, for example corticosteroids are not given when indicated.
5. Delays in treatement, for example when poor control over a period of time is not recognised.
6. Toxicity or over-use of drugs, especially with the use of xanthine derivatives.

Patients at high risk of death are :

1. Those with a history of a near-death episode requiring resuscitation.
2. Those with severe underlying disease.
3. Those recently discharged from hospital.
4. Those with poor self-care, who are non-compliant or are depressed.
5. Those with poor family or social support.
6. Those with discontinuity in follow-up care.

PATIENT SELF CARE

As asthma is a chronic disease, the 'mutual participation model' of doctor-patient relationship applies, and effective disease management requires care on the part of the physician as well as co-operation on the part of the patient. In order for this to be successful, the patient must first understand his disease, and know what to do under different circumstances. Hence patient education is important. The extent to which the patient comprehends what is taught will depend a lot on his interest and educational level. Needless to say, the interest of the doctor and the way he explains to the patient are also important factors.

What does the patient need to know and do? He must know the degree of severity of disease. He should be taught home monitoring by an objective method (e.g. routine measurement of PEFR using the peak flow meter), as it is well documented that patients often underestimate the severity of their own attacks[15]. The use of diary cards to record the daily events should be encouraged. Patients who are motivated in self care should be given specific instructions on what to do when they detect a deterioration in the parameters they are monitoring, including the instruction to start corticosteroids where applicable. They should also be taught when to seek medical treatment, upon observation that there is no improvement with self management. These self-management plans should preferably be written down, so that the patient can refer to them whenever necessary[16, 17, 18].

The 'asthma management zone system'[6], or 'traffic light concept', enhances patient understand-

ing. 'Green' is when all systems go and the patient can manage on his own. PEFR is around 80-100% of predicted or personal best value with minimal daily variability, and patient can perform all his normal daily activities with minimal symptoms. 'Yellow' indicates caution and the need for close monitoring. Should the patient not improve, or his condition deteriorates, the physician should be contacted. In these cases, the PEFR is usually between 50 to 80%, with diurnal variability of 20-30%, with the occurrence of symptoms, especially in the night. When the status is 'red', it is an indication of a 'medical alert'. PERF is <50% of predicted or personal best. If it remains <50% despite home use of use of bronchodilators, the patient should be instructed not to wait or to try his own treatment further but to seek immediate help, e.g. by calling the ambulance to send him to the hospital emergency department. It should be stressed that patients should institute self-care measures as instructed at the onset of detecting a deterioration in symptoms and routine PEFR, as this will reduce the frequency of 'sliding down' the treatment zones, and hence the occurrence of acute exacerbations.

Family support is important, and family members should be counselled and educated with regard to recognising symptoms associated with severity, encouraging patient compliance with medication, and life style changes. Their understanding of the disease and the part which they can play will be helpful to the attending physician. The availability of family and/or social support, the patient's understanding and degree of dependency, as well his ability to cope with instructions given, have all to be taken into considerion in individualising the level of colour-coding for each patient.

CONCLUSION

The recent work of expert panels clarify the new thinking in the pathophysiology of asthma, and shift the focus from episodic care to prevention and maintenance of control. These strategies spell promise in helping to reduce mortality as well as short and long term morbidity in bronchial asthma. They should therefore be incorporated into our management and care of the asthmatic patient.

References

1. Olivieri D, Chetta A. Therepy of asthma in adults : the control of bronchial inflammation. Eur Respir J 1989; 2 Suppl 6:540-4.
2. Laitinen LA, Heino M, Laiytinen A, HaahtelaT. Damage of the airway epithelium and bronchial reactivity in patients with asthma. Am Rev Respir Dis 1985; 131:599-606.
3. Hargreave FE, Dolovich J, Newhouse MT, eds. The assessment and treatment of asthma : a conferece report. J Allergy Clin Immunol June 1990; 1098-111.
4. Constain D, Harrison BDW, Holgate ST, Hopkins AP, Partridge MR, Barnes PJ, et al. Guidelines for management of asthma in adults : I-chronic persistent asthma. BMJ 1990; 301:651-3
5. Constain D, Harrison BDW, Holgate ST, Hopkins AP, Partridge MR, Barnes PJ, et al. Guidelines for management of asthma in adults : II-acute severe asthma. BMJ 1990; 301:797-800.
6. International Asthma Management Project. International consensus report on diagnosis and treatment of asthma. Eur Respir J 1992; 5:601-41.
7. Maxwell D. Asthma : strategies for whole-patient management. In: Asthma. Modern Medicine - Postgraduate Partwork Series June 1988; 1-12.
8. Gregg I, Nunn AJ. Peak expiratory flow in normal subjects. Br Med J 1973; iii:282-4.
9. Inouye I, Tarlo MB, Broder I, Corey P, Davies G, Leznoff A, et al. Severity of asthma in skin test-negative and skin test-positive patients. J Allergy Clin Immunol 1985; 75:313-319.
10. Bucknall CE. Who needs referral to the hospital specialist? Respiratory Medicine 1991; 85:453-5.
11. Neville E, Gribbin H, Harrison BDW. Acute Severe Asthma. Respiratory Medicine 1991, 85:463-74.
12. Barnes PJ. A new approach to the treatment of asthma. N Engl J Med 1989; 321:1517-21.
13. Brennan M, Gowdey CW. Avoiding asthma fatalities in family practice. Can Fam Physician 1989; 35:1509-14.
14. Strunk RC. Workshop on the identification of the fatality-prone patient with asthma. J Allergy Clin Immunol 1987; 80:455-8.
15. Ellis ME, Friend JAR. How well do asthma clinic patients understand their asthma? Br J Dis Chest 1985; 79:43-8.
16. Beasley R, Cushley M, Holgate ST. A self management plan in the treatment of adult asthma. Thorax 1989; 44:200-204.
17. Brewis RAL. Patient education, self-management plans and peak flow measurement. Respiratory Medicine 1991; 85457-62.
18. Charlton I, Charlton G, Broomfield J, Mullee MA. Evaluation of peak flow and symptoms only self management plans for control of asthma in general practice. BMJ 1990; 301:1355-9

COLONOSCOPY AND COLONOSCOPIC POLYPECTOMY

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INTRODUCTION

Fibre-optic endoscopy was first introduced in the seventies, making a major diagnostic and therapeutic revolution in the management of gastrointestinal diseases. The first colonoscope was introduced into clinical use at that time. Since then advances in instrumentation and technique have extended the range of its diagnostic and therapeutic capabilities. The introduction of the wire loop snare and electrocautery has enabled colonic polyp removal through the instrument.[1] Heater probes and contact laser points can now be introduced through the colonoscope's therapeutic ports. Completion rates, that is, either reaching the caecum or the end point (such as an obstructing carcinoma) are well over 90 per cent in experienced hands. The risks and complications are minimal and patient acceptance of the procedure is generally good.

One of the major activities in colonoscopy these days is in the field of 'prevention', that is, the prevention of colorectal malignancies. The colonic adenoma-carcinoma sequence is now widely accepted. Colonoscopic removal of adenomatous polyps are an attempt to interrupt the sequence and thus prevent the onset of carcinoma.[2]

INDICATIONS FOR COLONOSCOPY

The **indications** for colonoscopy are many (Table 1). It is generally agreed that colonoscopy supplements but does not completely replace the barium enema examination in the evaluation of colorectal diseases. Most instances the procedure is used either because barium enema study was inconclusive or because barium enema failed to identify the source when symptoms suggest a colonic lesion.[3] In two reports analysing unexplained rectal bleeding, 30% of patients were found to have significant lesions in the colon despite normal barium enema examinations[4,5].

Table 1 Indications for colonoscopy

- Confirmation of suspected or equivocal radiologic abnormality (filling defects, narrowing, polyps)
- Evaluation and follow up of inflammatory bowel disease (such as dysplasia)
- Differential diagnosis of diverticular disease or malignancy
- Presence of a rectal polyp with or without barium enema abnormality (synchronous lesion)
- Gastrointestinal symptoms (eg bleeding, abdominal pain, anaemia) where radiologic investigation has failed to reveal the source
- Follow-up of patient after colonic surgery for cancer
- Acute lower gastrointestinal bleeding
- Colonoscopic polypectomy
- Reduction of sigmoid volvulus

The **contraindications** to barium enema are few. These include patients with recent myocardial infarction and those with acute abdominal inflammation such as peritonitis, acute diverticulitis and bowel perforation. **Physiological changes** such as a decrease in pulse rate, respiratory rate and arterial oxygenation have been recorded. These have been attributed partly to the use of

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sedation and analgesia for the procedure rather than colonoscopy per se.

DIAGNOSTIC COLONOSCOPY

1. Barium Enema Abnormalities

Although barium enema examinations remain the primary diagnostic tool in suspected colorectal disorders, colonoscopy is frequently necessary to clarify radiological abnormalities such as strictures or unusual filling defects. Even if a carcinoma is found radiologically, total colonoscopy is indicated to search for co-existing synchronous lesions as barium enema has been shown to miss about 60% of synchronous colorectal cancers[6]. Colonoscopy has been reported to be superior to barium enema examinations in detecting synchronous colorectal cancers.[7,8,9,10] If complete colonoscopy is not possible pre-operatively due to an obstructing distal lesion, then it should be performed at the earliest convenient time following recovery from surgery[7].

2. Gastrointestinal Symptoms

Colonoscopy is also frequently performed for significant colonic symptoms such as unexplained abdominal pain and recent changes in bowel habit. Frequently lesions such as polyps, cancers and inflammatory bowel disease may be picked up on colonoscopy despite normal barium enema.

3. Rectal Bleeding

Colonoscopy is most helpful when rectal bleeding is intermittent or has spontaneously subsided[11]. The source of rectal bleeding can be identified in most instances by an experienced colonoscopist and may also provide a method for control of bleeding either by electrocoagulation or snare polypectomy.

4. Inflammatory Bowel Disease

Patients with troublesome diarrhoea and negative x-rays may need colonoscopy with biopsy to clarify the nature of the disease. In patients with inflammatory bowel disease colonoscopy is necessary to document the extent of colonic involvement and hence the prognosis. Also the increased risk of cancer in patients with universal ulcerative colitis exceeding ten years is well documented, hence requiring interval colonoscopy with multiple sequential biopsies and surveillance. The presence of severe dysplasia may necessitate early colectomy.

5. Post Colectomy Follow Up

Follow-up colonoscopies should be performed regularly for all patients after colon resection for malignancy. Colonoscopy should be done within six months of operation to search for any co-existing synchronous lesions and to examine the anastomotic site for any evidence of early recurrence. Subsequent colonoscopies can be done every three years once the colon has been cleared as adenomas that develop de novo take five years or more to reach a significant size with malignant potential[11].

THERAPEUTIC COLONOSCOPY

The colonoscope can be used for several therapeutic procedures which include haemostasis by electrocoagulation, removal of foreign bodies inserted into the rectum, reduction of sigmoid volvulus and passage of a decompression tube. However colonoscopic polypectomy is by far the most widely accepted and commonly performed therapeutic procedure.

COLONOSCOPIC POLYPECTOMY

The advent of colonoscopic polypectomy and its role in the 'prophylactic' treatment of colorectal carcinoma has been well documented as nearly all adenocarcinomas of the colon develop from adenomas — a process referred to as the adenoma-carcinoma sequence[12,13]. Most polyps can be removed or at least biopsied by the colonoscope. These adenomas are usually found after the age of 50 and there appears to be a shift in distribution towards the more proximal colon.

Adenomatous polyps can be classified into three main histological types; tubular, villous or tubulovillous. The malignant potential depends on the size, histological type and degree of dysplasia. When histology of the polyps removed shows invasive carcinoma, a colectomy is generally advised if the base of the polyp is involved by

cancer or if it is a poorly differentiated cancer. However if the carcinoma is superficial and contained above the muscularis mucosae, a complete polypectomy is a satisfactory definitive treatment.

POST POLYPECTOMY FOLLOW UP

All patients after polypectomy are at increased risk of developing new polyps and close follow-up interval colonoscopies are advised.

COMPLICATIONS OF POLYPECTOMY

1. Haemorrhage

This occurs in about one per cent of all cases and is often due to an inadequate coagulation of blood vessels within the pedicle and it is usually obvious immediately at the time of polypectomy. However presentation can be delayed for several days. Treatment is usually expectant as most bleeding will stop spontaneously.

2. Perforation

This is a rare complication occuring in less than 0.5% of cases. The commonest site of perforation is the sigmoid colon, usually secondary to an underlying lesion over that area such as diverticular disease, radiation colitis and inflammatory bowel disease. Most perforations can be managed by simple debridement and primary closure if the bowel preparation is satisfactory. Resection may occasionally be required if there is underlying disease.

3. Others

These include post-colonoscopy distention, retroperitoneal and mediastinal emphysema, bacteraemia and other minor medical complications (renal, cardiac and respiratory).

BARIUM ENEMA VERSUS COLONOSCOPY

There is little doubt that colonoscopy is more likely to detect small lesions in the colon that are likely missed on barium enema examinations, even with optimal preparation and double-contrast technique[14,15]. Furthermore colonoscopy allows for histological examination of any lesion detected in the colon, a clear advantage over the barium enema.

However the difference in cost between the two procedures still makes barium enema a much preferred procedure by many physicians, using colonoscopy only if barium enema is inconclusive.

CONCLUSION

The diagnostic and therapeutic potentials of the colonoscope are recognised internationally. The most recent development is in the field of video-endoscopy. This will make it easier to store and retrieve information, facilitate teaching and also provide an important tool for quality control and research.

References

1. Wolff WI, Shinya H. Polypectomy via the fibreoptic colonoscope: removal of neoplasms beyond the reach of the sigmoidoscope. N Engl J Med 1973;288:329-32.
2. Coller JA, Corman ML, Veidenheimer MC. Colonic polypoid disease: need for total colonoscopy. Am J Surg 1976;131:490-5.
3. Coller JA, Corman ML, Veidenheimer MC. Diagnostic and therapeutic applications of fibreoptic colonoscopy. Geriatrics 1974;29:67-73.
4. Brand EJ, Sullivan BH, Sivak MV, Rankin GB. Colonoscopy in the diagnosis of unexplained rectal bleeding. Ann Surg 1980;192:111-3.
5. Tedesco FJ, Pickens CA, Griffin JW, Sivak MV, Sullivan BH. Role of colonoscopy in patients with unexplained melaena: an analysis of 53 patients. Gastrointest Endosc 1981;27:221-3.
6. Heald RJ, Bussey HJKR. Clinical experiences at St Mark's Hospital with multiple synchronous cancers of the colon and rectum. Dis Colon Rectum 1975;18:6-10.
7. Eu KW, Seow CF, Goh HS. Characteristics of synchronous colorectal cancers. Dis Colon Rectum (in press).
8. Appel MF. Preoperative and postoperative colonoscopy for colorectal carcinoma. Dis Colon Rectum 1976;19:664-6.
9. Maxfield RG. Colonoscopy as a routine preoperative procedure for carcinoma of the colon. Am J Surg 1984;147:477-80.
10. Rami K, John PW. Quintuple synchronous colon carcinoma: a report of 2 cases. J Surg Oncol 1986;32:55-7.
11. Hunt RH, Waye JD. Colonoscopy: techniques, clinical practice and color atlas. Chapman Hall, London 1981.
12. Welch JP. Multiple colorectal tumours. An appraisal of natural history and therapeutic options. Am J Surg 1981; 142:274-80.
13. Nottle PD, Polglase AL, Hughes ESR, McDermott FT, Pihl EA, Price AB. Synchronous carcinomas of the large intestine. Aust NZ J of Surg 1983:53:329-32.
14. Dodds WJ, Stewart ET, Hogan WJ. Role of colonoscopy and roentgenology in the detection of polypoid colonic lesions. Dig Dis Sci 1977;22:646-49.
15. Knutson CO, Williams HC, Max MH. Detection of intracolonic lesions by barium contrast enema. JAMA 1979;242:2206-8.

BRONCHIOLITIS: DO DRUGS REALLY WORK?

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INTRODUCTION

Acute bronchiolitis is the most common severe lower respiratory tract infection of infancy, being a cause of considerable morbidity in this age group. Fortunately mortality from this disease in previously well infants stands at less than 1%. However in infants with significant pre-existing cardiorespiratory disease e.g. severe congenital heart disease, bronchopulmonary dysplasia, cystic fibrosis and immunosuppressed patients, mortality may reach 30%.

Besides the immediate mortality and morbidity, several studies have also shown that a high proportion of babies admitted to hospital with acute bronchiolitis will have subsequent attacks of coughing and wheezing associated with abnormal airway lability i.e. increased bronchial hyperactivity[1]. 75% of these babies will have symptoms of wheezing over the subsequent 2 years and 22% may have recurrent wheezing by 8-10 years[2]. Several studies have also shown that there is no increased incidence of atopy in these patients.

PATHOGENESIS AND PATHOPHYSIOLOGY

While other viruses such as influenza, parainfluenza viruses and adenovirus can cause bronchiolitis, in the majority of cases, the etiological agent is the respiratory syncytial virus. The virus causes damage to the respiratory epithelium and incites an inflammatory reaction in the bronchioles and smaller bronchi with resultant airway obstruction from mucosal edema, bronchospasm and mucus plugging. Clinically this results in hyperinflation of the lungs, tachypnoea, subcostal and intercostal retractions, hypercarbia from increased work of breathing and hypoxemia from ventilation-perfusion imbalance. The relatively narrower airways of infants makes them more susceptible to the marked air trapping that occurs in this disease.

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MANAGEMENT

It is generally agreed that the mainstay of treatment is good supportive care namely: close monitoring, adequate hydration, supplemental humidified oxygen and artificial ventilation if respiratory failure sets in as indicated by progressive hypoxemia and hypercarbia.

A whole host of various treatment modalities have been attempted and whether they are in any way efficacious in modifying the course of the disease is still by and large fairly controversial. These will be discussed as follows:

a) bronchodilators
b) antiviral agents
c) corticosteroids
d) theophylline

BRONCHODILATORS

ß2 Agonists

It is known that ß2 agonists work effectively in relieving bronchospasm in asthmatics but their

effectiveness in relieving airway obstruction in infants with bronchiolitis is debatable. Is it because infants have relatively deficient bronchiolar smooth muscle or that smooth muscle spasm plays only a small role in the overall pathogenesis? Or is it because infants have deficient functional ß2 receptors?

Pathological specimens have revealed the presence of smooth muscle down the full length of the infant's bronchial tree but are the ß2 receptors functional?

The most widely used ß2 agonist in the various studies is nebulised salbutamol. Basically 2 types of studies have been carried out: measuring lung function of these wheezing infants before and after salbutamol or measuring transcutaneous oxygen saturations and clinical scoring before and after the drug.

Lenney[3,4] et al measured respiratory resistance in 32 wheezing children and infants with a total body plethysmograph before and after nebulised salbutamol and found that there was no significant fall in resistance after salbutamol in children less than 18 months old, while in those older than 18 months, nebulised salbutamol produced a significant 20% fall in total respiratory resistance.

Rutter[5] et al similarly showed no improvement in lung function after nebulised salbutamol in infants less than 1 year old. This seemed to imply that infants do not have sufficient bronchial smooth muscle or functional ß2 receptors and explained the general impression that ß2 agonists were not very effective in infants, precisely the group most susceptible to bronchiolitis.

In another study, O'Callaghan[6] et al, using a total body plethysmograph, showed that nebulised water, being a hypo-osmolar solution, produced a significant increase in airway resistance in infants with recurrent wheezing attacks. However when nebulised water was given 20 minutes after nebulised salbutamol, this deterioration in lung function did not occur, implying that salbutamol protected the airways against the bronchoconstricting effect of water.

Similarly, Prendiville[7,8] et al studied the response to nebulised histamine in 5 wheezy infants with a body plethysmograph. They measured the dose of histamine needed to produce a 30% fall in maximum flow at functional residual capacity (PC30) and found that when nebulised salbutamol was given 30 minutes before histamine, this abolished the bronchoconstrictor action of histamine up to a maximum of 8 g/L of histamine.

Both the latter 2 studies imply that infants do indeed have functional ß2 receptors. So should nebulised salbutamol be used in bronchiolitis?

In 2 separate subsequent studies, O'Callaghan[9] as well as Prendiville[10] demonstrated a paradoxical deterioration in lung function (increased airway resistance) 15 minutes after nebulised salbutamol (0.5 mls salbutamol respirator solution to 1.5 mls normal saline) was given to infants with recurrent episodes of wheezing. This was attributed to the hypo-osmolality of the mixture (228 mmol/kg) as well as the acidic nature of the mixture (pH = 4.75), or possible ventilation-perfusion imbalance due to pulmonary vasodilatation caused by salbutamol.

Other investigators subsequently studied the effect of salbutamol on the clinical state of the patient as well as on their transcutaneous oxygen saturations.

Prendiville[11] et al as well as Ling Ho[12] et al both demonstrated a decrease in mean oxygen saturation by pulse oximetry of 1.4 to 2% after nebulised salbutamol was administered to 5 and 11 patients admitted for acute bronchiolitis.

From the above studies it might seem inadvisable to administer nebulised salbutamol in bronchiolitis. However two other recent studies from Canada dispute this. Klassen[13] et al showed that while there was no change in oxygen saturation with pulse oximetry, the clinical score of the patient based on respiratory rate, wheezing and chest retractions improved significantly in the group treated with nebulised salbutamol (0.1 mg/kg of salbutamol in 2 mls of 0.9% normal saline). Schuh[14] et al showed an increase of 0.76% in oxygen saturation as well as a significant improvement in clinical score after nebulised salbutamol. It must be noted that in both these studies, the nebuliser was driven by oxygen at 6-7 1/minute, unlike the previous studies where

compressed air was used.

It must be noted from clinical experience that pulse oximetry recordings are quite prone to movement artifacts and that actual serial blood gas monitoring via an intra-arterial line would probably have been more accurate.

So far no studies on the use of intravenous salbutamol in severe acute bronchiolitis has been done.

Studies done with other less selective bronchodilators such as nebulised isoprenaline and adrenaline have not shown any beneficial effect.

Lowell[15] et al did an interesting study in which subcutaneous adrenaline (1 mg/ml) at 0.01 ml/kg was given to patients with bronchiolitis and found an improvement in clinical score in 50% of those treated compared to 7% improvement to those given placebo. It was postulated that adrenaline was effective in reducing oedema around the airways by its vasoconstrictor action. Clearly further studies of this mode of administration are needed.

From the above conflicting studies, it can be seen than further more comprehensive studies using standard protocol to measure simultaneously the same parameter of lung function as well as clinical scoring and arterial oxygenation preferably via arterial blood gas monitoring during and after drug administration are needed before any firm conclusions are drawn.

Anticholinergics: Ipratropium Bromide (Atrovent)

Ipratropium, a quaternary ammonium derivative of atropine, produces bronchodilatation by competitive inhibition of cholinergic receptors on bronchial smooth muscle. It antagonises the action of acetylcholine at the receptor site and thereby blocks the bronchoconstrictor action of vagal efferent impulses. It acts locally with little systemic effect.

Stokes[16] et al found that nebulised ipratropium produced a significant decrease in work of breathing wheezing infants compared to salbutamol and water which both resulted in increased work of breathing.

Hodges[17] et al also showed an improvement in lung function i.e. a decrease in airways resistance of at least 15% in 40% of wheezing patients < 18 months old after nebulised ipratropium. Only 2 out of the 32 showed an increase in airway resistance after ipratropium.

Seidenberg[18] et al found neither any improvement nor any deterioration in lung function (partial expiratory flow-volume curves and maximum flow at functional residual capacity) in patients with acute bronchiolitis.

The problem about drawing a firm conclusion from the above studies is that each author measured a different modality of lung function. However it would appear that ipratropium does not seem to be associated with any deleterious side-effects and may be useful in some infants with bronchiolitis.

ANTIVIRALS: RIBAVIRIN

Ribavirin is a synthetic nucleoside analog resembling guanosine and inosine and interferes with the expression of messenger RNA and inhibits viral protein synthesis. It was designed specifically against respiratory syncytial virus and is delivered as an aerosol via a small particle generator into an oxygen hood or tent for 3 to 5 days for 12 to 20 hours per day. It is recommended for use within the first few days of RSV infection for maximum benefit so immunofluorescence techniques for rapid diagnosis should be available.

It costs about S$1500-2000 for a course of therapy and as such is not usually recommended for previously well babies with respiratory syncytial virus (RSV) infection in whom the expected mortality and morbidity is low.

A study by Hall[19] et al on high risk patients i.e. those with bronchopulmonary dysplasia and congenital heart disease showed a significant improvement in clinical score and arterial oxygenation in those treated with ribavirin compared to those treated with placebo as well as a decrease in virus shedding in the treated group.

The American Academy of Paediatrics (AAP)[20] suggested that nebulised ribavirin should be considered for the following patients with proved infection with RSV:

1) Patients at "high risk": those with congenital heart disease, bronchopulmonary dysplasia, premature infants, those with immune deficiency (especially severe combined immune deficiency), recent transplant patients and those on chemotherapy for malignancy.

2) severely ill infants with paO2 < 65 mmHg and rising pCO2.

3) infants with mild disease which may progress: infants under 6 weeks of age and those with an underlying condition such as multiple congenital anomalies, neurological and metabolic diseases.

An editorial review in the Archives of Diseases of Childhood[21] commenting on the AAP criteria agreed that ribavirin should be used in children with severe immunedeficiency but not necessarily in those on chemotherapy for malignancy. Commenting on the other criteria, it said that "there is no firm evidence that ribavirin reduces mortality even in high risk patients, nor that it obviates the need for artificial ventilation or reduces duration of hospital stay". It called for further multicentre double-blind placebo controlled trials.[22]

Ribavirin tends to precipitate in the respirator tubings so if it is to used in ventilated patients, then filters and one-way valves need to be used to prevent inadvertently high levels of positive end-expiratory pressure due to crystallisation of the drug.

So far no side-effects have been reported in patients treated with it. Teratogenicity has been observed in pregnant rodents given the drug orally for 2 weeks but has not been observed in treated pregnant baboons. No appreciable levels of the drug have been detected in either the serum or urine of patients or personnel exposed to the drug during one course of therapy.

However there is concern in some centres about repeated prolonged exposure to it among pregnant health care personnel especially if the drug is widely used during an RSV epidemic and they recommend that special precautions be taken when administering the drug[23,24].

CORTICOSTEROIDS

Systemic corticosteroids were tried as early as 1969 based on the hypothesis that their anti-inflammatory action would reduce bronchiolar inflammation and oedema. There were initially conflicting results based on several small trials using a variety of steroids at different doses. Subsequently 2 large double-blind controlled studies were done.

In a study by Connoly[25] et al, 100 patients between the ages of 3 weeks and 26 months with the first acute attack of bronchiolitis were given prednisolone in decreasing dosage: 15 mg on the first day, 10 mg on the second and third days, 5 mg on the fourth and fifth days and 2.5 mg on the sixth and seventh days as well as supplemental oxygen. There was no difference in severity of illness between those treated with prednisolone and those on placebo. There was also no difference in the duration of signs and symptoms in the two groups i.e. prednisolone treatment did not hasten the resolution of the illness.

In another large multicentre double-blind controlled study by Leer[26] et al involving 297 patients, intramuscular betamethasone sodium phosphate was administered 12 hourly over 72 hours to those admitted with acute bronchiolitis and a clinical scoring assessment was done for these in terms of chest retractions, wheezing, cyanosis, air-exchange and flaring of ala nasae. Although cyanosis occurred less frequently and lasted a shorter time in those treated with betamethasone (in only 29 of 148 patients treated with betamethasone cf. in 42 of the 149 placebo patients) and impaired air-exchange improved in less than 1 day in 19 patients treated with corticosteroid compared with 2 patients in the control group, analysis of the total clinical score did not show any statistically significant difference in response between those treated with steroid and those on placebo.

Subsequently Tal[27] et al in a study on 32 infants showed that the combination of intramuscular

dexamethasone for 3 days together with nebulised and oral salbutamol produced a significant improvement in clinical signs and symptoms compared to either salbutamol or dexamethasone alone. However there were no significant differences in paO2 or paCO2 in the various treatment groups.

While corticosteroids may not seem to have much therapeutic effect in the acute phase of bronchiolitis, recent studies show that inhaled steroids, either beclomethasone dipropionate or budesonide, are effective in decreasing the frequency of postbronchiolitic wheezing episodes.

Carlsen[28] et al in a double-blind placebo controlled study on 44 patients with recurrent episodes of wheezing after acute bronchiolitis, showed that the group treated with nebulised beclomethasone dipropionate (100 microgram q.d.s. for 2 weeks and then 100 micrograms b.d. for 4 weeks) had significantly fewer symptomatic respiratory illnesses and fewer episodes of bronchopulmonary obstruction during a follow-up period of 1 year. The patients given placebo had significantly higher obstructive scores during the study period and were treated with inhaled ß2 agonists and theophylline for longer periods of time during the follow-up period. Total IgE did not differ between the 2 groups.

Maayan[29] et al showed a significant improvement in specific airway conductance (a measure of airflow limitation), tachypnoea, chest retraction, and wheezing after 2 weeks treatment with nebulised beclomethasone dipropionate (300 microgram/day) in 9 infants aged less than 9 months with frequent wheeze and dyspnoea following bronchiolitis, but this benefit stopped rapidly after stopping therapy.

Bisgaard[30] et al in a study on 77 patients aged 11 to 36 months with moderately severe recurrent wheezing (whether due to viral infections or asthma) showed that the group given inhaled budesonide (400 microgram b.d.) via a spacer with facemask for 8 weeks had significantly improved symptom scores of wheezing, sleep disturbance and patient happiness compared to placebo during the treatment period. No side-effects were noted in these patients except for oral candidiasis in 3 patients.

THEOPHYINE

Brooks[31] et al did a retrospective review of theophylline therapy (5 mg/kg 6 hourly) in acute bronchiolitis and found no difference in rate of resolution of the illness compared to those not on theophylline and concluded that theophylline was had no beneficial effect on bronchiolitis. However serum levels of theophylline were not assessed and further prospective trials are needed.

CONCLUSION

Hence it can be seen that the management of acute bronchiolitis still remains largely supportive and the role of the various drugs discussed still remain fairly controversial. It is hoped that large multicentre trials using standard protocol as well as assessment of standard parameters of response will be carried out in future to further delineate the role of such drugs.

References

1. Henry RL, Hodges IGC, Milner AD, Stokes GM. Respiratory problems two years after acute bronchiolitis in infancy. Arch. Dis. Child. 1983, 58:713-6.
2. Pullan CR, Hey EN. Wheezing, asthma and pulmonary dysfunction 10 years after infection with respiratory syncytial virus in infancy. Br Med. J 1982, 284:1665-9.
3. Lenney W, Milner AD. At what age do bronchodilators work? Arch. Dis. Child. 1978, 53:532-535.
4. Lenney W, Milner AD. Alpha and beta adrenergic stimulants in bronchiolitis and wheezy bronchitis in children under 18 months of age. Arch. Dis. Child. 1978, 53:707-9.
5. Rutter N, Milner AD, Hiller EJ. Effect of bronchodilators on respiratory resistance in infants and young children with bronchiolitis and wheezy bronchitis. Arch. Dis. Child. 1975, 50:719-2.
6. O'Callaghan C, Milner AD, Swarbrick A. Nebulised salbutamol does have a protective effect on airways in children under 1 year old. Arch. Dis. Child. 1988, 63:479-483.
7. Prendiville A, Green S, Silverman M. Bronchial responsiveness to histamine in wheezy infants. Thorax 1987, 42:92-99.
8. Prendiville A, Green S, Silverman M. Airway responsiveness in wheezy infants: evidence for functional beta adrenergic receptors. Thorax 1987, 42:100-104.
9. O'Callaghan C, Milner AD, Swarbrick A. Paradoxical deterioration in lung function after nebulised salbutamol in wheezy infants. Lancet 1986, ii:1424–1425.
10. Prendiville A, Green S, Silverman M. Paradoxical response to nebulised salbutamol in wheezy infants assessed by partial expiratory flow-volume curves. Thorax 1987, 42:86-91.
11. Prendiville A, Rose A, Maxwell DL, Silverman M. Hypoxemia in wheezy infants after bronchodilator treatment. Arch. Dis. Child. 1987, 62:997-1000.
12. Ling Ho, Collis G, Landau LI, Le Souef PN. Effect of salbutamol on oxygen saturation in bronchiolitis. Arch. Dis. Child. 1991, 66:1061-1064.
13. Klassen TP, Rowe PC, Sutcliffe T, et al. Randomised trial of

salbutamol in acute bronchiolitis. J. Pediatr. 1991, 118:807-11.
14. Schuh S, Canny G, Reisman JJ, et al. Nebulised albuterol in acute bronchiolitis. J. Pediatr. 1990, 117:633-7.
15. Lowell DI, Lister G, Von Koss, McCarthy P. Wheezing in infants: The response to epinephrine. Pediatrics 1987, 79:939-45.
16. Stokes GM, Milner AD, Hodges IGC, Henry RL, Elphick MC. Nebulised therapy in acute severe bronchiolitis. Arch. Dis. Child. 1983, 58:279-283.
17. Hodges IGC, Groggins RC, Milner AD, Stokes GM. Bronchodilator effect of inhaled ipratropium bromide in wheezy toddlers. Arch. Dis. Child. 1981, 34:729-32.
18. Seidenberg J, Masters IB, Hudson I, Olinsky A, Phelan PD. Effect of ipratropium bromide on respiratory mechanics in infants with acute bronchiolitis. Aust. Paediatr. J, 1987, 23:169-172.
19. Hall CB, Mcbride JT, Gala CL, Hildreth SW, Schnabel KC. Ribavirin treatment of respiratory syncytial viral infection in infants with underlying cardiopulmonary disease. JAMA 1985, 254:3047-3051.
20. Committee on infectious diseases. Ribavirin therapy of respiratory syncytial virus. Pediatrics 1987, 79:475-6.
21. Isaacs D, Moxon ER, Harvey D et al. Ribavirin in respiratory syncytial virus infection. Arch. Dis. Child. 1988, 63:986-990.
22. Wald ER, Dashefsky B, Green M. In re ribavirin: A case of premature adjudication. J. Paeds. 1988, 112:154-8.
23. Fackler JC, Flannery K, Zipkin M, Mcintosh K. (letter) Precautions in the use of ribavirin at the Children's Hospital. JAMA. 1989, 261:1879-80.
24. Guglielmo RJ, Jacobs RA, Locksley RM (letter) The exposure of health care workers to ribavirin aerosol. JAMA. 261:1880-81.
25. Connolly JH, Field CMB, Glasgow JFT, Slattery CM, Maclynn DM. A double-blind trial of prednisolone in epidemic bronchiolitis due to respiratory synyctial virus. Acta Paediat. Scand. 1969, 58:116-120.
26. Leer JA, Bloomfield NJ, Green JL et al. Corticosteroid treatment in bronchiolitis. Am. J. Dis. Child. 1969, 117:495-503.
27. Tal A, Bavilski C, Yohai D et al. Dexamethasone and salbutamol in the treatment of acute wheezing in infants. Pediatrics. 1983, 71:13-18.
28. Carlsen KH, Leegaard J, Larsen S, Orstavik I. Nebulised beclomethasone dipropionate in recurrent obstructive episodes after acute bronchiolitis. Arch. Dis. Child. 1988, 63:1428-1433.
29. Maayan C, Itzhaki T, Bar-Yishay E et al. The functional response of infants with persistent wheezing to nebulised beclomethasone diproprionate. Pediatr. Pulmonol. 1986, 2:9-14.
30. Bisgaard H, Munck SL, Nielsen JP, Petersen W, Ohlsson SV. Inhaled budesonide for treatment of recurrent wheezing in early childhood. Lancet. 1990, 336:649-651.
31. Brooks LJ, Gerd JA, Cropp A. Theophylline therapy in bronchiolitis. Am. J. Dis. Child. 1981, 135:934-936.

WANDERING IN DEMENTIA: POSSIBLE CAUSES AND AN APPROACH TO ITS MANAGEMENT

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SUMMARY

Wandering is a commonly encountered yet distressing symptom in dementia. It can endanger the life of the patient and cause great difficulties to the care-giver. Two cases of wandering, one in an elderly and the other in a middle-aged patient are described. The various causes and an approach to its management are also discussed.

Keywords

Wandering, Elderly, Dementia

INTRODUCTION

Wandering is a common and frequently serious behavioural change seen in dementia and it makes management of such patients difficult. There are several underlying mechanisms that could account for the wandering and these include an increased overall amount of walking activity, avoidance of being alone, loss of navigational ability, faulty goal-directed behaviour and diurnal rhythm disturbance.[1] An understanding of the various causes of wandering can help us in the management of this problem. Two cases of dementia with wandering behaviour, one in an elderly and the other a middle-aged patient, are described below.

CASE 1

TSP is a 75-year-old Chinese widow who presented with a one-year history of being irritable and quarrelsome. She kept leaving her home and getting lost. She was found by the Police and taken to Woodlands Home for the Aged four or five times over the past year. Her family members had tried to lock the doors of her home but she would use a hammer to break the lock and then leave home. She also gave away or lost money and accused others of stealing from her. She was sometimes not able to recognise her family members and was seen praying to her own portrait and once tried to feed the portrait. She slept poorly and was unable to tend to her personal hygiene.

Mental state examination on admission revealed a spritely old woman who was restless, agitated and disorientated in time, place and person. She had marked short and long term memory difficulties and confabulated a great deal. She was unable to count and recognise money and there was evidence of nominal aphasia.

Physical examination was normal and there were no neurological deficits.

Investigations did not reveal a reversible cause for the dementia and an electroencephalogram revealed generalised excessive slow wave activity which is consistent with senile dementia of the Alzheimer's type.

Registrar
Unit II, Woodbridge Hospital
Singapore 1954

She was treated with Thioridazine, with improvement of her agitation, and Lorametazepam which improved her sleep. She however continued to be disorientated and kept following her daughter around the house.

CASE 2

CHC first presented to Woodbridge Hospital at the age of 43 with a six-month history of being restless and pacing about continuously. He was unable to sit still for more than a few seconds and would slap his thighs continuously.

He was initially diagnosed to have an atypical psychosis but did not improve with anti-psychotic treatment. He instead developed impairment of memory and began to neglect his personal hygiene. An organic pathology was suspected but all investigations, including a CT Scan were normal. He was then treated with anti-depressants but again failed to improve. One year later, a repeat CT Scan showed prominent cerebral sulci, subarachnoid spaces and ventricles due to gross cerebral atrophy. He was then diagnosed to have pre-senile dementia and treated with Thioridazine to reduce his agitation. Two years following this, he became non-ambulant and incontinent.

DISCUSSION

Wandering in dementia may occur as a result of the following[1]:

1. Motor agitation leading to increased overall amount of walking activity
2. Avoidance of being alone
3. Disturbances of navigational ability
4. Diurnal rhythm disturbance
5. Faulty goal-directed behaviour

Specific brain lesions have been reported to lead to continuous pacing in animal studies[3]. One of the hypotheses regarding the increased motor agitation in dementia therefore is that it may be a direct result of brain pathology. However, this hypothesis requires confirmation by post-mortem studies. In both the cases described here, motor agitation was an important symptom. In TSP, it improved markedly with Thioridazine but improvement in CHC was minimal although it did improve with progression of the disorder. This difference in response may have been due to the fact that the elderly are more sensitive to the effects of anti-psychotics[7].

Avoidance of being alone has similarities with separation anxiety seen in children. It suggests that there might be a degree of insight in that the demented person is aware that he is not able to look after himself and so attaches himself to a care-giver. Once the agitation in TSP was controlled, she continued to follow her daughter around the house, asking if there was anything she could do to help at home.

Disturbances of navigational ability lead to a loss of one's ability to find one's way around and results in wandering behaviour. Faulty goal-orientated behaviour refers to excessive walking directed at an inappropriate cause e.g. visiting the same shop several times a day.

Patients who wander at night may do so as a result of a disturbance of diurnal rhythms as a result of brain damage. Swaab et al[5] have reported a decrease in cell numbers in the suprachiasmatic nucleus in patients with Alzheimer's disease.

Management of wandering behaviour should be directed at the cause and of these, motor agitation is the most easily treated. Anti-psychotic medication is used and Promazine and Thioridazine have less extra-pyramidal side-effects and would be the treatment of choice in the elderly. It is important to avoid high doses of anti-psychotics because this may cause akathisia which increases restlessness, confusion and drowsiness and may lead to falls.

It is important to ensure that the patient is kept occupied during the day and exercises regularly so that he utilises the excess energy that he has[8]. Day centres can play an important role here.

It is also important to keep the environment safe and to keep the doors locked. It may be necessary to get new and more complicated bolts and locks[6]. Hussian and Brown have found that placing masking tape in a grid pattern in front of exit doors is effective in a number of cases because demented patients misperceive two-dimensional patterns as barriers[2].

Elderly patients may need to go to bed later because they need less sleep and benzodiazepines with short half-lives like Loramet may be required to ensure a good night's sleep. Benzodiazepines with longer half-lives may cause drowsiness and confusion during the day. It may also be helpful to leave a night light on as this will reduce the confusion when waking up in the dark.

It is essential to get patients who wander, to wear a necklace or bracelet with the patient's name, address, telephone number and the statement "memory impaired"[4]. A bracelet that is securely fastened so that the patient cannot take it off is probably safer than a necklace. This will help anyone who finds the person if he gets lost. While some forgetful people find it useful to carry identity cards in their pockets or wallets, they are less easily accessible to others and the demented patient may throw it away.

When a demented patient insists on going out, it is often difficult to stop him and it is perhaps best to allow this, accompany him for a short distance, distract his attention and then bring him home.

Care-givers of demented patients often feel frustrated, fatigued, discouraged and overburdened. Day-care centres, support groups for carers and respite admissions are useful to alleviate such distress. Planned respite admissions are useful as they provide carers with breaks that they can look forward to.

In nursing homes, entrances and exits may be electrically monitored to prevent wandering patients from inadvertently going outside and potentially coming to harm. Wandering patients can wear non-obstrusive signaling devices which electronically alert staff to potentially harmful movements[6].

References

1. Hope RA and Fairburn CG. The Nature of Wandering in Dementia — A Community-based Study. International Journal of Geriatric Psychiatry 1990; 5:239-245.
2. Hussian RA and Brown DC. Use of two-dimensional grid patterns to limit hazardous ambulation in demented patients. J Gerontol 1987; 42(5):558-560.
3. Lanier LP, Petit TL and Zornetzer SF. Discrete anteriomedial thalamic lesions in the mouse: The production of acute post-operative hyperactivity and death. Brain Res. 1975; 91:133-139.
4. Mace NL and Rabins PV. The 36-Hour Day. John Hopkins University Press, 1981.
5. Swaab DF, Fliers E and Partiman TS. The suprachiasmatic nucleus of human brain in relation to sex, age and senile dementia. Brain Res. 1985; 342:37-44.
6. Verwoerdt's Clinical Geropsychiatry, third edition, Edited by David Bienenfeld. Williams & Wilkins, 1990.
7. Wattis JP and Hindmarch I. Psychological Assessment of the Elderly. Churchill Livingstone, UK, 1988 pp 180-184.
8. Coping with Dementia: A Handbook for Carers, Scottish Health Education Group, 1987.

INHALED CORTICOSTEROIDS IN BRONCHIAL ASTHMA

Omar B S T, MBBS(S'pore), MCGP (S'pore), FRACGP

INTRODUCTION

Until fairly recently, major emphasis was placed on the use of bronchodilator drugs in the control of asthma symptoms. These agents, especially ß2-agonists, still have an important role, particularly in episodic asthma. The emphasis in treatment has, however, now shifted and anti-inflammatory drugs, especially inhaled corticosteriods (ICS), are now recognised to be the most important form of therapy in most patients.

MODE OF ACTION

Corticosteroids appear to inhibit the chronic inflammatory process in asthma and have inhibitory effects on almost every step of the inflammatory response. The molecular mechanism of corticosteriods is still not understood in detail, although their most important action may be in inhibiting the synthesis of cytokines, peptide mediators which are involved in orchestrating the chronic eosinophilic inflammation in asthma.

CLINICAL EFFECTS

ICS given regularly reduce the inflammatory response in asthmatic airways, reduce airway hyper-responsiveness and control symptoms. The late asthmatic response is rapid, but ICS need to be given regularly for about 4 weeks before they block the immediate asthmatic response. ICS reduce asthma morbidity, improve the quality of life of patients, are likely to reduce the mortality from asthma if used correctly, and they may be capable of reducing the progressive decline in lung function which commonly occurs in asthmatic patients.

DOSAGE AND CLINICAL USE

There is now a wide range of dosage of ICS that can be prescribed to asthmatic patients from 100 to 2000 ug daily of beclomethasone diproprionate (BDP) or from 100 to 1600 ug of budesonide. Although low-dose BDP (up to 400 ug daily) was initially used as the standard dose to treat asthma and high-dose BDP (up to 2000 ug daily) as an oral steroid-spring therapy, it is now recognised that the right amount of ICS needed to control chronic asthma for each individual patient can span the whole dosage range.

ICS requirements may vary considerably both between patients and in any one patient from time to time. The beneficial effects of ICS are also related to the dose and most patients will respond irrespective of the particular characteristics of the disease, such as atopic status and age. A higher dosage of ICS may be required to normalize pulmonary function than to simply control symptoms or reduce the need for bronchodilator drugs. It is important to step down ICS therapy once control is achieved, while monitoring the patient carefully, so that the minimum dose is used for long-term control. Patients with asthma should however realise that from time to time an increase in steroid dosage (2 to 4 times the standard daily dose) is called for in the face of worsening symptoms, such as those induced by upper respiratory viral infections or by exposure to specific allergens.

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It has been traditional to administer ICS 3 or 4 times daily. Recent studies have shown that this dosing frequency may be reduced to a twice-daily daily regime, which can be equally as effective as 3 or 4 times daily provided the asthma has been well stabilized beforehand. This is helpful in maximising patient compliance. Use of a once-daily dosage does not appear to be effective. It has been suggested that whenever asthma enters an unstable phase a 4 times daily dose is more effective than a twice-daily one.

ICS may be used exclusively as the steroid to control asthma but in some patients they have a useful role in enabling the dose of oral prednisolone to be kept to a minimum and thus can be used in combination with prednisolone to control symptoms. A dose of about 400 ug BDP daily appears to be equivalent in potency to prednisolone in a dose of between 5 and 12.5 mg daily.

Topical steroid aerosols do not, however, have a role in the management of acute severe asthma because of the difficulties in achieving high therapeutic concentrations in the presence of severe asthma. Thus patients who require ICS therapy to control their symptoms must be advised to take it regularly.

It is important to realise that one reason for apparent failure of ICS therapy is related to lack of compliance. Some patients may have a genuine phobia of taking steroids in all forms and are afraid of taking this therapy for long periods of time. Others plainly forget to take their regular medication, particularly once symptoms have been brought under control. This is where education of the patient is essential towards reducing non-compliance; in addition, a regular check on inhalational technique is important.

METHODS OF DELIVERY

The use of valved spacer devices, such as Aerochambers®, Nebuhalers® and Volumatics®, attached to conventional metered dose inhalers has improved the efficacy of delivery of inhaled steroid aerosols to the airways. There is no need to coordinate inspiratory effort with actuation of the inhaler and there is significant reduction in the amount of steroid aerosol deposited in the upper airway, particularly in the pharyngeal and laryngeal areas. In addition, an increased amount of steroid aerosol is deposited within the lungs.

ICS can also be delivered in a dry powder form either as the pure drug (as via a Turbuhaler®) or mixed with a lactose carrier (as in the Diskhaler®). The advantage of the dry powder inhaler is that it eliminates the problems of coordination usually observed when conventional metered dose inhalers are not used in conjunction with spacer devices. Inhalation of the dry powder formulation appears to be as effective as the metered dose inhaler. The deposition characteristics of powder inhalers appear to be similar to those from a metered dose inhaler. Nebulizer solutions of BDP or budesonide are now available but their place in clinical practice remains to be determined, although they may be useful in infants and young children.

SIDE EFFECTS

Many side effects have been anticipated with use of corticosteriods aerosols but only the local ones affecting the pharynx and larynx appear to be important. At present, there is no evidence to show that steroid aerosols damage the epithelial layer of the trancheobronchial free. Indeed, steroid aerosols may normalize the damaged airway epithelium. There is also no evidence that ICS therapy promotes lung infection. However, it is first important to review the potential systematic side effects of ICS.

Suppression of morning cortisol levels can occur in adults taking more than 1500 ug daily, but in children suppression was observed even with doses as low as 400 ug daily. The clinical relevance of these small but statistically significant changes are not certain and that these are not in any way life threatening, whereas the clinical benefits are often very substantial.

This suppressing effect on the adrenal glands in children has led to concern about the possible adverse effect of ICS on growth. However, recent studies into the potential side effects of ICS therapy in children have produced very reassuring results. Todate ICS therapy in children has not

been implicated in causing clinically significant adverse effect on growth. Fear of growth suppression should never dominate management at the expense of good control of asthma.

Other side effects have been reported rarely and it is still uncertain whether high-dosage ICS cause alterations in calcium metabolism and osteoporosis. In the elderly, especially in high dosages, ICS may increase the risk of cataract formation and purpura.

Very large doses of ICS are foetotoxic and teratogenic in animals. However, normal doses of ICS appear safe in pregnancy, and this contrasts with the well-documented adverse effects of uncontrolled asthma on both mother and foetus. ICS can be used safely in lactating mothers.

Local side effects often contribute to lack of patient compliance. The prevalence of oral candidiasis is strongly related to total daily dosage of ICS and can be reduced by lowering the frequency of administration as well as the total number of puffs per day. The incidence of oral candidiasis can also be reduced by administering ICS with spacer devices followed by effective mouth rinsing with water.

Hoarsness and weakness of the voice (dysphonia) is a more frequent side effect. Dysphonia is more common and persistent in those who use their voice frequently in daily life, such as teachers and singers. Vocal cord myopathy is the main cause of difficulty with phonation, and results from local absorption of steroid deposited on the larynx. Although dysphonia is not related to candidiasis, it will improve with the same measures but, occasionally, rigorous voice rest is needed together with cessation of ICS.

In a few susceptible patients ICS delivered by conventional CFC inhalers may cause local irritation, cough and, perhaps most significantly, a possible reduction in bronchodilatation or even occasionally "paradoxical" bronchospasm, provoked by the freon content of the inhaler. These side effects may be attenuated by the prior use of ß2 agonists or by the inhalation of the pure drug delivered in a dry powder form.

CONCLUSION

The past decade has witnessed the solid implantation of inhaled corticosteriods therapy in the management of chronic asthma and there is increasing agreement that it should be introduced at an early stage of the disease to treat the underlying pathogenesis. The likely role of ICS in preventing irreversible structural changes and preventing the development of fixed airway obstruction, so frequently seen in the poorly controlled adult asthmatic, may be important in encouraging the regular use of these medications in their lowest effective dose on a long-term basis for children and even for patients with mild asthma. Since its introduction two decades ago, no serious side effects have been reported. Individual susceptibility probably plays a major role in the expression of side effects, and variability of aerosol deposition. Local and systemic side effects can be reduced if spacer devices are used and if the mouth is swilled out with water immediately after use of the inhaler. As we move into the next century, new ICS with greater topical potency but with more rapid metabolism and with even more efficacious methods of delivery will be introduced.

References

1. Barnes PJ, Persson CGA, (Ed). Asthma: Its Pathology and Treatment, Marcel Dekker, Inc. New York 1991.
2. Boyd G, (Ed). The Role of Inhaled Steroids in Asthma. Research and Clinical Forums 1986 Vol. 8 No. 2.
3. Clark TJH, Godfrey S, Lee TH, (Ed). Asthma, Chapman & Hall Medical, London, 1992.
4. Connolly K. Inhaled Corticosteroids in the Management of Asthma. Prescribers' Journal, 1992 Vol. 32 No. 3 pg 99-105.
5. Corticosteroids: Their biologic mechanisms and application to the treatment of asthma. An International Conference, Vienna, September 3 and 4 1988. Royal Society of Medicine Services Limited, U.K. 1989.

Multiple Choice Questions on next page.

Multiple Choice Questions

(1) In the treatment of bronchial asthma

A. ß2-agonists have useful clinical effects on chronic inflammation.
B. Corticosteroids have a direct effect on smooth muscle contraction.
C. ß2-agonists have mast cell stabilising effects.
D. Corticosteroids result in a fall of airway hyper-responsiveness.
E. If the patient needs to use inhaled ß2-agonist daily or more often than three times a week, an inhaled anti-inflammatory drug should be added.

(2) With regard to the use of inhaled corticosteroids in the management of asthma

A. They are of greatest benefit in acute severe asthma.
B. To be effective they should be administered four times daily.
C. They have to be given regularly for about 4 weeks before they block the immediate asthmatic response.
D. They are best avoided in children.
E. Gastrointestinal absorption does not occur.

(3) Known side effects of inhaled corticosteroids include

A. Oral candidiasis.
B. Hoarseness of voice.
C. Increased cough.
D. Frequent lung infections.
E. Severe osteoporosis.

(4) The following statements about inhaled corticosteroids are correct

A. Bronchospasm may occur.
B. There may be a small degree of transient growth retardation in some children.
C. Local side effects can be reduced by washing the mouth out thoroughly after use.
D. Systemic absorption from large droplets should be reduced by the use of spacer devices.
E. Optimal effect may not be seen for up to 2 months.

Answers

(1) C D E
(2) C
(3) A B C
(4) A B C D E.

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Further reading

1. INTERNATIONAL COMMITTEE OF MEDICAL JOURNAL EDITORS. Uniform requirements for manuscripts submitted to biomedical journals. Ann Intern Med 1988; 108:258-265.

2. Bailar III J C and Mosteller F. Guidelines for Statistical Reporting in Articles for Medical Journal(s). Ann Intern Med 1988:108:266-273.

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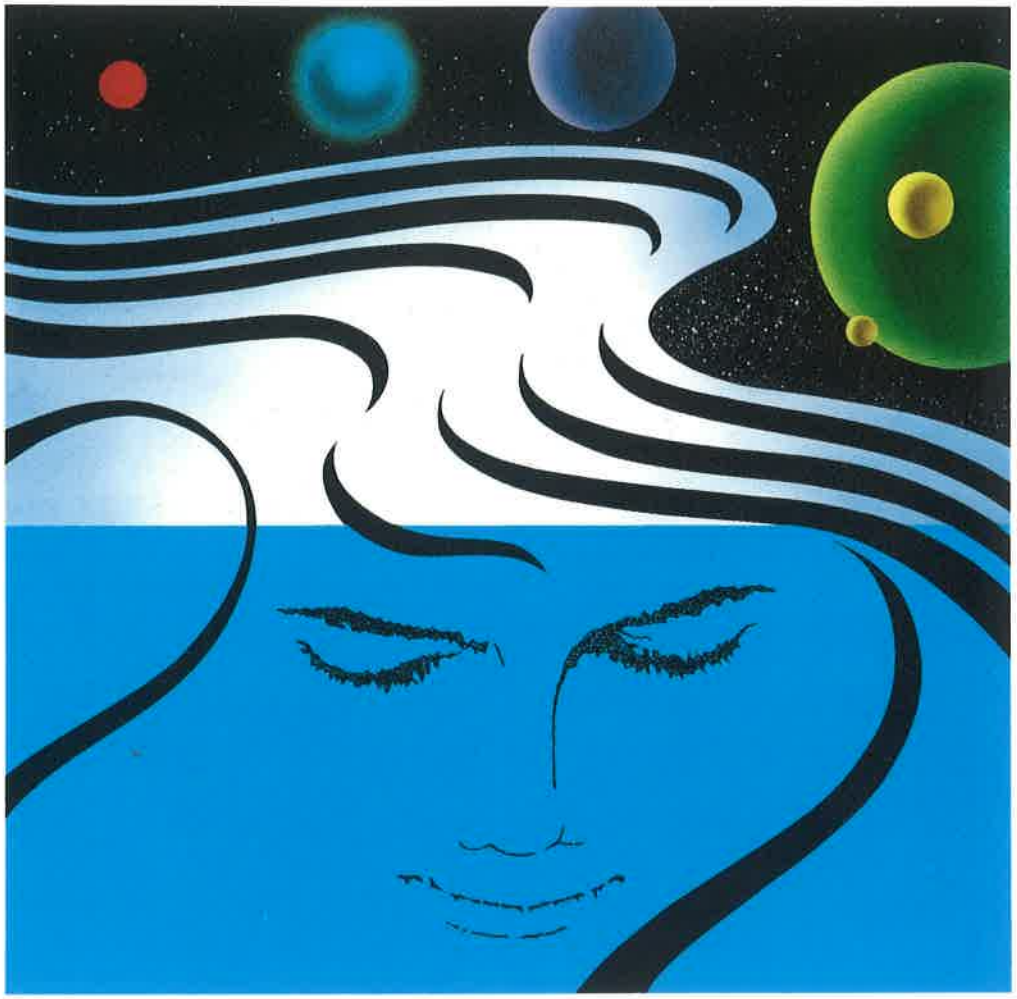

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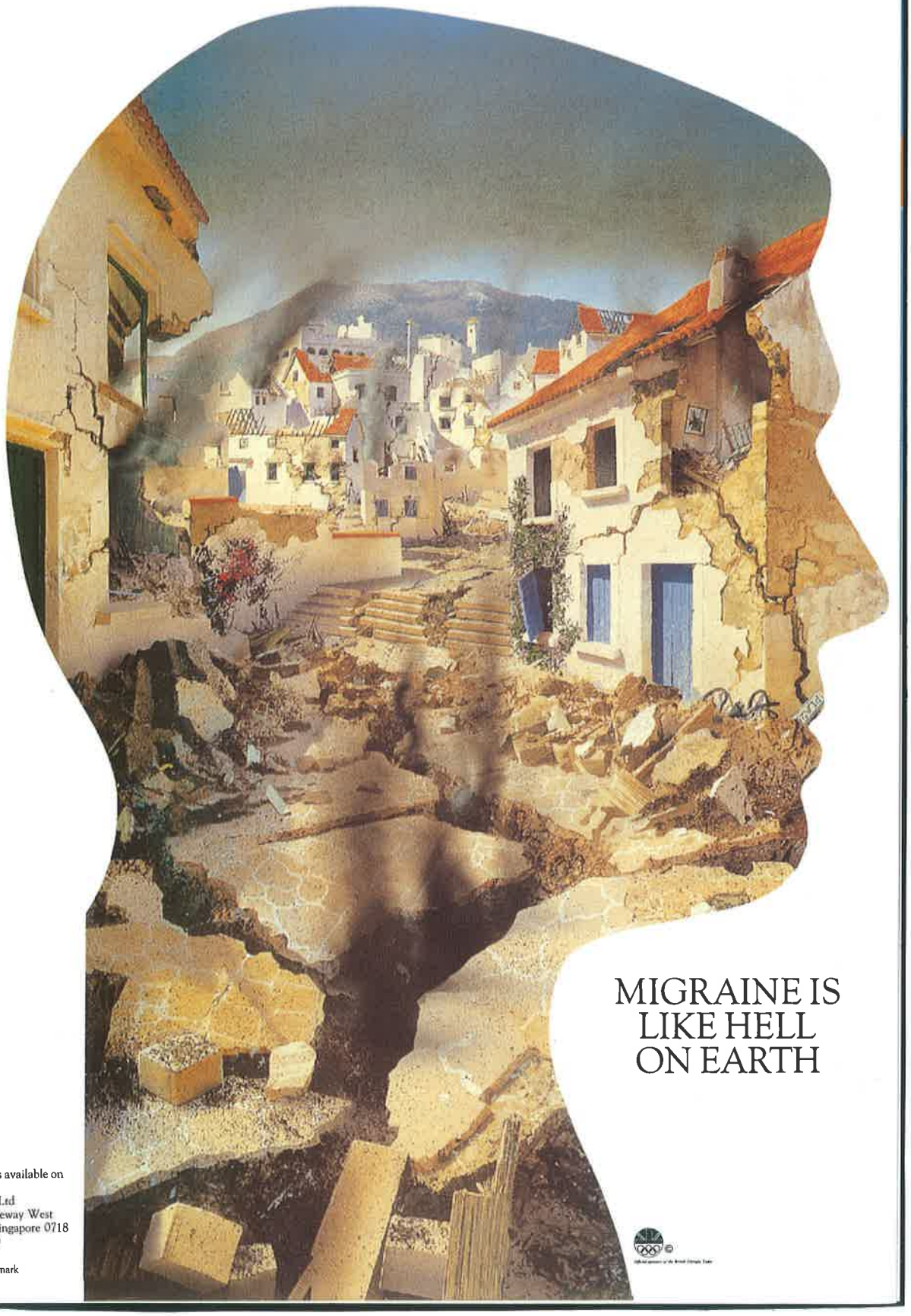

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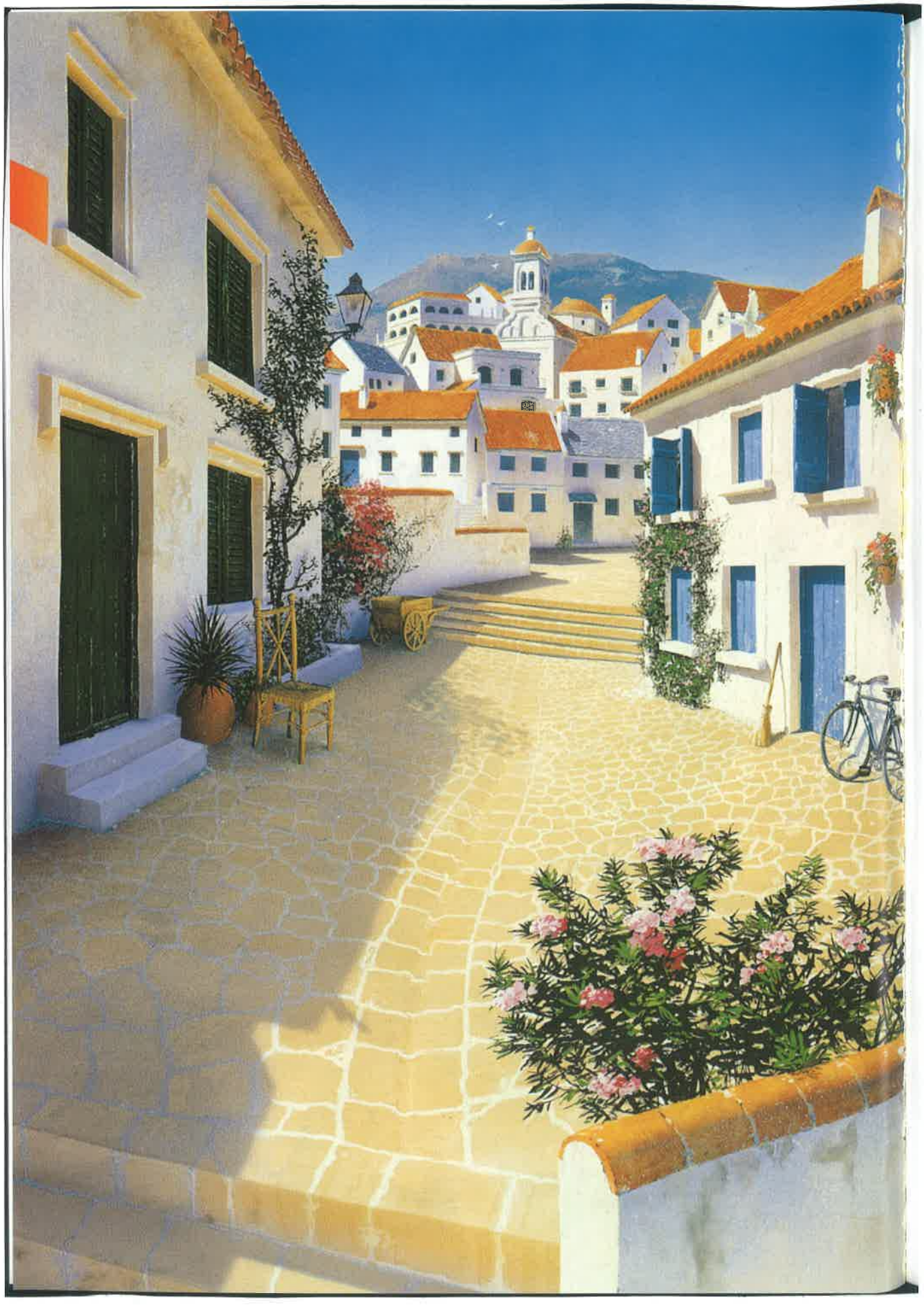

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Non-Benzodiazepine
Simply a more natural night's sleep
Superseding the barbiturates and benzodiazepines, Imovane is the first 3rd generation hypnotic. Imovane offers:
A full night's sleep
Ideal 5hr half-life provides a full night's sleep without residual effects.[1]
More natural sleep architecture
Measured by EEG, sleep induced by Imovane resembles natural sleep more closely than that induced by, for example, triazolam.[2-11]
Rapid onset of action
Sleep induction with Imovane is at least as rapid as that with benzodiazepines such as triazolam.[12]
Less "hangover"
Daytime psychomotor performance with Imovane is superior to that with benzodiazepines such as flurazepam.[13]
Less rebound insomnia
Compared with triazolam, the incidence of rebound insomnia is lower with Imovane.[14]
Prescribing Information
Indications:
Insomnia, including difficulties in falling asleep, nocturnal awakening and early awakening, transient situational or chronic insomnia, and insomnia secondary to psychiatric disturbances, in situations where the insomnia is debilitating or is causing severe distress for the patient.
Dosage:
Adult – one tablet of 7.5mg zopiclone at bedtime.
Elderly – start with ½ tablet. Increase to one if necessary
Side-effects and contra-indications
Mild metallic after-taste and some gastro-intestinal disturbances including nausea and vomiting. Use of zopiclone in pregnancy is limited, hence its use should be avoided if a safer alternative is known.
New non-benzodiazepine
Imovane®
Z O P I C L O N E
The new generation hypnotic
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