THE COLLEGE OF GENERAL PRACTITIONERS SINGAPORE

The SINGAPORE FAMILY PHYSICIAN

DIABETES MELLITUS II

- Pregnancy
- The Eye
- Diet in Special Situations
- Hypertension in the Diabetic

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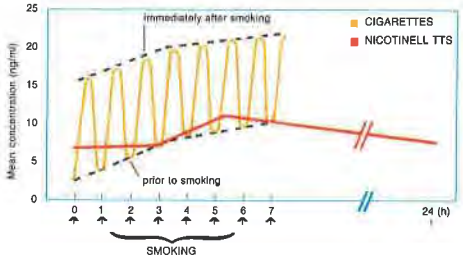

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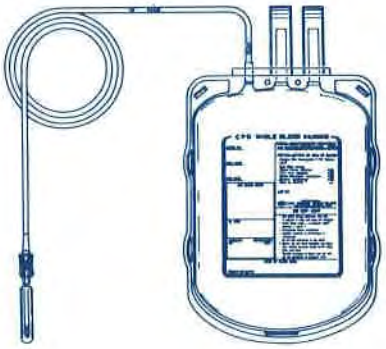

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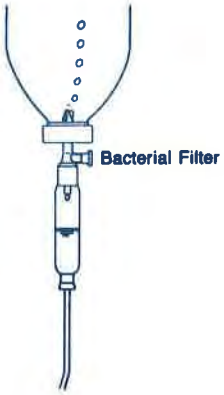
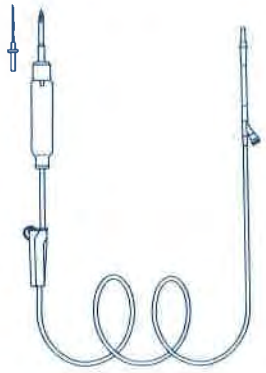


Bacterial Filter



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Further information available on request

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EDITORIAL

HEALTH CARE COSTS AND THE FAMILY PHYSICIAN

The escalation of healthcare costs in Singapore over the years is mainly due to increase in the cost of secondary and tertiary healthcare services, the cost of primary healthcare not having increased significantly. So, if family doctors services are not costing much more to the public, how is the Family Physician involved in helping to curb these costs?

First of all, we must remember that a decreased confidence in primary healthcare doctors, including family doctors, can cause the public to "doctor-hop" and further, directly consult specialists, sometimes even the wrong specialists, for their problems, thus increasing medical costs. Herein lies the importance of primary healthcare as frontline medical care. With good family physicians who can build up the confidence of the public and cement good doctor-patient relationships, patients will be more willing to use their services and seek their advice rather than rush off to specialists on their own. Thus, the family physician needs to be cost-effective, and for this, he needs proper training, continuing medical education and good practice management.

As our country progresses socially and economically, and our population becomes more educated and affluent, the public has greater expectations of better medical care. The new generations of healthcare technology and the improved healthcare facilities and services provided by both private and the government's restructured hospitals lead to increased consumer demand. Family physicians can help educate the public on the costs of different health services, and advise their patients on the prudent use of their Medisave funds, so that they do not ask for hospital admissions for tests and procedures which can be done as out-patients, and so that they do not use their Medisave funds to have more expensive treatment or wards that they may not really need.

Another area where the family physician can play a big part in helping to cut down healthcare costs for his patients is by practising preventive medicine and providing health education for his patients — to promote healthy lifestyles and to encourage his patients to have routine, regular medical checks for early detection of certain illnesses, e.g. diabetes and cancer, and for early detection of risk factors for disease e.g. hyperlipidaemia and coronary artery disease.

In this, the family physician's job would be made that much easier if the government were to allow the use of Medisave funds for health screening (perhaps with some element of cost-sharing to curb excessive demand for such screening). Such a scheme would mean more paperwork for the family doctor, and also the possibility of delayed payments, as has been experienced with the Medisave Funds for Hepatitis B Vaccination scheme, but this would be a small price to pay for a more healthy population.

MV

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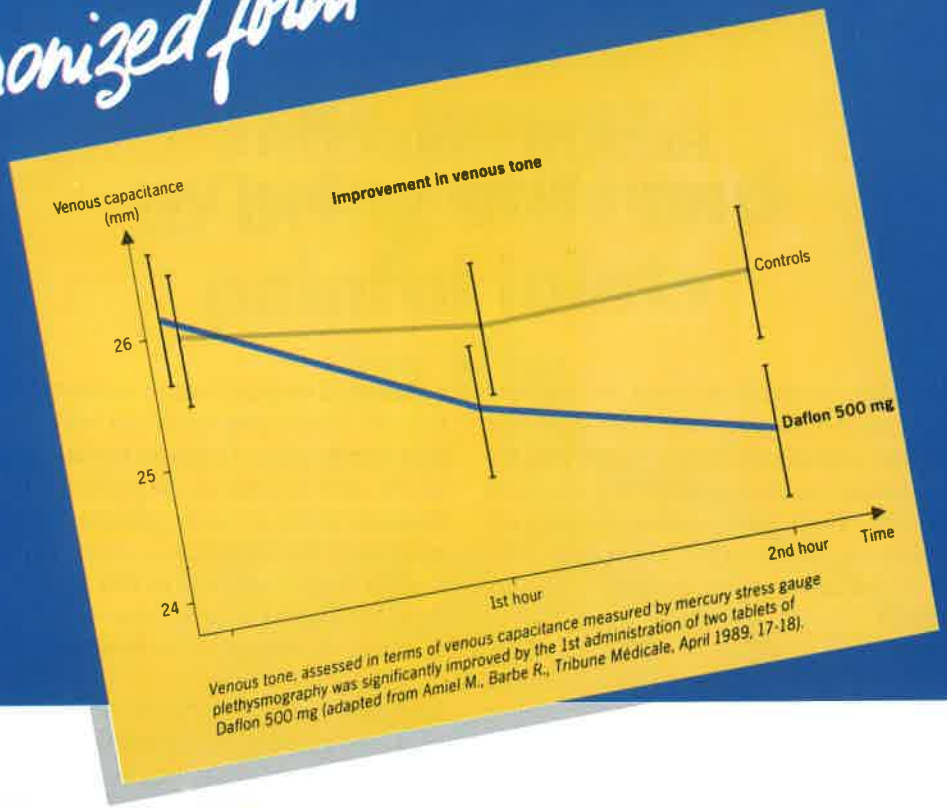

Venous tone, assessed in terms of venous capacitance measured by mercury stress gauge plethysmography was significantly improved by the 1st administration of two tablets of Daflon 500 mg (adapted from Amiel M., Barbe R., Tribune Médicale, April 1989, 17-18).

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– 2 tablets daily

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DIABETES MELLITUS IN PREGNANCY

Tey B H,
MBBS (Singapore), MMed (Int. Med), FAMS

The incidence of diabetes mellitus in pregnancy varies between 1.1% and 13.1% in previous reports. Perinatal survival has steadily improved over recent years and favourable outcomes can now be expected in greater than 90% of all diabetes-associated pregnancies, and greater than 97% in those without diabetic complications.

PRE-PREGNANCY PLANNING

As congenital malformations remain the most serious problem in the infants of diabetic mothers, the management of diabetic pregnancies should begin with pre-pregnancy planning. Tight metabolic control with self-blood glucose monitoring should be carried out at least three months prior to conception, through conception and throughout pregnancy.

CONTRACEPTION

During the period of pre-pregnancy planning, contraception should be used. Oral contraceptives are popular, 99% effective, and generally safe. Oestrogen raises blood sugars, blood cholesterol and triglycerides. In some women, oestrogen has caused an elevation of blood pressure.

Triphasic oral contraceptives are the best currently available with minimal metabolic effects, followed by progresterone-only oral contraceptives.

Other contraceptive methods (ie. IUDs, barrier methods, rhythm method) have their drawbacks and high failure rates.

Head and Consultant Endocrinologist
Department of Medicine
Changi Hospital

GESTATIONAL DIABETES

Gestational diabetes mellitus is defined as carbohydrate intolerance of variable severity with onset or first recognition during the present pregnancy. In relation to perinatal risk, gestational diabetes can be subdivided into two major groups. The first and milder group comprises those women with normal fasting blood sugars, but mild post prandial abnormalities, often controlled with diet alone. The second group comprises those with fasting hyperglycaemia despite diet therapy.

Because the onset of gestational diabetes most often is during the second or third trimester, the most propitious time to screen patients is between 24 and 28 weeks in the normally progressing pregnancy. A community-based screening program in Cleveland studied by Merkatz and colleagues revealed the 2-hour glucose test following a 75 gm glucose challenge as the most cost-effective. The researchers designated 120 mg/dl capillary blood as the top range for normal.

The O-GTT should be repeated 6 weeks post-natally to detect those patients with persistent glucose intolerance.

ANTENATAL MANAGEMENT — OBSTETRIC

Gestational age of the pregnancy is established accurately with abdominal and ultrasound examination. Monitoring of foetal health should include ultrasound examination and oestriol estimations. Amniocentesis for measurement of lecithin-sphingomyelin ratio (L/S) is used to determine foetal pulmonary maturity. Measurement of phosphatidylglycerol and phosphatidylinositol in amniotic fluid are also

done to confirm lung maturation. Patients should be hospitalised for obstetric reasons (ie. abnormal presentations, disproportion, hydramnios, etc) or for treatment of inter-current illness (ie, urinary tract infection, severe acidosis).

ANTENATAL MANAGEMENT — DIABETIC

A clinical monitoring protocol should be followed:

During early office visits, patient should be educated on diabetes mellitus and the various problems associated with diabetes and pregnancy. Education should include home blood glucose monitoring, dietary advice and the need for tight blood glucose control. Daily self-blood-glucose-monitoring (ie. four pre-meal blood glucose values per day plus two post-prandial blood glucose values in one of the days per week) should be done. Urine test for ketones should be done three times/week. Complete urinalysis at every clinic visit to detect pyuria and/or proteinuria. Weigh patient at every clinic visit and dietary changes made if indicated. Fundoscopy at each clinic visit. Initial and continuing evaluation of vascular complications should be done. Patients are classified in relation to perinatal risk assessment using the White's classification of diabetes in pregnancy or its simplified version.

Patient is reviewed monthly during the first and second trimester and biweekly during the third trimester. With complications, more frequent visits may be needed.

DIET

An additional 1.25 KJ (300 calories) is required above pre-pregnancy levels. The diet should contain at least 45% carbohydrate, additional protein (ie. 1.3/kg body weight in the mature female to 1.7g/kg body weight for adolescent pregnancy). Pregnancy is not the time for weight reduction in the obese. Insufficient calorie intake leads to ketonemia which may result in neurological damage to the foetus. Calcium, in the form of milk or milk products, and iron supplements are needed/ With a well-balanced diet, vitamin supplement is not needed.

INSULIN THERAPY

Use only human insulin for treatment. Intensive therapy with multiple insulin injections is the rule. An initial period of hospitalisation is often required to determine the optimum insulin regimen and to familiarise patient with the technique of self-injection of insulin.

TARGETS OF BLOOD GLUCOSE CONTROL

Fasting blood glucose	: ≤ 90 mg/dl
Pre-meal blood glucose	: ≤ 110 mg/dl
2-hour post-prandial blood glucose	: ≤ 120 mg/dl

If these targets of blood glucose control are achieved, perinatal mortality and morbidity are reduced to a minimum except congenital malformations.

DELIVERY

Elective delivery should be planned for about 38 weeks, later in gestational diabetes. The exact timing depends on a balance between obstetric and diabetic factors, but is never more that 40 weeks. Vaginal delivery should be aimed for. Caesarean section is considered for prolonged labour or in the presence of complications (in approximately 25% of cases). Possible indications for caesarean section include pre-eclamptic toxaemia, uncontrolled hypertension, abnormal presentations, disproportion, placenta praevia, foetal distress, failure of induction.

INTRA-PARTUM DIABETIC MANAGMENT

The aim is to maintain satisfactory blood glucose control, while avoiding hypoglycaemia. On the morning of the induction, omit the usual dose of insulin. An intravenous infusion of Dextrose/Saline solution is commenced.

Insulin therapy is continued/commenced with:

either:
subcutaneous insulin 4 hourly according to blood glucose values.

or:
intravenous insulin infusion, starting at 1 to 2 units per hour; subsequent rate is adjusted according to blood glucose levels, with monitoring of blood glucose levels hourly to 2 hourly.

With the removal of the placenta, insulin requirements drop dramatically. Insulin infusion should be discontinued to avoid hypoglycaemia. Post partum blood glucose values will serve as a guide in resuming insulin administration.

PROBLEMS OF THE NEONATES OF DIABETIC MOTHERS

The neonates of diabetic mothers are prone to develop transient metabolic abnormalities which include hypoglycaemia (related to foetal hyperinsulinism resulting from persistent maternal hyperglycaemia), hypocalcaemia and hyperbilirubinemia. Hyaline membrane disease results from changes due to prematurity. Long standing diabetics with vascular disease often give birth to small and underweight babies (ie. foetal microsomia). Foetal macrosomia id due to foetal hyperinsulinism, secondary to maternal hyperglycaemia. Both foetal microsomia and macrosomia increase the risk of perinatal mortality. Anoxia and delayed maturation of the liver enzymes cause polycythaemia and jaundice.

DIABETES AND THE EYE

Ang B C, MBBS (Singapore), FRACS, FAMS

Diabetes mellitus causes widespread damage to many parts of the body including the eyes. Often it is the ocular complications which frighten the patient because of fear of blindness.

The exact mechanism of the ocular complications is not known but certainly impaired carbohydrate metabolism is a factor. The ocular complications occur many years (10 - 20 years) after the onset of the disease. With improved management of diabetics who then live longer, the long term complications become manifest. Blindness from diabetic retinopathy is now an important problem in the developed countries.

Diabetic Retinopathy

Diabetic retinopathy is a common cause of blindness and now accounts for almost one-quarter of blindness registration in the western world.

The presence and degree of retinopathy seem to be more closely related to the duration of the disease than to its severity. Juvenile diabetics develop retinopathy within 20 years in 60-75% of cases.

A review of the concepts of pathogenesis of diabetic retinopahty may help understand the clinical presentation of the disease. The postulate is that high levels of blood glucose or its metabolites damage the retinal capillaries producing retinal ischaemia which in turn injures the retina and marshals tissue process of repair and this reparative process is haphazard which causes visual disturbance.

Eye Dept
National University Hospital
Singapore

In the early stage, there is vasodilation. With time, the vasodilation becomes chronic leading to structural and functional incompetence. This is seen as tortuous, irregular vessels., microaneurysms, retinal oedema, hard exudates, and intra-retinal haemorrhage. These are the clinical features of **background retinopahty** (or non-proliferative diabetic retinopathy - NPDR). When the macular area is involved, the vision deteroriates — a condition called **maculopathy**. Maculopathy can be due to localised leakage — called **Focal maculopathy**. This has a better prognosis and responds well to focal laser photocoagulation. Maculopathy can be **diffused** due to widespread leakage — the prognosis is not good but can still be treated with the laser. However, the third type — **ischaemic** maculopathy — is due to capillary non-perfusion of the macula and laser treatment will not help.

There is an intermediate stage of non-proliferative diabetic retinopathy called Pre-proliferative Diabetic Retinopathy in which there are signs of severe retinal ischaemia but no neovascularization or fibrosis. It is charactarized by many cotton wool spots (retinal infarcts), large blot haemorrhage, and retinal venous tortuosity and looping. Some opthalmologists will teat these cases with a limited pan-retinal photocoagulation (PRP) with the laser in an attempt to prevent progression to the proliferative stage.

The most serious retinopathy is Proliferative Diabetic Retinopathy (PDR). This is charaterized by neovascularization of the retina at the optic disc and /or retinal surface and on to the detached vitreous face. Because these vessels are fragile they easily rupture causing pre-retinal and vitreous haemorrhage with sudden blindness. Subsequently fibrosis occurs which causes tractional retinal detachment and blindness. In the early stage pan-

retinal photocoagulation (PRP) has been proven to prevent blindness in more than 60% of cases. With modern microsurgical techniques, vitreous haemorrhage and tractional retinal detachment can be treated (para plana vitrectomy) with reasonable success. However, the visual result is usually not good because of the damage already done to the macula by the disease. Early treatment is therefore important.

This leads to the need to detect retinopathy early. We have recommended that NIDD patitents should have their fundi examined when they are first diagnosed and if no retinopathy is found, they are reviewed yearly. For IDD, especially the juvenile type, they should be examined 5 years after onset and then yearly.

Screening for retinopathy can be done by photography of the fundi as most of the lesions are at the posterior pole. This will save on medical manpower. Patients with retinopathy or suspicious retinopathy can then be referred for more detailed examination by an ophthalmologist and treated if necessary.

Len Changes

True diabetic cataract if rare. Senile cataract in the diabetic is common and tends to occur at a younger age than in non-diabetics. Sudden change in refraction is sometimes seen in newly diagnosed diabetics undergoing metabolic control. This stabilizes in a few weeks.

Iris Changes

Glycogen infiltration of the iris pigment epithelium and sphincter and dilator muscles may cause difficulty in dilating the pupil.

Rubeosis irides of iris neovascularization is a sign of poor prognosis indicating severe ocular ischaemia.

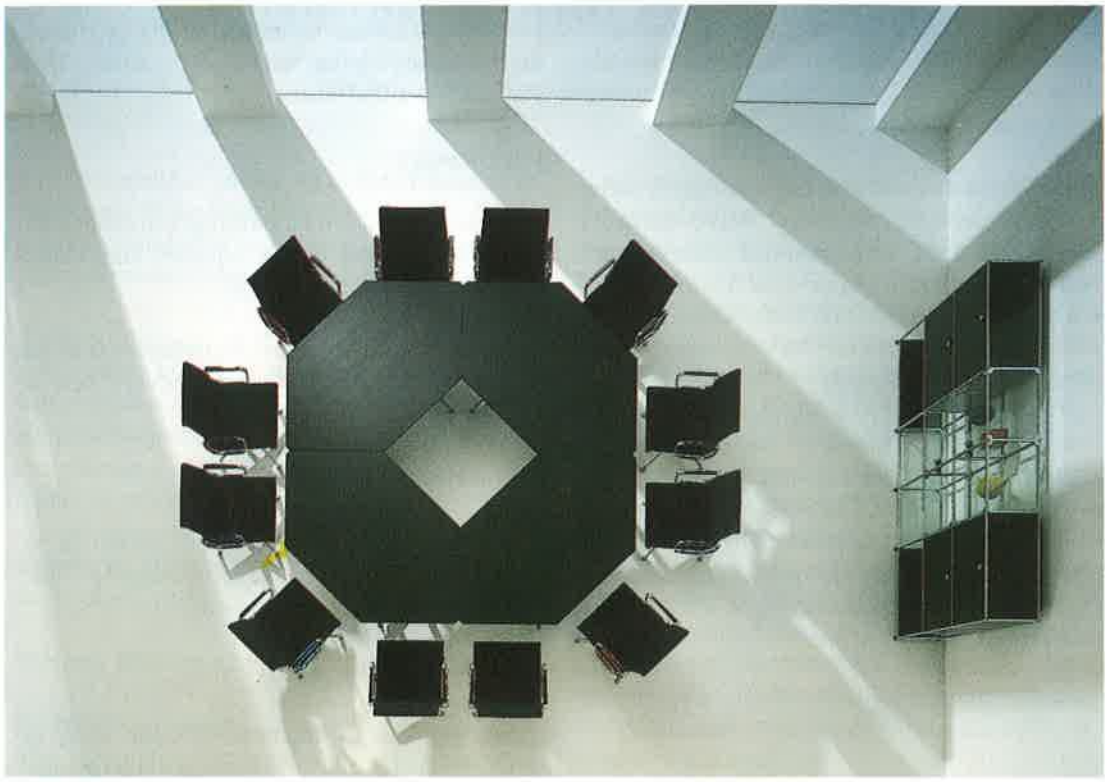
Extraocular maculopathy

Sudden onset of diplopia is not uncommon in diabetes due to cranial nerve neuropathy. This usually recovers in a few months.

Optic Neuropathy

Visual loss due to infarction of optic nerve is fortunately not common. No treatment is presently available to restore visual function.

There are forms which keep for ages!



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DIET IN DIABETES — SPECIAL SITUATIONS

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EATING OUT

Eating out has become a part of our lifestyle. Many of us eat out at least once a day for convenience. The various food outlets are: hawker stalls, canteens, cafeterias, fast food centres and restaurants. Diabetic people are no different from ordinary people and they too have to eat out. Like everyone else, they should be prudent and select the dishes wisely, bearing in mind the need to have a healthy meal plan. A knowledge of portion sizes, carbohydrate and fat content of commonly eaten foods is useful (See Appendix).

General points

As a rule, simply-prepared foods tend to be much lower in fat. Some points for everybody to remember when eating out are:

* Avoid oily and greasy foods.
* Avoid salty dishes and foods flavoured with MSG; instead select dished cooked with spices and herbs.
* Avoid sugary foods.
* Choose steamed, boiled, baked or grilled foods whenever possible.
* Have generous serves of vegetables; request for salads if these are available. Request salad dressing be placed on the side of the plate to allow controlled amounts to be eaten.
* Have fresh fruits for dessert.
* Drink Chinese tea or water.
* Limit alcohol to one or two drinks per day: choose 'dry' wines. If it is possible, avoid alcohol altogether.

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Hawker Foods

Food found in hawker centres, canteens and cafeterias are generally high in calories, fats and sodium and low in fibre. Some foods however, have lower calorie value than others and are therefore more preferable.

One should choose foods which are:

* steamed, boiled or baked, for example, fish soup, yong tau foo, roast/steamed chicken.
* watery and soupy, for example, noodles, porridge.
* bulky and crispy, for example, salads, rojak, popiah, fresh fruits, vegetables.

One should avoid foods which are:

* oily and greasy, as for example, fried noodles, fried bananas, fried carrot cake, laksa, curries, sambal, rendang.
* sweet and starchy, as for example, desserts (bubo chacha, chendol), cakes, nonya cakes, soft drinks.
* thick and creamy, as for example, gravies, sauces, curries (coconut based).

Fast foods

Most fast foods consist of beef, chicken or fish and are therefore high in protein. As they are usually deep-fried, they are high in calories and fats. They also provide a good percentage of Vitamin B

Table 12. Hawker foods: Preferred choices

Less preferred:	
1 plate fried kuay teow	- 600 Cals
1 bottle soft drink	- 150 Cals
1 bowl bubo chacha	- 300 Cals
Total	- 1050 Cals
Preferred:	
1 bowl beehoon soup	- 350 Cals
1 glass soya-bean drink	- 60 Cals
1 fresh fruit	- 60 Cals
Total:	470 Cals

complex. Beef-burgers, for example, are excellent sources of iron and zinc. The type of fat from fast foods is mainly saturated (more than 40%) as beef fat is usually used in frying. There is hardly any dietary fibre. Eating at a fast food centre can be made "healthier" by making appropriate food choices. One should avoid foods which are:

* deep fried, as for example, fried chicken, potato chips and pastries.
* high in simple sugars, as for example, soft drinks, desserts, apple pie and sundae.

The following are better food choices: jacket, baked or mashed potatoes, salads, corn-on-the-cob without butter, sandwiches, buns, coffee, tea and fruit juices. Wherever possible ask for grilled food, low fat milk or yoghurt.

Table 13. Fast foods: preferred choices

Less preferred:	
Whopper sandwich	- 628 Cals
Fried chips (large)	- 227 Cals
Coke	- 150 Cals
Total:	- 1005 Cals
Preferred:	
Cheeseburger	- 317 Cals
Fresh fruit	- 60 Cals
Yogurt	- 150 Cals
Tea without sugar	- 0 Cals
Salad with no dressing	- 28 Cals
Total:	- 555 Cals

Restaurant foods

Fine dining once in a while is not a problem so long as one is careful in selecting the food. A few simple rules are usefully.

Rules for choosing restaurant foods are:

* Before making a reservation find out whether special requests can be entertained.
* Study the menu carefully and select the healthier alternatives. Ask for less oil and salt; request that no MSG be added in the cooking.
* If there are sauces, request that they be served separately.
* If a low cholesterol diet is required, avoid organ meat (e.g., liver, brain), crabs, lobsters and prawns.

ALCOHOL

Alcohol is high in energy. One gram yields 7 calories. Table 14 shows the alcohol content of common drinks. It is quickly absorbed and can worsen hypoglycaemia in diabetics who are on medication especially if meals are missed or delayed. Tips for alcohol use are given in Table 15.

Table 14. Alcohol content of drinks

	Household measure	Wt (g)	CHO (g*)	Alcohol (g)+	kcal
Whisky	1 brandy glass (1 oz)	30	none	10½-12	75-78
Brandy, gin & rum	1 brandy glass (1 oz)	30	none	10½-12	75-90
Liqueurs, cordials	1 cordial glass (2/3 oz)	20	4-10	4-7	50-80
Beer, ale stout	1 glass (8 oz)	240	8-14	7-14	80-150
Light beer	1 glass (8 oz)	67	none	9-10	65
Wines (sweet)	1 wine glass (3½oz)	100	8-14	13-15	140-165
Wines (dry)	1 wine glass (3½ oz)	100	½-4	10-11	75-90

Source: Joslin: Diabetic Manual, 12th ed, 1989:321

Note: (*) 1g carbohydrate supplies 4 kcal, (+) 1g alcohol supplies 7 kcal

Table 15. Alcohol use for people with diabetes

* DO NOT drink alcohol if you are:
  - having uncontrolled diabetes
  - pregnant
  - on a weight reduction programme
  - not feeling well.
* Keep to 'dry' wines (e.g., white wines), sherry, spirits (eg., brandy, whisky, gin and vodka). Avoid sweet wines, regular beers and liqueurs as they have high content of calories and carbohydrates.
* A 50/50 mix of alcohol and water or mineral water halves the alcohol and calorie content. Do not use sweetened mixers.
* Drink slowly and immediately before/after or with meals.
* Limit to only 1 to 2 drinks per week if you are culturally used to drinking.

Table 16. Action to take on sick days

* Do not omit insulin or tablets even if unable to eat. See the doctor for advice early.
* If the usual diet cannot be tolerated, replace it with soft foods.
  * These should contain at least 150g of carbohydrates (10 carbohydrate portions) for the day, divided into 6 small meals spaced through the whole day.
  * Each of the following portions contains 15g carbohydrates (1 portion):
    1 small bowl porridge of oatmeal
    1 slice bread/toast
    3 cream crackers
    1 cup milk (250ml).
* If food cannot be retained because of vomiting, change to liquids. If the blood sugar is 3.5 mmol/l or less, liquid food should be provided.
  * Divide the 150g carbohydrate requirements (10 portions) into smaller portions to be taken every 15-30 minutes.
  * Each of the following items contains 15g of carbohydrates:
    3 teaspoons glucose or honey
    3 teaspoons ovaltine/milo/horlicks
    Half cup orange juice
    Half cup ice-cream
    1 cup regular soft drink.

SICK DAYS

Any illness can pose special problems for diabetics. The following is the information to be given to people with diabetes about sick days and the action that needs to be taken.

HYPOGLYCAEMIA

Hypoglycaemia may be the result of delayed or missed meals, insufficient food especially carbohydrates at meal or snack time, too much medication (tablets or insulin), too much exercise without extra food or drinking alcohol on an empty stomach. The patient may feel shaky, sweaty, hungry, weak, dizzy or confused. The blood sugar will be 3.6 mmol/l or lower.

Preventing hypoglycaemia

The risk of hypoglycemia is real in patients on insulin therapy. For them, meals and snacks should be taken regularly and should include the full carbohydrate allowance. Additional carbohydrate should be taken for extra activity such as strenuous games. If alcohol must be drunk, it should be taken with meals. Some sweets should be carried at all times.

Treating hypoglycaemia
The patient should be taught to take some sugar immediately when such a situation occurs. It can be one of the following: 3 teaspoons glucose/ sugar, 3 glucose tablets or half cup fruit juice/ regular soft drinks.

The above treatment should be repeated if the patient continues to feel weak and shaky after 10 to 15 minutes. This should raise blood sugar level. Follow by taking some longer-acting carbohydrate such as bread, biscuits or porridge.

HYPERGLYCAEMIA

Hyperglycemia can occur with too much food eaten, missed or insufficient medication, less activity/exercise than usual and infection.

Action to take
The patient is likely to be able to recognise the symptoms of thirst, polyuria and polyphagia as being due to uncontrolled diabetes. He should be taught that if the blood sugar level is persistently above 15 mmol/L (270mg/dl) he should seek treatment and call on the doctor as soon as possible.

He could do the following in the meanwhile:

* drink more unsweetened fluids to prevent dehydration
* administer insulin or tablets
* test urine for ketones
* replace solid foods with soft foods or nourishing fluids if he feels unwell.

EXERCISE

Exercise helps to control blood sugar levels and weight. While this is very beneficial, it is essential to adjust food and medication, especially insulin, so that diabetes remains under proper control. Table 17 gives the points for the patient to remember about exercise. The general guidelines of food adjustment for individuals with diabetes for exercise is given in Table 18.

Table 17. Points to remember about exercise

* DO NOT exercise when your blood sugar is above 15 mmol/L (270mg/dl).
* Monitor your blood sugar level to find out the amount of food adjustment needed.
* Carry some sweetened softdrinks/ sweets/candy just in case you need these because of hypoglycaemia

Table 18. Exercise and extra food

Exercise	Blood sugar mmol/L(mg/dl)	Extra food to take
Walking half mile, leisurely cycling for less than 30 minutes	< 5.5 (< 100)	1 slice bread or 1 fruit
	5.5 or > (100 or>)	no extra food
Tennis, jogging, swimming, golfing, gardening, bicycling	< 5.5 (<100)	1 slice bread with meat and 1 fruit
	5.5-10(100-180)	1 fruit or 1 slice bread
	10-15(180-270)	no extra food
	>15(>270)	DO NOT exercise

Table 19. Energy output for various activities

	For 1 minute	For 30 minutes	
ACTIVITY	Per kilogram body weight Cals	Male (64kg) Cals	Female (55kg) Cals
Sleeping	0.018	35	30
Sitting quietly	0.021	40	35
Knitting, sewing	0.022	42	36
Sitting (eating)	0.023	44	38
Standing (quietly)	0.026	50	43
Typing (electric)	0.027	52	45
Sitting (writing)	0.029	56	48
Typing (manual)	0.031	60	51
Ironing	0.033	63	54
Music playing - piano (sitting)	0.040	77	66
Music playing - violin (sitting)	0.045	86	74
Canoeing (leisure)	0.044	84	73
Cooking	0.045	86	74
Volleyball	0.050	96	83
Dancing (Ballroom)	0.051	98	84
Walking slowly - 4 km/hr (male)	0.051	106	-
Walking slowly - 4 km/hr (female)	0.055	-	96
Light housework	0.060	115	99
Fishing	0.062	119	102
Mopping floor	0.062	119	102
Food shopping (male)	0.058	111	-
Food shopping (female)	0.062	-	102
Cycling (leisure)	0.064	123	106

Ministry of Health. Your Personal Calorie Counter, 1992. MOH: Singapore *Cont'd*

Table 19 (cont'd). Energy output for various activities

	For 1 minute	For 30 minutes	
ACTIVITY	Per kilogram body weight Cals	Male (64kg) Cals	Female (55kg) Cals
Archery	0.065	125	107
Gymnastics	0.066	127	109
Table tennis	0.068	131	112
Walking briskly-6km/hr (female)	0.073	-	120
Walking briskly-6km/hr (male)	0.074	142	-
Golf	0.085	163	140
Badminton	0.097	186	160
Dancing (aerobic medium)	0.103	198	170
Canoeing (racing)	0.103	198	170
Tennis	0.109	209	180
Jogging (slow 7 km/hr)	0.112	215	185
Climbing hills (with no load)	0.121	232	200
Gardening (digging)	0.126	242	208
Swimming (crawl, slow)	0.128	246	211
Climbing hills (with 5kg load)	0.129	248	213
Football	0.132	253	218
Dancing (aerobic intense)	0.135	259	223
Basketball	0.138	265	228
Marching (rapid)	0.142	273	234
Digging trenches	0.145	278	239
Running (slow, 8km/hr) (male)	0.146	280	-
Running (slow, 8km/hr) (female)	0.153	-	-
Swimming (crawl, fast)	0.156	300	257
Swimming (breaststroke)	0.162	311	267
Swimming (backstroke)	0.169	324	279
Cycling (racing)	0.169	324	279
Running (med, 10km/hr) (male)	0.174	334	-
Running (med, 10km/hr) (female)	0.182	-	300
Judo	0.195	374	322
Running (fast, 12 km/hr)	0.200	384	-
Skin diving, moderate motion	0.206	396	340
Squash	0.212	407	350

Ministry of Health. Your Personal Calorie Counter, 1992. MOH: Singapore

TRAVELLING

People with uncontrolled diabetes should improve their blood sugar level before travelling.

Travel Tips

Advise the patient on the following:

* Check with the airline the type of meals which will be served. If there is a need for special meals, order them in advance.

* If on insulin, always carry some snacks such as plain biscuits, sweets or a small can of juice "just in case" a hypoglycemia reaction occurs. NIDDM patients, unless on insulin, are not in danger of hypoglycemia.

* Keep to local time while travelling. When the destination is reached, adjust meal times gradually to the new local time.

* Travelling across time zones: westwards

lengthens the day and eastwards shortens the day; travelling north or south does not affect the length of the day and is not a problem. For those on multiple doses of insulin, it will be necessary to see the doctor about the need to adjust dosages of insulin during travel. For those on oral medications and relatively small single doses (about 20 units) of insulin, there is no problem and no adjustment is needed.

PREGNANCY

To have a healthy baby, good blood glucose control is essential both before conception and during pregnancy. Planning meals for the mother should include the needs for the baby as well. Diet for a pregnant diabetic woman is the same as that of a non-pregnant diabetic except that sufficient calories-minerals such as calcium and iron, and vitamins must be provided. Generally women need about 1800-2200 kcals during pregnancy. The calorie needs can be determined by taking the body weight before pregnancy and multiplying it by the daily calorie needs shown in Table 20. Meals and snacks should be regular and should be divided into 3 main meals with 2-3 snacks. The bedtime snack is essential to prevent hypoglycemia during the night

Table 20. Calorie needs during pregnancy*

Weight status	Calorie needs (kcalories per pound)	Calorie needs (kcalories per kg)
Normal weight	13-16	29-35
Underweight	17	37
Overweight	11	24
Adolescent	18-21	40-46

Source. International Diabetes Centre, Living with Diabetes; Summer 1991. (*) Based on healthy body weight before pregnancy

Weight gain for a normal-sized woman is expected to be about 10 to 12kg during pregnancy and that of an overweight woman is about 6 to 10kg. An underweight woman is expected to gain between 12 to 14kg. Gradual weight gain is ideal. Pregnancy is not the time for weight reduction. Extra weight gain during pregnancy should be shed after delivery. Breast feeding accelerates weight loss.

CHILDHOOD DIABETES

The nutritional needs of a diabetic child are the same as that of a non-diabetic child. The meal plan should include sufficient nutrients for growth and development. As a general rule, a child of average size needs 1000 calories daily at age one, with 100 calories per day added each year up to puberty. Meals must be planned carefully so as to coordinate food with time of insulin injection. Meals and snacks should be eaten at the same time daily. In-between meal snacks are important to prevent blood glucose from dropping too low. As children are generally very active, rapidly absorbable carbohydrate should be available at all times.

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FAST FOODS

1 CARBOHYDRATE PORTION = 15G CARBOHYDRATE Food Item	Carbohydrate Portion	Measure	Wt (g)	Cals
McDONALDS				
Hashbrown potatoes	1	1 pc	53	131
Hamburger				
- small	2	1 no	102	255
- big Mac	3	1 no	215	562
French fries	2	regular	68	220
Apple pie	2	1 no	83	262
Chicken McNuggets	1	6 pc	113	288
Egg McMuffin	2	1 no	138	293
Cheeseburger	2	1 no	115	308
Quarter pounder	2	1 no	166	414
Quarter pounder with cheese	2	1 no	194	517
Fillet-o-fish	2 1/2	1 serve	142	442
KENTUCKY				
Chicken fried				
- wing	1	2 pc	112	362
- drumstick	1	3 pc	174	441
- sidebreast	1	2 pc	190	552
- thigh	1	2 pc	192	556
Mashed potatoes	1	1 serve	80	59
- with gravy	1	1 serve	86	62
Cole slaw	1	1 serve	79	103
Baked beans	1	1 serve	89	105
Potato salad	1	1 serve	90	141
Corn-on-the-cob	2	1 no	143	176
Kentucky fries	2	1 serve	119	268
CHURCH'S FRIED CHICKEN				
French fries	1	regular	85	138
Leg		1 pc	83	147
Fried chicken breast	1/2	1 pc	120	147
Onion rings	2	1 serve	-	280
Wing, breast	1/2	1 pc	135	303
Thigh	1/2	1 pc	120	306
Hot dog	1 1/2	1 serve	-	320
Hot dog with cheese	1 1/2	1 serve	-	330
Fish filet	3	1 serve	-	430
Fish filet with cheese	3	1 serve	-	483

Sources: Marion J Franz. Fast food facts, 1990,
Peggy Stacey, Diana Osler & Allen Borushek, The Family Diet, 1988.

Cont'd

FAST FOODS

1 CARBOHYDRATE PORTION = 15G CARBOHYDRATE Food Item	Carbohydrate Portion	Measure	Wt (g)	Cals
PIZZA				
Thin-n-crispy pepperoni	3	1/2 of 10"	-	430
Thin-n-crispy cheese	3 1/2	1/2 of 10"	-	450
Thick-n-chewy cheese	5	1/2 of 10"	-	560
Thick-n-chewy pepperoni	4 1/2	1/2 of 10"	-	560
LONG JOHN SILVER'S				
Dinner w/fries, seafood salad w/crackers	1	1	-	406
Fish and fries	4	3	-	853
Tender chicken	4	3	-	885
Dinner w/fries, slaw, shrimp & fish	5	1 fish 3 shrimps	-	917
BURGER KING				
Salad bar no dressing	-	1	-	28
Chef salad	-	1 order	-	180
Chicken tenders	1	6 pc	-	204
French fries	1 1/2	regular	-	227
Onion rings	2	1 order	-	274
Hamburger	2	1	-	275
Breakfast croissant	1 1/2	1	-	304
Cheeseburger	2	1	-	317
Whopper sandwich Junior	2	1	-	322
Chicken bundles	2	1 order	-	410
French toast sticks	3	1	-	499
Whopper sandwich	3	1	-	628
Chicken specialty sandwich	4	1	-	688
Whopper sandwich w/cheese	3	1	-	711
CAKES, SCONES				
Sponge Cake, plain	1	1pc	30	95
Swiss roll	1 1/2	1pc	40	120
Pancake-plain	1	4" diam	50	120
Scone-plain	1	1 no	30	105
Scone-sultana	1 1/2	1 no	35	130

Sources: Marion J Franz. Fast food facts, 1990,
Peggy Stacey, Diana Osler & Allen Borushek, The Family Diet, 1988.

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32% of patients free of panic attacks during first week[1]

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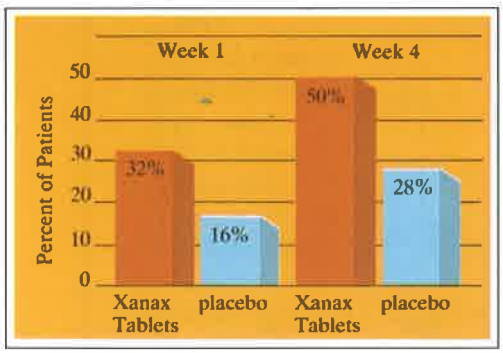
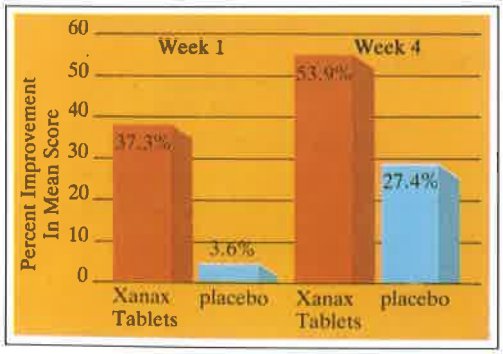


Significant improvement on Physician's Global Evaluation from first week[2]

Improvement in Physician's Global Evaluation



Availability: Xanax Tablets are available as 0.25mg (White), 0.5mg (peach), and 1mg (lavender) scored, ovoid-shaped table in bottles of 100, 500.

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THE TREATMENT OF HYPERTENSION — WITH SPECIAL REFERENCE TO THE DIABETIC PATIENT

Chew L S W, MBBS (Singapore), MRACP, FRACP

The aim of the treatment of hypertension is to lower the blood pressure. Raised blood pressure results in damage to end organs:

(i) **On the small arteries and arterioles:**
The hall mark of vascular damage is fibrinoid necrosis of the resistance vessels. These include the small arteries and arterioles. The consequences of fibrinoid necrosis are increased resistance to blood flow and haemorrhage e.g. cerebral haemorrhage and retinal haemorrhage.

(ii) **Left ventricular hypertrophy:**
Besides promotion of coronary atherosclerosis and ischaemic heart disease, left ventricular hypertrophy results from increased resistance to blood flow. Left ventricular hypertrophy may be the cause of sudden death from ventricular arrhythmia. In fact, left ventricular hypertrophy is a better predictor of morbidity and mortality than the level of hypertension.

(iii) **Kidney damage:**
The kidney itself may be the initiator of hypertension e.g. chronic glomerulonephritis, chronic pyelopephitis) or may itself be severely damaged by the chronicity and the level of the blood pressure.

Head and Consultant Physician
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Hence in the treatment of hypertension, special study must be made of the kidney status — as either treatment of the kidney disease or hypertension, essential or secondary to kidney disease, may be the course to take for the management of the raised blood pressure.

Intrinsic to the kidney is the neurohormonal Renin-Angiotension-Aldosterone system. This may often be the cause of the hypertension e.g. renal artery stenosis from unilateral renal artery atherosclerosis. The system, too, may be aggravated by falls in blood volume from treatment by diuretics or increase in peripheral resistance by propranolol. Certainly, in the hypertensive diabetic who is prone to kidney disease (diabetic kidney), renal artery atherosclerosis or renal infection, it is best to use medications that do not aggravate the intrinsic renal neurohormonal system or compromise renal blood flow. ACE-inhibitors and calcium blocking agents fulfill these criteria. ACE-inhibitors directly suppress the production of angiotension II - the hypertensive agent. Calcium blockers diminish response of vascular smooth muscle cells to angiotension II stimulation.

In the diabetic patient, too, there are problems associated with hyperinsulinaemia and dyslipidaemia. Chlorothiazide and beta-blockers have been shown to increase insulin-resistance and aggravate dyslipidaemia (raise serum cholesterol). It is, therefore, necessary

and best to use ACE-inhibitors, which have properties of reducing insulin resistance (e.g. captopril and perindopril). Calcium blockers are neutral in this respect, neither raising or lowering insulin resistance or lipid levels.

ACE-inhibitors have cardiac and renal protective effects. They improve cardiac failure and have also reduced left ventricular hypertrophy with their use. In the diabetic patient with early renal functional deficiency as shown by the presence of microalbuminuria (>300 ugm/dl), ACE-inhibitors have reversed microalbuminuria. With gross albuminuria, a 1 year trial with perindopril has prevented deterioration of renal function i.e. no increase in gross albuminuria. Calcium blockers (nifedipine) in one trial paper, was shown to cause deterioration in albumin excretion.

Finally, the greater consideration, apart from cost of therapy, is patient compliance. The hypertensive patient has a chronic disease state that requires regular and daily medication. A once-daily drug regime has shown the greater compliance.

TETANUS — A FORGOTTEN DISEASE

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Sin F L K M, MBBCh (Belfast), MRCP (Ireland), FAMS
Chew L S W, MBBS (Singapore), MRACP, FRACP

With the adoption of a very comprehensive programme of immunization for the population of Singapore, tetanus has become a rare disease. Not that it was previously common, but rather that it was seen frequently enough to be recognized by medical students and doctors. Doctors, too, were conscious of the disease such that they would liberally immunize any one who suffered a wound with an injection of tetanus toxoid. It was even not unusual to see patients with bee stings being immunized with tetanus toxoid! Today, whole generations of medical students go through a medical education without even seeing a case of tetanus. The clinical picture of tetanus is quite obvious, with risus sardonicus, muscle spasms and board-like rigidity of the abdominal muscles. The site of injury may be trivial and the injury may have healed. This unusual clinical picture of a patient in total spasm of both his flexor and extensor muscles is quite frightening and life threatening. The necessity of his admission to hospital for management is without doubt. But really the morbidity of tetanus, and often, the mortality associated with this disease need not happen. We should remember to immunize all our patients who have suffered injuries with tetanus toxoid. We describe, below, a patient who developed tetanus from a very trivial penetrating wound. He was not immunized with tetanus toxoid when he was first treated for the injury.

Case Study:

P K is a 33 year old Thai worker in Singapore. Six days prior to this admission, he was admitted for wound debridement as he had stepped on a nail. He was given eusol dressing and discharged after 1 day's stay. He was not given tetanus toxoid immunization. At admission, he complained of difficulty of swallowing, stiffness of the neck, inability to open his mouth and abdominal pain. He complained of difficulty in breathing. It was obvious that he had the features of risus sardonicus and spasm of his muscles caused by tetanus toxin. His foot wound had healed.

He went into muscle spasms of the whole body with the slightest stimulation. His respiration would cease during these episodes. As these spasms were life threatening, he was intubated and paralysed with d-tubocurare. He was given antitetanus immunolglobulin 300 units, tetanus toxoid and penicillin. He remained in intensive care for 32 days before it was deemed safe to take him off his controlled respiration and paralysis. He was discharged to the general ward with residual muscles in tetanic contraction after 35 days' stay in intensive care.

The last large study of tetanus in Singapore was in 1968. Then Dr KH Chee et al reported on patients with tetanus in whom tetanus toxoid was used in conjunction with antitetanus immunoglobulin. The patients then were sedated with valium and phenobarbitone and nursed in a darkened room to reduce external stimuli that would induce tetanic spasm. Over the past two years, seven patients were admitted to hospitals in Singapore for tetanus. All the patients were Thai. The question one invariably would ask is why had tetanus only affected Thai foreign workers? We know that Clostridium tetanus spores are ubiquitous, occurring in soil that has been contaminated by human faeces or animal manure. It is very likely

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that these Thai foreign workers, who lived in less than hygenic construction sites, had their wounds contaminated with animal manure or human faeces. But above and beyond this, they were probably not immunized against the disease in childhood or adult life. Again, secondary immunization too could have been omitted as in this patient.

Tetanus in a disease that could affect all ages of life. Neonatal tetanus was once common enough in the recent past. It was the practice of village midwives (bidans) to sever the umbilical cord of the new born with a sharpened piece of bamboo. This was thought to be the cause of the introduction of tetanus spores to the umbilical stump and the eventual onset of neonatal tetanus. Neonatal tetanus was invariably fatal.

Amongst adults, tetanus was common amongst farmers who in the past used human faeces to fertilize the land. But with the use of chemical fertilizers, newer methods of farming, and loss of farm land to industrialization, tetanus is virtually not seen any more. Again, not always is the site of infection obvious. In the patient described, the wound was in the left foot, but the infection was not obvious as it had healed. In the past, the site of tetanus infection had also been reported to occur in the middle ear. The infection had been introduced through ear-wax clearing with contaminated wooden spicules.

In all patients with tetanus, immunization with tetanus toxoid is mandatory. The infection itself does not produce enough toxin to result in immunity of the patient to the disease. It also necessary to complete a course of monthly injections of tetanus toxoid for 3 months to achieve good immune response. In addition, it is always necessary to secondarily immunize a person with an additional dose of toxoid when he is injured. One of our late biochemistry professors cut himself with a can opener. This seemingly trivial injury was to result in tetanus infection. He unfortunately succumbed.

Death is not an uncommon event in tetanus infection. During muscle spasm induced by tetanus toxin, both flexor and extensor muscles go into severe and violent contraction. Respiration stops, bone fractures may occur and inhalation of saliva or food may cause respiratory embarrassment. But really none of these should occur if we remember to secondarily immunize all patients with tetanus toxoid. The cost per dose of tetanus toxoid for secondary immunization is less than 50 cents!

FACIAL PAIN

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SUMMARY

The proper management of patients with facial pain, headache and neck pain involves a firm multidisciplinary understanding of the nature and treatment of their causes. This article only discusses facial pain, with causes of headache and neck pain omitted although the latter two may cause symptoms ovelapping with the former. With this short discussion, it is hoped that readers will appreciate the role of the different disciplines including medicine, surgery, and dental surgery; and the need to look beyond the confines of a particular region in order to reach an accurate diagnosis and provide proper care when managing a patient with facial pain.

Keywords

Facial pain

The recognition of the causes of facial pain, an essential precursor of treatment, demands an understanding of certain fundamental principles about pain generally[1].

INTENSITY OF PAIN. This depends on:
(1) The strength of the stimulus causing it;
(2) The area stimulated, e.g. the cornea is more sensitive than the skin; and
(3) The sensitivity of the 'sensorium' which varies with each individual's threshold for pain.

REACTION TO PAIN. This is not necessarily related to the strength of the stimulus. It varies with the psychical, physical and metabolic state of the victim and many other factors. Facial pain tends to produce stronger reactions than pain in more caudal areas of the body, partly because the face seems to be a more intimate part of one's personality and partly, no doubt, due to the denser innervation of the face and head.

REFERRED PAIN. Irritation of a viscus often produces pain which is felt not in the viscus itself but in some somatic structure that may be a considerable distance away. Such pain is said to be referred to the somatic structure. Deep somatic pain may also be referred, but superficial pain is not. Obviously, a knowledge of referred pain and the common sties of pain referral from each of the viscera is of great importance to the physician. Perhaps the best-known example is referral of cardiac pain to the inner aspect of the left arm. Additional instances abound in the practice of medicine, surgery, and dentistry. However, sites of reference are not stereotyped, and unusual refence sites occur with considerable frequency. Heart pain, for instance, may be purely abdominal, may be referred to the right arm, and may even be referred to the neck.

QUALITY OF PAIN. This is classified into two types:
(1) superficial; and
(2) deep.

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Superficial pain produced by a brief stimulus, e.g. pin prick, cut, heat, cold, faradic current, etc. is felt as a sharp pain varying in intensity with the strenth of the stimulus and the patient's threshold for pain, and is accurately localised. Deep pain has a dull, aching quality and is often accompanied by nausea and changes in pulse rate; its localisation is imprecise and often vague.

The classification of pain into superficial and deep is not explicit, for all shades and combinations occur. For practical purposes it can be said that superficial pain arises from skin, buccal mucosa and periosteum, and deep pain from bone, joint, muscle, blood vessels and viscera: the latter is usually 'referred'. Pain arising from the air sinuses tends to be deep in quality though in the case of the frontal and maxillary sinuses it is related and localised to a great extent to their sites; whereas from the ethmoidal and sphenoidal sinuses pain tends to be felt at the base of the nose and the temporal regions.

WORKING CLASSIFICATION OF THE CAUSES OF FACIAL PAIN[2]:

Primary neuralgias
Secondary neuralgia - intracranial
- bony skullbase, and
- extracranial causes

Local causes - diseases of paranasal sinuses
- diseases of the teeth and jaw
- diseases of the ear and referred otalgia
- diseases of the eyes
- diseases of the temporomandibular joint
- fibromyalgia syndrome
- diseases of the salivary glands
- post-traumatic pain

Facial pain of vascular origin
Atypical facial pain

NEURALGIAS

'Neuralgia' is often used by the public as a synonym for pain but it should be confined to pain which is felt in the area of distribution of a sensory nerve — whether the noxious stimulus is applied to a nerve trunk, posterior spinal root or ganglion. On this basis two forms of neuralgia can be differentiated clinically[3].

(1) Primary; and
(2) Secondary

Characteristics of primary neuralgia:

1. It is NOT associated with any objective signs of impaired nerve conduction.
2. It has characteristic qualities — paroxysmal, shock-like, lightning pains like the stab of a knife or a red hot needle, which last only seconds, but with remissions in between which may be so short as to be overlooked by the patient who will complain of the pain being continuous unless directly questioned. The pain is excruciatingly severe.
3. There are 'trigger zones' which when stimulated, e.g. by touching, brushing, or eating, precipitate a paroxysm of pain. The 'trigger zones' tend to be localised and since they are less likely to be stimulated during sleep, the nights are usually freer from pain than the days.

The cranial nerve in which primary neuralgia occurs most commonly is the trigeminal, but is occasionally appears in the glossopharyngeal, the facial and superior laryngeal. In all these nerves, ganglia are found in their first sensory pathway to the central nervous system and the frequency of neuralgia is related directly to the size of these ganglia. Very occasionally the 'tic'-like pain of a primary neuralgia recurs as the presenting sign of a pathological lesion, e.g. from compression of the Gasserian ganglion by a tumour, granuloma, or aneurysm in the middle or posterior cranial fossa, but as a rule this stage is short-lived, and signs of motor or sensory paralysis appear. In slowly progressive lesions though, e.g. multiple sclerosis, cranial hyperostosis, Paget's disease, etc., the primary neuralgia may persist for a longer period without signs of nerve dysfunction though the causative lesion is usually unmasked easily.

Many explanations have been advanced to explain the mechanism of primary neuralgia. It has been suggested that it is analogous to epilepsy, namely a sudden, explosive, irregular discharge of energy from the ganglion, hence the value of anticonvulsive drugs, e.g. carbamazepine ('Tegretol') in trigeminal neuralgia.

Characteristics of secondary neuralgia:
It is due to a gross pathological lesion directly involving the nerve (e.g. trauma, neoplasm), posterior root (e.g. tabes dorsalis) or ganglion (e.g. herpes zoster). Three features characterise secondary neuralgia.

(1) The pain is usually a mixture of superficial and deep; sometimes it is pricking and burning but usually it is boring and aching; it is often continuous, with frequent exacerbations and rare remissions;
(2) Associated with the pain are objective signs of interruption of continuity of the nerve, viz. anaesthesia, paresis, muscle wasting, diminished or absent reflexes, or trophic changes; and
(3) These objective signs tend to progress gradually and involve neighbouring structures.

PRIMARY NEURALGIAS

1. **Trigeminal neuralgia**
   Trigeminal neuralgia or tic douloureux is the most frequent disorder affecting the trigeminal nerve[2]. It is characterised by a sharp lancinating pain that usually involves the distribution of the second and third divisions of the nerve. Two subtypes of trigeminal neuralgia are recognised: an idiopathic (primary) type with a mean age of onset of 52 to 58 years, with a female to male ratio of 3:2; and a symptomatic (secondary) type (conveniently discussed here) caused by trauma, tumour, vascular compression or demyelinating diseases. The age of onset of the symptomatic type is 30 to 35 years. Surgery is often necessary to treat the symptomatic type. The idiopathic type is treated with a variety of medications, including carbamazepine, phenytoin, or clonazepam; a response rate of about 50% is common. Trigeminal neuralgia may rarely be combined with hemifacial spasm or glossopharyngeal neuralgia. Patients with trigeminal neuralgia should be thoroughly investigated with either computerised tomography (CT) scans or magnetic resonance imaging (MRI) to rule out any causative factors prior to placing them on a medical treatment.
2. **Post-herpetic neuralgia**
   It is a most undesirable sequel of herpes zoster infection. Chronic pain following zoster outbreak is reported in 16.5% of patients and in 47.5% of patients over 70 years old. An autopsy study showed the loss of large inhibitory fibres with intact small (excitatory) fibres. This apparent loss of nociceptive inhibition is theorised as the cause of the pain[2].
3. **Glossopharyngeal neuralgia**
   This neuralgia is much rarer than trigeminal neuralgia and commonly overlooked. It is similiar to tic douloureux, except it occurs in the distribution of the glossopharyngeal nerve. These patients experience lightning-like, severe paroxysms of pain starting in the tonsillar area and the base of the tongue and radiating into the ipsilateral ear. The pathologic features of this condition are similiar to those of tic douloureux, and the disorder is treated in a similiar fashion.
4. **Geniculate neuralgia**
   Geniculate neuralgia arises from the geniculate ganglion of the facial nerve. Although this nerve is predominantly motor in function, it has a sensory component which is well recognized in Ramsay Hunt's syndrome (geniculate herpes) where the facial paresis is associated with pain and rash in the external auditory meatus. In geniculate neuralgia pain is felt here, just anterior to the ear, and occasionally in the tongue and palate. Its character is typical of primary neuralgia and it is cured by section of the nervus intermedius.
5. **Superior laryngeal neuralgia**
   Superior laryngeal neuralgia is usually felt deep in the throat, base of tongue and supraglottic portion of the larynx, though occasionally in the neck, lower face, and gums.
6. **Sphenopalatine neuralgia**
   It is questionable whether the so-called sphenopalatine (or Sluder's) neuralgia is a true primary neuralgia. Its features resemble more closely the 'cluster' headaches of vascular origin.
7. **Great auricular neuralgia**
   Very occasionally, tic-like pain is felt in the area of the angle of the mandible and arises in the posterior root ganglia of the second and third cervical nerves.

SECONDARY NEURALGIAS

Pain in the face may arise from any lesion which

irritates its nerve supply at any level. Such lesions are conveniently divided into three groups:

1. **Intracranial,** e.g. neoplasms, granulomata, reticuloses, aneurysms, syringobulbia, thrombosis of posterior inferior cerebellar artery.
2. **Lesions of the bony skull base**, e.g. fractures, carcinomatosis, osteitis deformans, cranial hyperostosis, petrous osteitis.
3. **Extracranial**. These are too numerous to detail but there are two which worthy of mention.

   The first is a *nasopharyngeal carcinoma*, originating in Rosenmuller's fossa, which spreads beneath the mucosa along the base of the skull involving first the maxillary division of the trigeminal nerve (but if unchecked may spread to all divisions) and later the glossopharyngeal and vagus nerves. Its importance lies in its insidious growth and how often it goes undetected in its early stages, when it is often amenable to cure by radiotherapy. Rarely, however, cure is followed by scar tissue involving the nerve(s) and the resulting neuralgia may necessitate root section for the relief of pain. The second, *Frey's syndrome*, is associated with operations or trauma to the parotid gland, and is characterized by discomfort, sweating and redness of the skin overlying the parotid area occurring during and after eating. It is due to the severed ends of parasympathetic secretomotor fibres growing into the skin; when the patient eats, these fibres are stimulated (as they formerly were for the production of saliva) and they cause vasodilatation and sweating. Spontaneous resolution within 6 months is usual, but a small number of patients require active treatment. This involves tympanic neurectomy which divides the parasympathetic pathway.

OTHER CAUSES OF FACIAL PAIN:

Localised facial pain

Causes of localized pain in the face are legion. Amongst these are:

1. Diseases of paranasal sinuses, especially infection or neoplasm.

Frontal sinus pain may be due to acute infection, chronic sinusitis, intermittent blockage of the ostium by oedematous mucous membrane, blockage of the duct by an osteoma, and, rarely, blockage by tumour.

Acute frontal sinusitis is usually associated with an upper respiratory tract infection and is characterized by frontal pain that often begins after arising from bed. The patient may note tenderness of the walls of the sinus. The frontal recess is often occluded, and no intranasal pus is present. If the sinus is draining, pus will be seen in the anterior half of the middle meatus of the nose. The sinus may be opaque to transillumination. Treatment consists of administration of an oral antibiotic and a topical nasal vasoconstricting drop or spray preparation. When the infection is recalcitrant, a nasal culture is taken from the middle meatus, to guide one in the choice of antibiotic. When an infection fails to respond to treatment and a patient develops severe pain, or when swelling of the forehead or upper eyelid develops, it may be necessary to admit the patient to the hospital for intensive treatment with intravenous antibiotics and, probably, for trephination of the sinus. Nowadays, Functional Endoscopic Sinus Surgery should play a role in creating drainage for the sinus before resorting to trephination.

Frontal osteomas may be a cause of facial pain. They are diagnosed by x-ray studies and can be removed surgically.

Maxillary sinus infection. Patients develop infections of the maxillary sinus following upper respiratory tract infections and exacerbations of chronic maxillary sinusitis, from acute occlusion of the maxillary sinus ostium during allergic attacks, and sometimes as the result of apical or periapical maxillary tooth infection, usually the first molar, extending into the maxillary sinus. The patient has pain and tenderness of the cheek. The patient may have drainage from the nose, and if this drainage is foul smelling and foul tasting, the focus of infection will be a maxillary tooth. Treatment consists of antibiotics given according to the results of aerobic and anaerobic pus culture. If the infection fails to respond after suitable trials of antibiotics, sinus irrigation performed through the inferior meatus of the nose or through the natural ostium of the sinus may be helpful. If one suspects the focus of infection to be a tooth, the

appropriate dental x-ray studies should be obtained, and the patient should be referred to a dentist.

Maxillary sinus tumours. Malignant and sometimes benign tumours within the maxillary sinus may produce cheek pain. Clues to the presence of a tumour, rather than an infection, include erosion of one or more of the walls of the sinus as seen on radiographs, swelling or expansion of the superior maxillary alveolus, and numbness and hyperaethesia of the skin of the cheek and upper lip. These patients may have intranasal findings, possibly including blood-stained mucoid drainage. The diagnosis is made by biopsy of the tumour intranasally. Treatment is based on the nature of the tumour.

Sphenoid sinus disorders

On occasion, disease processes including infections, mucocoele, and tumours of the sphenoid sinus produce pain in the side of the face. The diagnosis is made on the basis of the patient's medical history, examination, and radiographs. Treatment is according to the disease process.

2. Diseases of teeth and jaws are the commonest cause of facial pain. Examples are caries, periapical infection, lateral periodontal abscesses, pulp stones, oesteomyelitis, fractures, rarefying osteitis following tooth extraction, and neoplasms. Pain due to a diseased tooth may be referred to a normal tooth but the affected tooth can be indicated by the pain being aggravated by temperature extremes or percussion and eased by local anaesthesia.

3. Diseases of the ear and referred otalgia: pain in the ear may originate from within the ear itself, or may be referred in nature. If a patient complains of otalgia do not feel obliged to see an abnormal or 'slightly erythematous' ear drum. It may be absolutely normal. The sensation to the ear is from 3 cranial nerves and 2 cervical nerves. Some of the causes of otalgia can be listed as below:

OTOLOGIC
- Otitis externa
  Otitis media
  Mastoiditis
  Tumours of external or middle ear

REFERRED OTALGIA
- Vc: Impacted wisdom teeth
  Molar caries
  Temporomandibular joint dysfunction
  Adenoidectomy
- IX: Tonsillitis
  Tonsillectomy
  Glossopharyngeal neuralgia
  Tongue base tumour
  Eagle's syndrome
- X: Laryngitis
  Laryngeal cancer
- C2, 3: Cervical disc lesion
  Cervical arthritis

The commonest causes are from the teeth and after tonsillectomy, but note that rarely it can be due to a tumour. ***Eagle's syndrome*** is characterised by a sore throat, pain on swallowing, and referred otalgia[4]. It is caused by the compression and irritation of the surface of the carotid artery by an elongated styloid process or a calcified stylohyoid ligament. The diagnosis is made by palpation and manual examination of the tonsil area as well as with radiological evidence of the elongated styloid. Resection of the styloid process may bring these patients relief.

4. Diseases of the eyes:

(1) ***Infections and inflammatory disordes*** of the adnexa and orbital structures include such conditions as blepharitis, conjunctivitis, dacryocystitis, iritis etc. If evidence of such a disorder is present, the patient should be referred to an ophthalmologist.

(2) ***Orbital cellulitis and orbital abscess*** usually are complications of frontal or ethmoid sinusitis. The patient has orbital pain, possible swelling of the upper eyelid, and conjunctivitis with limitation of eye movement, in addition to signs of serious illness. The patient requires hospitalization for intensive antibiotic treatment and, possibly, sinus and orbital drainage. The diagnosis can be made accurately by computed tomograms of the orbit.

(3) ***Orbital tumours*** may produce pain as well as displacement of the eye and interference with vision. Such tumours may be primary in the orbit (for example, melanoma, rhabdomyosarcoma) or may represent extension of a malignant tumour of a paranasal sinus, or represent metastases from remote sites. These patients require extensive medical and radiologic evaluation including CT

scanning of the orbit and sinuses. Treatment depends on the nature of the tumour.

(4) ***Mucocoele and pyocoele*** are benign masses that originate in the frontal, ethmoid or, rarely, the sphenoid sinuses with extension into the orbit. When infected (pyocoele), they produce pain as well as swelling, displacement of the globe, and limitation of motion of the globe. After evaluation, these patients require surgical removal of the mass and an appropriate sinus operation.

(5) ***Glaucoma*** must always be considered as a cause of facial pain in patients over 40 years of age. Intermittent narrow-angled glaucoma is an even likelier dianosis when vision is altered without restriction of extra-ocular muscles. Opthalmologic referral is indicated.

5. Diseases of the temporomandibular joint commonly gives rise to preauricular and temporal pain, in, for example, rheumatoid arthritis. But in ***Costen's syndrome*** pain arises in the absence of any evidence of gross pathology in the joint. This syndrome normally occurs in edentulous patients, over the age of 40, in which the joint is overstrained due to malocclusion, loss of molar support with over-closure of the jaws or to non-equilibrated dentures. Amongst the factors said to be responsible are compression of the chorda tympani and auriculotemporal nerves, and pressure on the eustachian tube. It has been suggested that distortion of the thin tympanic plate, which ends in a free edge at the petrotympanic fissure, may compress the fifth, seventh, ninth and tenth cranial nerves with pain of wide distribution including the tongue and side of the nose with local tenderness. Costen's syndrome is commoner in women than in men and may by aggravated by associated emotional disturbance. Its onset may be sudden and brought on by a wide yawn, vigorous chewing, or a blow on the jaw. Usually, however, its onset is gradual and pain is aggravated by jaw movements in eating when occasionally a clicking sound may be detected over the joint. There is often tenderness on pressure over the joint and the mandible tends to deviate to the painful side on opening. Although temporary relief may be obtained by infiltration of the joint with a local anaesthetic or corticosteroids, early referral to a prothodontist is necessary. Excision of the mandibular condyle has been recommended but correction of dental occlusion is usually satisfactory.

6. Fibromyalgia. This is applied to patients with muscular head and neck pain. Previous terms used to describe this syndrome include pain-dysfunction syndrome, soft tissue rheumatism, fibrositis, myofibrositis, and fibromyositis[5]. In most instances, this disorder is a result of chronic anxiety, and these patients demonstrate tenderness of the muscles including the temporal, masseter, and the pterygoid muscles. Treatment consists of psychotherapy by the primary-care physician, possibly supplemented by the administration of muscle-relaxing drugs. Patients with bruxism may require the fitting of a dental retainer to be worn between the teeth.

7. Diseases of the salivary glands

(1) ***Calculi*** in the duct of the parotid gland are not as common as calculi in the submandibular gland. These ductal calculi obstruct the duct, distend the gland, and cause pain.

(2) ***Infections*** of the parotid gland occur most commonly in children with mumps and in debilitated adults, who are frequently in a post-operative and dehydrated state and who develop staphylococcal parotitis. In this condition, the gland is distended and tender, and the overlying skin is red.

(3) ***Sialectasia*** causes recurrent episodes of pain, pressure, and swelling of the parotid gland. In this condition, the ductal system of the parotid is ectatic, and a functional obstruction of the ductal system occurs, with periods of swelling caused by stagnation of parotid secretions.

(4) ***Tumours*** of the parotid gland are usually not painful. When a patient has a painful, non-inflammatory swelling of the parotid gland and the physician suspects a tumour, the growth may well be malignant, especially if the patient also has weakness of the facial muscles indicating tumour involvement of the facial nerve.

8. Post-traumatic pain

Some patients may continue to have pain in previously traumatised or fractured areas, for example, the nose, maxillary or mandibular regions even after the fracture or injury has healed. These individuals should be treated with mild analgesics and reassurance that the pain will eventually

disappear. In patients expecting financial compensation, this pain can be expected to last until the claim has been settled.

FACIAL PAIN OF VASCULAR ORIGIN

There are two conditions to which special attention should be drawn:

1. **Cluster headaches (migrainous neuralgias).** These appear to be a form of migraine. Attacks occur in clusters every 12 to 18 months and consist of very severe pain around an eye occurring often at the same time every night for 1-3 weeks. The eye becomes injected and watery and the nostril of the same side blocked up. It often responds to ergotamine.
2. **Temporal arteritis (giant cell arteritis).** It is a local expression of a connective tissue disease. The pain is felt in the temples or over the entire scalp and the affected artery is usually dilated and tender. The patient usually feels ill and the ESR is greatly raised. Biopsy is performed if it can be done rapidly and the patient given steroids without delay lest blindness supervenes.

ATYPICAL FACIAL PAIN

This refers to a syndrome with episodes of constant ache in the jaw and cheek lasting several hours occurring usually in young to middle-aged women who often are depressed. It is often bilateral. Treatment is difficult but patients may respond to antidepressants or antihistamines.

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FACIAL PAIN OF DENTAL AND ORAL ORIGIN

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Facial pain in dental and/or oral conditions may originate from the teeth and their supporting tissues, the oral mucosa and the jaw bones.

TEETH AND SUPPORTING TISSUES

The commonest causes of pain of the jaw and face are dental pulpal pain and pain from the periodontal ligament of the teeth.

Pulpal pain

Within the core of each tooth is the pulp which contains highly sensitive neurovascular structures. Surrounding the pulp is a hard layer of dentine which consists of tiny tubules within which are nerves endings which are extensions of the pulp. Protecting the dentine and its sensitive nerve endings are the enamal cap on the crown of the tooth and cementum on the root of the tooth. Pulpal pain can therefore be evoked if the nerve endings within the dentinal tubules or the pulp itself are exposed to stimuli. Such stimuli include hot or cold food or drinks. Usual causes of pain are deep caries, large and unprotected restorations, exposed dentine or cementum and fracture of the tooth (Figure 1).

Periodontal pain

The periodontal ligament serves to anchor the tooth to its bony socket. Pulpal infection can sometimes spread to the periodontal ligament at the apical region of the root resulting in apical periodontitis (Figure 2). The patient then experiences pain on chewing food.

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Another inflammatory condition of the periodontal ligament can also give rise to pain especially during an acute exacerbation. This is the lateral periodontal abscess. The aetiology of this condition is that of a preexisting periodontal defect which is usually caused by a lack of care of the supporting tissues of the dentition.

Acute pericoronitis

This condition is caused by an acute infection of the gum surrounding a partially erupted wisdom tooth (Figure 3). The patients are usually in their late teens or early twenties and they present with pain and swelling on the affected side of the face. There may be some limitation in mouth opening and when the site of complaint is inspected, a partially erupted tooth is seen with inflammation of the soft tissue around the tooth. Some patients may complain of fever and regional lymphanopathy. Treatment consists of controlling the infection and subsequent removal of the impacted tooth.

ORAL MUCOSA

The common oral mucosal lesions that cause pain are herpes simplex, recurrent aphthous ulcers and erosive lichen planus (Figure 4). These lesions are however easily detected and diagnosed. Herpes zoster and oral malignancies may pose some difficulties and these are therefore discussed in more detail.

Herpes zoster

Herpes zoster of the face is caused by the virus varicella zoster infecting the trigeminal nerve. The patient first complains of fever and then pain and sensitivity of the skin and oral mucosa corresponding to the nerve distribution. At this stage the patient often mistakes the pain to be toothache and may therefore seek dental treatment. The pain and sensitivity is then followed by

formation of vesicles which often break down forming crust. The late complication of this condition may be post-herpetic neuralgia of the trigeminal nerve.

Oral mucosal malignancies

Oral malignancies like other malignancies of the body usually do not present in the form of pain in the early onset. Pain is usually associated with late stage of the tumour or when the tumour is secondarily infected. While the tumour is easily detected in the more accessible part of the oral cavity it is however important that attention be paid in carrying out examination of the more inaccessible part of the oral cavity like the posterior part of the tongue, the pharyngeal area and the posterior part of the oral cavity. An oral mucosal malignancy may present as an indurated lesion or ulcers that do not heal (Figure 5).

JAW BONES

The benign tumours of the jaw bones are generally painless even in the very advanced stage when the tumours have assumed very large proportions. In some occasions when the tumour has broken through the oral mucosa and is secondarily infected the patient may then complain of pain. Malignant tumours of the jaw bone on the other hand, be they primary or secondary, may present with severe pain or parasthesia in the very early stage of the disease.

Osteo-sarcoma

Osteo-sarcoma of the jaw bone is characterised by pain or numbness of the jaw. The patient, who is usually a young adult, notices a hard swelling of the jaw which enlarges very rapidly. In the initial stage, the oral mucosa appears healthy but radiographs will show evidence of rapid bone destruction which progresses and penetrates the cortical layer, the periostium and into the surrounding soft tissue causing the hard swelling. The condition deteriorates rapidly with massive destruction of tissues locally as well as distant metastasis. Treatment consists of very radical resection of the tumour and chemotherapy but the prognosis remains very poor.

Secondary malignancies

Patients presenting with secondary malignancies in the jaw are usually in the older age group whose primary malignacies are usually in the lung, breast and prostate. The usual presentations are swelling and pain or numbness of the jaw and face. Diagnosis is usually confirmed by a biopsy and diagnostic imaging of the jaw and other parts of the body.

Jaw fractures

Jaw fractures are usually the result of an accident or an assault. They may be associated with other injuries or may present as an isolated injury with no external soft tissue injuries to suggest a fracture. The fractures per se usually do not present with severe jaw or facial pain. However, when the bony edges are rubbed against each other as in jaw movement or during examination then the patient will complain of pain (Figure 6).

Osteomyelitis

Osteomyelitis of the jaw bone is usually the result of a dental infection and in some occasions the complication of deep x-ray therapy for the treatment of tumour in the head and neck region. The patient complains of dull to severe pain depending on the severity of the bone infection. The pain is deep-seated and throbbing. The overlying gum or facial soft tissue is swollen and red and there may be regional lymphadenitis.

The management is drainage and removal of necrotic tissue followed by a suitable antibiotic.

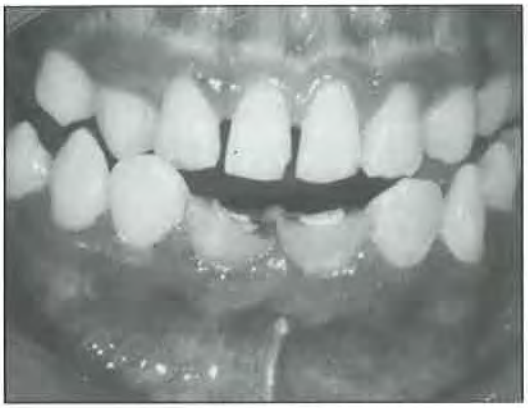

Fig 1: A decayed tooth causing pulpal pain.

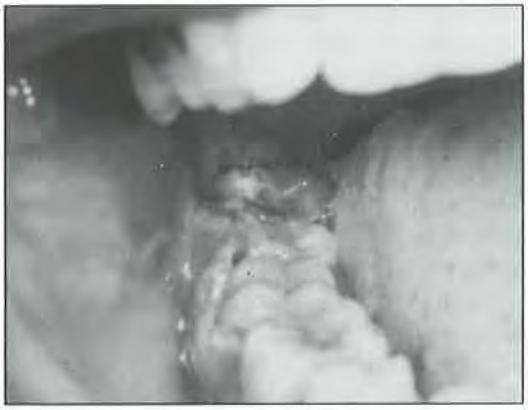

Fig 3: Inflammation of mucosa around a partially erupted mandibular third molar.

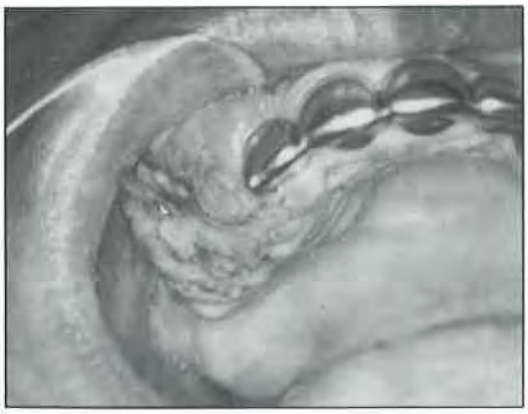

Fig 5: Squamous cell carcinoma of the left palate.

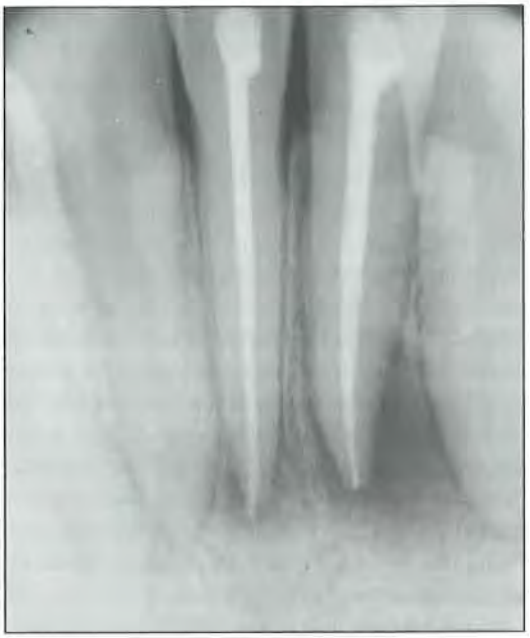

Fig 2: Apical infection of the lower incisors.

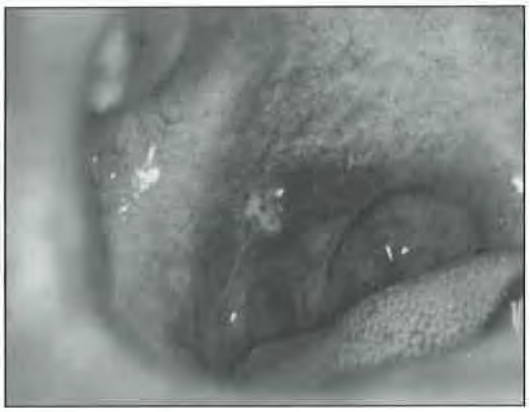

Fig 4: Ulcer on the soft palate and inflammation of the surrounding tissues.

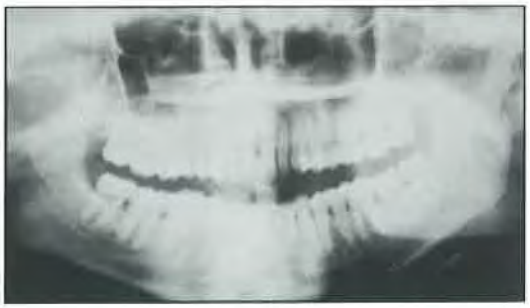

Fig 6. Fractured and displaced left condyle of the mandible.

Acknowledgment:
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FOETAL MACROSOMIA IN A SINGAPORE POPULATION: AN OBSTETRIC VIEWPOINT

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ABSTRACT

A retrospective study on the obstetric features on foetal macrosomia was performed for patients delivering a baby weighing 4000gm or more. The prevalence of foetal macrosomia was 2.8%. The maternal risk factors for foetal macrosomia were multiparity, maternal weight more than 71 kg, previous history of macrosomic babies and gestational diabetes. Dystocia in labour was not common in this study. Operative delivery rates were higher. Shoulder dystocia and primary postpartum haemorrhage were real risks. With appropriate treatment, majority of cases had favourable outcome as was reflected in the Apgar score.

Key words:

Foetal, Macrosomia

INTRODUCTION

Macrosomic babes have always posed problems for the obstetricians[1,2]. A macrosomic baby lends itself to complications, particularly during labour[3]. There is an increase incidence of dystocia in mothers of large infants including failure to progress and cephalopelvic disproportion. Shoulder dystocia is a real hazard. Operative delivery rates are high. Maternal morbidity, perinatal morbidity and mortality are significantly increased. The perinatal period is tumultuous for the macrosomic baby, especially after a traumatic vaginal delivery. Metabolic disturbances are difficult to treat. The problems associated with foetal macrosomia can be largely overcome if macrosomia is detected early, its problems anticipated during labour and prompt treatment instituted.

A baby can be considered macrosomic when the birthweight is greater than 4000gm. Most studies have based their analyses on this figure, although varying birthweights have been used.

The prevalence of foetal macrosomia varies in different populations. In Canada, Body[4] reported an overall prevalence of 10.0% of all deliveries. Abudu and Awonuga[5] reported a prevalence of 4.9% in 6376 singleton births in Lagos Nigeria. Chuah[6] reported a prevalence of 3.3% in a retrospective review of all deliveries in the University Hospital, Kuala Lumpur, Malaysia.

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The aims of this study are to study (i) the prevalence of foetal macrosomia in a sample size Singapore population, (ii) the characteristics of mothers who delivered macrosomic babies in order to develop a predictive risk assessment system and (iii) the mode and outcome of delivery, in particular the incidence of birth asphyxia and shoulder dystocia.

MATERIALS AND METHODS

The birthweights of all deliveries in Kandang Kerbau Hospital, Singapore are recorded in the computers. The case records of all the women who had delivered a baby weighing 4000gm or more in the Department of Reproductive Medicine in this hospital for a six month period between April 1990 and October 1990 were studied. There were 2907 deliveries during this period. There were 75 mothers who delivered a macrosomic baby.

These mothers were delivered by qualified obstetricians, medical officers (trainee) and midwives. The decisions for operative vaginal deliveries (vacuum and forceps) and Caesarean section were made by obstetricians. All operative vaginal deliveries and Caesarean section were carried out by obstetricians and medical officers (trainee).

Episiotomies were routinely performed for all vaginal deliveries. Shoulder dystocia, if present, was documented in the medical records. The Apgar scores of the babies were routinely assessed after delivery.

RESULTS

A. PATIENT CHARACTERISTICS

1 **Overall Prevalence**

The total number of deliveries in the study period of April to October 1990 was 2907. There were 75 newborns weighing 4000g or more. The prevalence of macrosomic babies was 2.8% of the total number of deliveries.

2 **Ethnic Distribution**

The distribution of macrosomic babies delivered to the different ethnic groups is shown in Table 1. Among the different ethnic groups, the Indians had the lowest percentage of macrosomic babies (1.9%) as compared to the Chinese (2.8%) and Malays (2.2%).

TABLE 1:
PREVALENCE OF MACROSOMIC BABIES AMONG THE ETHNIC GROUPS

Race	Total No of Deliveries	No of Macrosomic Babies	Percentage
Chinese	1902	53	2.8
Malay	725	16	2.2
Indian	254	5	1.9
Others	24	1	3.8
Total	2907	75	2.8

3 **Age Distribution**

The distribution of macrosomic babies among the different age groups of women is shown in Table 2. A high prevalence of macrosomic babies was seen among the 26-30 years age group (42.7%) and 31-34 years age group (26.6%).

TABLE 2:
THE PREVALENCE OF MACROSOMIC BABIES IN DIFFERENT AGE GROUPS OF WOMEN

Age (in years)	Number	Percentage
Less than or equal to 20	2	2.7
21 - 25	9	12.0
26 - 30	32	42.7
31 - 34	20	26.6
35 and above	12	16.6
Total	75	100

4 **Gravidity**

The influence of gravidity on foetal macrosomia is shown in Table 3. A high percentage of these babies were born to mothers in their third pregnancies. It was also found that a lower percentage of macrosomic babies was born to mothers in their fourth (9.3%) and fifth (8.0%) pregnancies.

TABLE 3:
GRAVIDITY AND MACROSOMIC BABIES

Parity	Number	Percentage
1	15	20.1
2	21	2.0
3	26	34.7
4	7	9.3
5 or more	6	8.0
Total	75	100

5 **Weight At Term**

Not all patients had their weight documented at term. There were 68 mothers who had their weight measured at term. The weights were taken from the antenatal notes at thirty-seven completed weeks of gestation. Table 4 shows the maternal weight at term in those who delivered a macrosomic baby. More macrosomic babies were delivered by mothers weighing 71kg or more. Fourty-three (63.2%) of 68 mothers who delivered a macrosomic baby weighed 71kg or more.

TABLE 4:
RELATION BETWEEN MATERNAL WEIGHT AT TERM AND MACROSOMIC BABIES

Weight at 37 weeks (kilogrammes)	Number	Percentage
<60	5	7.4
61 - 70	20	29.4
71 - 80	25	36.8
81 - 90	12	17.6
91 and above	6	8.8
Total	68	100

6 **Previous History of Macrosomic Babies**

Fourteen patients (18.7%) had previously delivered a macrosomic baby.

7 **Presence of Gestational Diabetes Mellitus**

Gestational diabetes is a well known risk factor for foetal macrosomia. It was found that only 13 (7.3%) of 75 mothers had gestational diabetes.

8 **Gestation at Time of Delivery**

Table 5 shows the gestational age at delivery of a macrosomic baby. Two of the mothers did not have the period of gestation documented. The majority of macrosomic babies (91.8%) were delivered at term between 38 to 42 weeks. However, the remaining babies were delivered pre-term (5.5%) and post-term (2.7%).

TABLE 5:
GESTATION AT DELIVERY WITH MACROSOMIC BABIES

No of completed weeks	Number	Percentage
Less than 38	4	5.5
38 to 42	67	91.8
More than 42	2	2.7
Total	73	100

B. CHARACTERISTICS OF LABOUR

1 **Onset of Labour**

Seventy-one patients underwent a trial of labour. Eleven patients (15.5%) had the labour induced. Sixty patients (84.5%) had spontaneous onset of labour. Four patients had elective Caesarean section.

2 **Length of First Stage of Labour**

The length of the first stage of labour was taken from the time of diagnosis of established labour to the time of full cervical dilatation. Only 64 patients had full documentation of the first stage of labour. The relation of the first stage of labour and macrosomia is shown in Table 6. It appeared that the duration of the first stage of labour was not prolonged. There were 26 patients (40.6%) who had a duration of less than 5 hours and 21 patients (32.8%) who had a duration of between 5 to 10 hours of the first of labour.

TABLE 6:
RELATION OF DURATION OF FIRST STAGE OF LABOUR AND MACROSOMIC FOETUS

Length (in hours)	Number	Percentage
Less than 5	26	40.6
5 to 10	21	32.8
More than 10	17	26.6
Total	64	100

3 **Length of Second Stage of Labour**

Table 7 shows the relation between the duration of the second stage of labour and macrosomic foetus. Fifty-two patients had accurate documentation of this stage of labour. The majority (82.7%) had a second stage lasting less than 30 minutes. Only 3.8% had a prolonged second stage lasting 60 minutes or more.

TABLE 7:
RELATION BETWEEN THE DURATION OF THE SECOND STAGE OF LABOUR AND MACROSOMIC FOETUS

Length (minutes)	Number	Percentage
Less than 30	43	82.7
30 to 60	7	13.5
More than 60	2	3.8
Total	52	100

4 **Mode of Delivery**

The mode of delivery of these macrosomic babies is shown in Table 8. Macrosomic babies were delivered by normal vaginal delivery (61.3%), operative vaginal delivery (10.7%) and Caesarean section (28.0%).

TABLE 8:
MODE OF DELIVERY

Mode	Number	Percentage
Normal vaginal	46	61.3
Forceps/Ventouse	8	10.7
Caesarean Section	21	28.0
Total	75	100

5 **Indication for Caesarean Section**

Table 9 shows the indication for Caesarean section when a macrosomic baby was delivered by this mode. The majority of the Caesarean section was for cephalo-pelvic disproportion (38.1%). Of the 21 Caesarean sections, 17 were performed as emergency operations and 4 were elective operations.

TABLE 9:
INDICATIONS FOR CAESAREAN SECTION

Indication	Number	Percentage
Cephalo-pelvic disproportion	8	38.1
Poor progress	2	9.6
Foetal Distress	4	19.0
Others e.g. Previous Caesarean section, Breech.	7	33.3
Total	21	100

6 **Colour of Liquor**

Meconium staining of liquor was taken as suggestive evidence of foetal distress. Table 10 shows the frequency of meconium staining of the liquor during the delivery of these macrosomic babies. A high proportion (34.7%) of the babies had meconium staining during labour.

TABLE 10:
COLOUR OF AMNIOTIC FLUID

Colour	Number	Percentage
Clear	49	65.3
Mecomium Stained	26	34.7
Total	75	100

TABLE 11:
APGAR SCORE AT 1 MIN

Score	Number	Percentage
3 or less	2	2.7
4 to 6	2	2.7
7 to 10	71	94.6
Total	75	100

7 **Sex of Baby**
Of the 75 babies delivered, 40 babies (53.5%) were male, and the other 35 (46.7%) were female.

8 **Apgar Scores**
The scores were assessed at one minute and five minutes after delivery of these babies. The scores were taken as indication of the presence or absence of birth asphyxia at the time of delivery. A score of 3 or less was taken as indicative of severe asphyxia and 4 to 6 as moderate asphyxia. The Apgar scores are shown in Table 11 and 12. The majority of macrosomic babies did not show signs of birth asphyxia. Only 1.4% had severe asphyxia at 5 minutes of delivery and 98.6% of the infants did not show signs of birth asphyxia.

9 **Presence of Shoulder Dystocia at Time of Delivery**
Shoulder dystocia is a known complication in the delivery of macrosomic babies. Eight (12.5%) of 75 macrosomic babies had shoulder dystocia. The antepartum and intrapartum characteristics of these pregnancies are shown in Table 13. There was poor association between gestational diabetes and shoulder dystocia in this study. The length of the second stage of labour was longer (> 50 mins) in those babies with birth weights greater than 4200 gm. Only 2 babies were delivered by forceps and one of these had poor Apgar score (1 at 1 minutes, 4 at 5 minutes) and its birthweight was 4520 gm. The majority of the babies had good Apgar score at 5 minutes despite shoulder dystocia.

TABLE 12:
APGAR SCORE AT 5 MIN

Score	Number	Percentage
3 or less	1	1.4
4 to 6	0	0
7 to 10	74	98.6
Total	75	100

10 **Presence of Post-partum Haemorrhage**
Eleven (14.7%) of 75 mothers had primary post-partum haemorrhage. This was defined as loss of 500ml or more of blood during the first twenty-four hours after delivery.

DISCUSSION

The objectives in this retrospective study of macrosomic infants were threefold. The first was to ascertain its prevalence in the study population. The others were to determine the characteristics of mothers of such babies and the mode and outcome of their deliveries. In addition, special attention was given to the problem of shoulder dystocia.

The prevalence of foetal macrosomia amongst the 2907 deliveries in the study period was 2.8%. This is indeed less that the 10% in a Canadian population, as recorded by Boyd[4], and 4.9% in a Nigerian population[5]. However, this figure is comparable with the 2% reported in study in Malaysia[6]. This difference could be explained by the smaller build of our population as compared with the Caucasians.

TABLE 13:
SHOULDER DYSTOCIA

Serial Number	Birthweight	Presence of Maternal Diabetes	Length of Second Stage (Minutes)	Mode of Delivery	Apgar Scores at 1 & 5 Minutes
1	4210	Yes	60	Spontaneous	8,9
2	4050	Yes	35	Forceps	2,7
3	4120	No	6	Spontaneous	8,9
4	4300	No	10	Spontaneous	9,9
5	4520	No	55	Forceps	1,4
6	4075	No	30	Spontaneous	7,9
7	4120	No	10	Spontaneous	9,9
8	4010	No	10	Spontaneous	9,9

Macrosomia can be attributed to a few maternal factors[7]. It is a reflection of the economic affluence of the community which brings with it better nutrition and antenatal health care thus ensuring optimum fetal growth. Other factors include diabetes mellitus, genetic predisposition, prolonged pregnancy and multiparity. Racial differences were generally not significantly different, although the Indians were noted to have the least percentage of macrosomic babies. It is a known fact that the Indians in Singapore have the highest incidence of low birthweight babies among the ethnic groups. It is indicative of the lower economic and nutritional status of this ethnic group.

Most of the mothers who delivered macrosomic babies were between 26-30 year old. This is probably insignificant because most mothers who delivered in the hospital are within this age group.

There is an association between parity and macrosomic babies in this study. Twenty percent of the mothers were primipara and the rest were multipara. Eight percent of macrosomic babies were delivered by mothers who had five or more deliveries previously. This demonstrates that macrosomia is more common among the multipara.

The maternal weight may have a direct influence on the weight of babies[8,9]. In this study 63.2% of macrosomic babies were delivered by mothers who were heavier than 70kg at term. Hence, there is preponderance of mothers with high maternal weight to deliver macrosomic babies. The Body Mass Index of the mother may have been a more refined yardstick in analysing this relationship to macrosomic babies. In addition, it was found that 18.7% of the multiparas had delivered macrosomic babies previously. This is an added risk factor.

Only 17.3% of mothers who delivered a macrosomic baby in this study had gestational diabetes. While diabetes in mothers has been shown to be a predisposing factor in macrosomic infants, random studies of populations of such babies did not reveal a large number of diabetics[10].

Most of the macrosomic babies were delivered at term. Post-term pregnancies are known to be at risk of producing macrosomic babies. However, in this study, only 2.7% of macrosomic babies were post-term. Surprisingly, 5.5% of these deliveries had occurred pre-term. This is due to macrosomic babies who were delivered by mothers with uncontrolled gestational diabetes.

It is expected that mothers with macrosomic foetuses may have a higher incidence of prolonged labours. In this study, only 26.6% (17 patients) of the mothers had the first stage of labour with a duration longer than 10 hours. Of the 52 patients

who delivered per vaginam 82.7% (43% patients) had a second stage that lasted less than 30 minutes. Therefore, the incidence of prolonged labour was not as high as expected in this population. Prolonged labour among macrosomic babies is also an inconsistent finding in other studies.

Operative deliveries are higher for macrosomic babies. In this study, 38.7% of the macrosomic babies had operative delivery, 10.7% were delivered with the forceps or ventouse, and 28% were delivered by Caesarean section. Other studies have also reported Caesarean section rates of 28.8%[3], 27.6%[5] and 30.2%[6].

In this study, Caesarean section were performed primarily for cephalo-pelvic disproportion (38.1%). This is not surprising as the powers and passage cannot accommodate so large a passenger. Caesarean sections were performed for foetal distress in 19% of these mothers.

A significant finding in this study was the high incidence of meconium stained liquor (34.7%). The presence of meconium in the liquor could be a reflection of an adverse reaction of these foetuses in labour. It is not necessarily equated with foetal distress, although it will alert the obstetricians to such a predisposition. Li et al[11] reported an incidence of 14.9% meconium staining amongst macrosomic infants.

Several authors have reported birth asphyxia rates of 6% to 7% in macrosomic infants. In this study, the Apgar score of 98.6% of babies was seven and above. Only 1.4% were severely asphyxiated at five minutes. Such favourable results could be attributed to the experience and skill of the medical personnel at the time of delivery.

Shoulder dystocia was found to be a frequent complication[12]. In this study, 2.5% of macrosomic babies had shoulder dystocia. A standard protocol in emergency handling of shoulder dystocia in this hospital has enabled immediate treatment for this condition. A favourable perinatal outcome is reported in this study.

Forceps were applied in two mothers whose second stage lasted 35 and 55 minutes respectively. These two babies had the lowest Apgar scores. This demonstrates that the combination of a big baby, the use of forceps and the presence of shoulder dystocia increases the risks of birth asphyxia.

Primary post-partum haemorrhage was seen in 14.7% of the mothers. This is not surprising as foetal macrosomia causes uterine overdistension. Overdistention of the uterus is a risk factor for post-partum haemorrhage. Most of this complication was prevented by the routine use of parenteral oxytocics at time of delivery, with other simple measures taken to reduce the bleeding, such as rubbing up the uterus, and controlled cord traction delivery of the placenta.

There was no significant difference in the distribution of the sexes amongst the big babies.

In conclusion, this study has highlighted certain common risk factors for foetal macrosomia. These include multiparity, excessive weight at term, a previous history of macrosomic babies and gestational diabetes. Although at risk of dystocia, most macrosomic babies in this study did not shown any abnormal features in the course of labour. Almost 40% of the patients required operative or instrumental delivery, in particular Caesarean section. Cephalo-pelvic disproportion is the main indication for Caesarean section. One striking feature is the high incidence (30%) of meconium stained liquor among these macrosomic foetuses and this must be countered by closer foetal surveillance during labour. Asphyxia is a reality in macrosomic babies, particularly if shoulder dystocia occurs. Primary post-partum haemorrhage is common and the third stage of labour must be actively managed. Finally, the early recognition of macrosomic foetus is important. The review for risk factors for foetal macrosomia in the mother, and realising that they predispose to special problems in labour need not be stressed further.

ACKNOWLEDGMENT

We like to thank Ms Kala Ramaiya for the many hours she had spent in typing this manuscript.

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HELICOBACTER PYLORI AND PEPTIC ULCER DISEASE

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INTRODUCTION

Peptic ulcer disease has steadily declined in incidence in the past 50 years or so, but the number of publications relating to its natural history, aetiology and management continues to rise. Interest has been fuelled by advances in endoscopic techniques for examination of the upper gastrointestinal tract, and the development of specific and powerful drugs. However, no clear means of altering the natural history of the disease had emerged, and the frequency of major complications remained unaltered until recently.

The situation has been changed by the discovery of the strong association between the colonisation of the gastric mucosa by *Helicobacter pylori* and peptic ulcer disease, coupled with evidence that eradication of *H.pylori* reduces the relapse rate after ulcer healing. The avoidance of relapse and to some extent prevention of complications is increasingly likely.

EPIDEMIOLOGY

H.pylori is a spiral shaped, gram-negative motile bacterium which in humans is found only in association with gastric epithelial cells. Originally isolated in Perth in 1982, it has subsequently been found in human stomachs world-wide. In the developed world, the infection is more common with increasing age — approximately 20% of 20-year-olds, and 60% of 60-year-olds harbour *H.pylori*. In many parts of the developing world, most young adults are infected with *H.pylori*. The organism is uniquely suited to its niche in the stomach and can be difficult to eradicate from this protected habitat. The mode of transmission of *H.pylori* is not clear, but would appear to be from person to person.

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DISEASE ASSOCIATIONS

Gastric mucosal infection with *H.pylori* results in chronic antral (type B) gastritis, an inflammatory response seen microscopically which has a normal endoscopic appearance in most patients. Gastritis is generally not associated with acute clinical symptoms, although it poses a risk of developing ulcer disease. *H.pylori* incites a local inflammatory response an a systemic immune response; pharmacological clearance of the organism leads to healing of gastritis.

H.pylori infection of gastric epithelium is found in 95% of patients with duodenal ulcers (DU) and 70% with gastric ulcers (GU). Gastric metaplasia in the duodenal mucosa is found in 60-100% of DU patients with accompanying *H.pylori* infection.

Perhaps the strongest data supporting a causal role for *H.pylori* are in the relapse of DUs after therapy; if *H.pylori* persists, the majority of uclers will recur. DU relapse is markedly reduced if *H.pylori* is successfully eradicated. While half of non-ulcer dyspepsia patients (those with ulcer symptoms but no ulcer crater) are infected with *H.pylori*, an aetiological role for *H.pylori* in non-ulce dyspepsia is unproven.

The role of *H.pylori* in the development of gastric carcinoma is unclear. Although there is a high prevalence of *H.pylori* infection in patients that have cancer and premalignant lesions of the stomach it must be emphasized the *H.pylori* is present in over 50% of normal stomachs. So there must be a modifying factor.

DIAGNOSIS

A number of methods are available to diagnose *H.pylori* infection. Endoscopic biopsies may reveal the presence of *H.pylori* on histological examination and provide material for culturing the organism, which remains the gold standard for identification.

H.pylori is the only gastric bacterium which produces a large, extracellular amount of the enzyme urease. This has been used as the basis for the rapid assessment of antral biopsy specimens and the *H.pylori* breath test (HPBT). The HPBT is a non-invasive test in which ^{13}C- or ^{14}C- labelled urea is given orally. If *H.pylori* is present, labelled carbon dioxide resulting from the breakdown of urea by bacterial urease is measured in breath samples with a sensitivity of 95% and specificity of 100%. The development of the HPBT has provided a useful method to assess bacterial eradication therapy.

H.pylori infection results in a humoral immune response and specific antibodies can be measured by various methods, the most sensitive of which is the ELISA. Serological diagnosis of *H.pylori* infection has found its greatest use in epidemiological studies.

ERADICATION OF H.PYLORI

Eradication of *H.pylori* infection in patients with duodenal ulceration is associated with a reduced incidence of ulcer relapse. Use of bismuth compounds alone reduces the one-year relapse rate of peptic ulcer from 80 to 50 percent. This is almost certainly due to the partial eradication of *H.pylori* associated with colloidal bismuth.

Promising early studies indicate that the one-year relapse rate can be decreased further to about 11 percent by completely eradicating *H.pylori* using bismuth plus an antibiotic (metronidazole, amoxycillin or tetracyline). In practice, a combination of two antibiotics its often used to try to prevent emergence of antibiotic resistance in the organism. The most commonly used regime is colloidal bismuth subcitrate 120 mg qds, plus amoxycillin 250 mg tds, plus metronidazole 400 mg tds for 14 days. This is followed by bismuth alone for a further 28 days.

Drug schemes designed to eradicate infection with *H.pylori* are not entirely free of side-effects. Diarrhoea is common, and pseudomembranous colitis has been reported in some patients. Repeated courses of bismuth-containing compounds are not recommended in view of the know systemic absorption of bismuth. Less complex dual regimes using non-bismuth combinations are currently being evaluated world-wide. These have shown early promise and may result in fewer side-effects.

At present, eradication treatment is best reserved for patients with endoscopically proven ulcer and confirmed *H.pylori* infection, who present management difficulties such as frequent ulcer recurrences of complications. Eradication is defined as the absence of *H.pylori* infection one month after therapy has been completed. Breath testing provides an ideal non-invasive method to document infection prior to treatment and to evaluate the success of eradication. However, it is unclear whether patients who fail to eradicate or have recurrent infection should be treated with a second course of antimicrobials or with an alternate therapy such as H_2 receptor antagonists.

CONCLUSION

There have been many changes in our approach to the management of peptic ulcer disease and in our understanding of how it is caused over the years. However, the recent recognition of the important aetiological role played by *H.pylori* is probably the single most important development so far. The goal of treatment for all to alter the natural history of the disease and to prevent complications seems now tantalisingly close to realisation.

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3. Rauws EAJ, Tygat GNJ. Cure of duodenal ulcer associated with Helicobacter pylori. Lancet 1990; (335): 1233-5.
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Multiple Choice Questions

1 Helicobacter pylori

A. in humans is found only in association with gastric epithelial cells
B. does not infect the stomachs of young adults
C. is never found in normal human stomachs
D. is almost invariably found in the gastric epithelium of patients with duodenal ulcer
E. infection is associated with histological chronic antral gastritis

2 The following statement(s) is(are) correct

A. prevalence of *H.pylori* infection increases with age
B. eradication of *H.pylori* appears to decrease the rate of duodenal ulcer relapse
C. presence of *H.pylori* in the gastric mucosa is invariably associated with gastric ulcer
D. stool cultures may reveal the presence of *H.pylori*
E. the *H.pylori* breath test provides a useful non-invasive method to assess bacterial eradication therapy

3 With regards to treatments for *H.pylori* infection

A. no single drug can reliably eradicate the organism
B. bismuth compounds are unsuitable for maintenance therapy
C. H2-receptor antagonists have been proven to be the drugs of choice
D. adverse effects with triple drug therapy are rare
E. for most patients, routine treatment to eradicate *H.pylori* is strongly recommended

Answers:
(1). ADE
(2). ABE
(3). AB

GUIDELINES FOR AUTHORS
THE SINGAPORE FAMILY PHYSICIAN

Authors are invited to submit material for publication in the Singapore Family Physician on the understanding that the work is original and that it has not been submitted or published elsewhere.

The following types of articles may be suitable for publication: case reports, original research work, audits of patient care, protocols for patient or practice management and review articles.

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The whole paper

* Normally the text should not exceed 2000 words and the number of illustrations should not exceed eight.

  Type throughout in upper and lower case, using double spacing, with three centimetre margins all round. Number every page on the upper right hand corner, beginning with the title page as

  1. Make all necessary corrections before submitting the final typescript.
     Headings and subheadings may be used in the text. Indicate the former by capitals, the latter in upper and lower case underlined.

     Arrange the manuscript in this order: (1) title page, (2) summary, (3) text, (4) references (5) tables, and (6) illustrations.

* Send three copies of all elements of the article: summary, text, references, tables and illustrations. The author should retain a personal copy.

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* The title should be short and clear.

* Include on the title page first name, qualifications, present appointments, type and place of practice of each contributor.

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* The summary should describe why the article was written and give the main argument or findings.

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The text should have the following sequence:

* Introduction: State clearly the purpose of the article.

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  Drugs must be referred to generically; all the usual trade names may be included in parentheses. Dosages should be quoted in metric units.

  Laboratory values should be in SI units with traditional unit in parentheses.

  Do not use patient's names, initials or hospital numbers.

* Results: Present results in logical sequence in the text, tables and illustrations.

* Discussions: Emphasise the new and important aspects of the research and the conclusions that follow from them. Indicate the implications of the findings and limitations. Relate the observations to other relevant studies.

Illustrations

* Diagrams, line drawings, photographs or flow charts are valuable but their use will be subject to editorial policy. Transparencies or prints are acceptable for colour reproduction at the authors' expense.

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Further reading

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THE WORLD ORGANIZATION OF FAMILY DOCTORS

14TH WONCA WORLD CONFERENCE

10-14 June 1995

Hong Kong Convention & Exhibition Centre

"FAMILY MEDICINE - MEETING NEW CHALLENGES"

HOSTED BY
The Hong Kong College of General Practitioners

Message from Dr J. T. N. Chung, Chairman, Host Organising Committee

The Hong Kong College of General Practitioners takes great pleasure in hosting the 14th WONCA World Conference, 10-14 June 1995, and on behalf of the College, I extend a warm welcome to family doctors of the world to Hong Kong.

As we move on in the last decade before the 21st Century, we see Family Medicine facing various challenges coming from health care needs, society, advances in technology and the environment. These issues will make up the theme of the 14th WONCA World Conference. A full array of scientific sessions will be devised to be presented by internationally renowned personalities in Family Medicine. A comprehensive Medical Exhibition will accompany the Scientific Programme, displaying the latest in medical products, technology and educational wares.

June in Hong Kong is early summer when it is comfortably warm. Hong Kong being an eclectic mix of the East and West is already a popular holiday destination, and well placed to offer a wide range of pre-and post-Conference tours.

Come and partake in the fellowship of WONCA and make new friends in a World Conference. We look forward to welcoming you all in Hong Kong in 1995.

WONCA ORCHESTRA, HONG KONG JUNE 1995

During recent years, WONCA has proved itself to be more than an organisation of family doctors; it has shown itself to be a meeting point of hidden talents and fine musicians as well. The WONCA Orchestra, composed of doctors, their families and health personnel, was born in 1989 at the 12th WONCA World Conference in Israel. The musical tradition was picked up at the 13th World Conference in Vancouver in 1992.

It is now the turn of the Hong Kong College of General Practitioners to carry on this musical tradition at the 14th World Conference in 1995. The WONCA Orchestra will be reconvened for a performance at the Opening Ceremony and we intend to make this mini concert as memorable as those before.

We are now recruiting and appeal to those who have participated before to play again, and to those who can play an instrument (to a competent level) but have not tried anything like this, to come and join us. The Orchestra will be under the baton of a professional conductor.

Please fill in the reply slip and send it back to us now!

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Please give a brief description of music experience and level of playing _____

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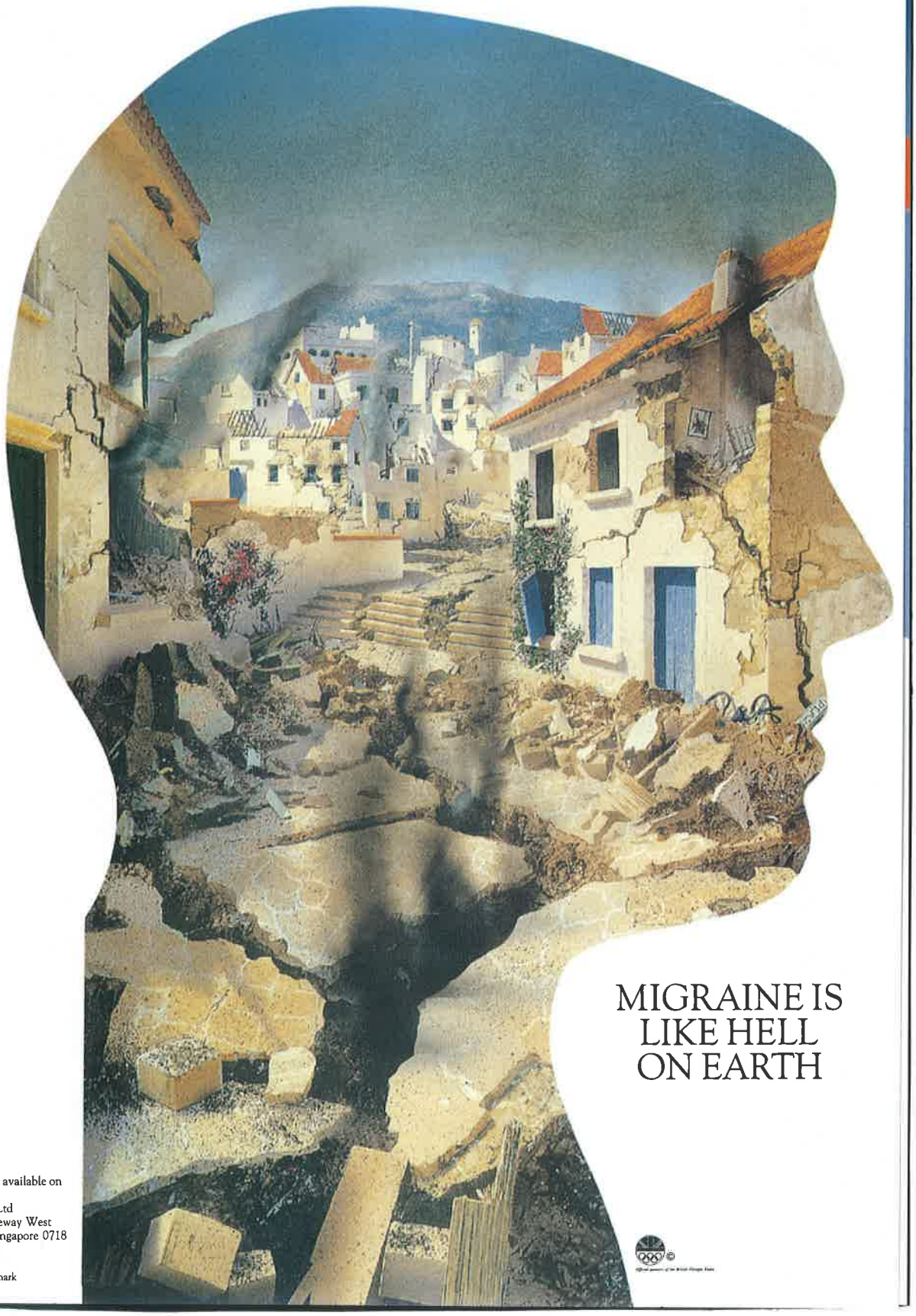

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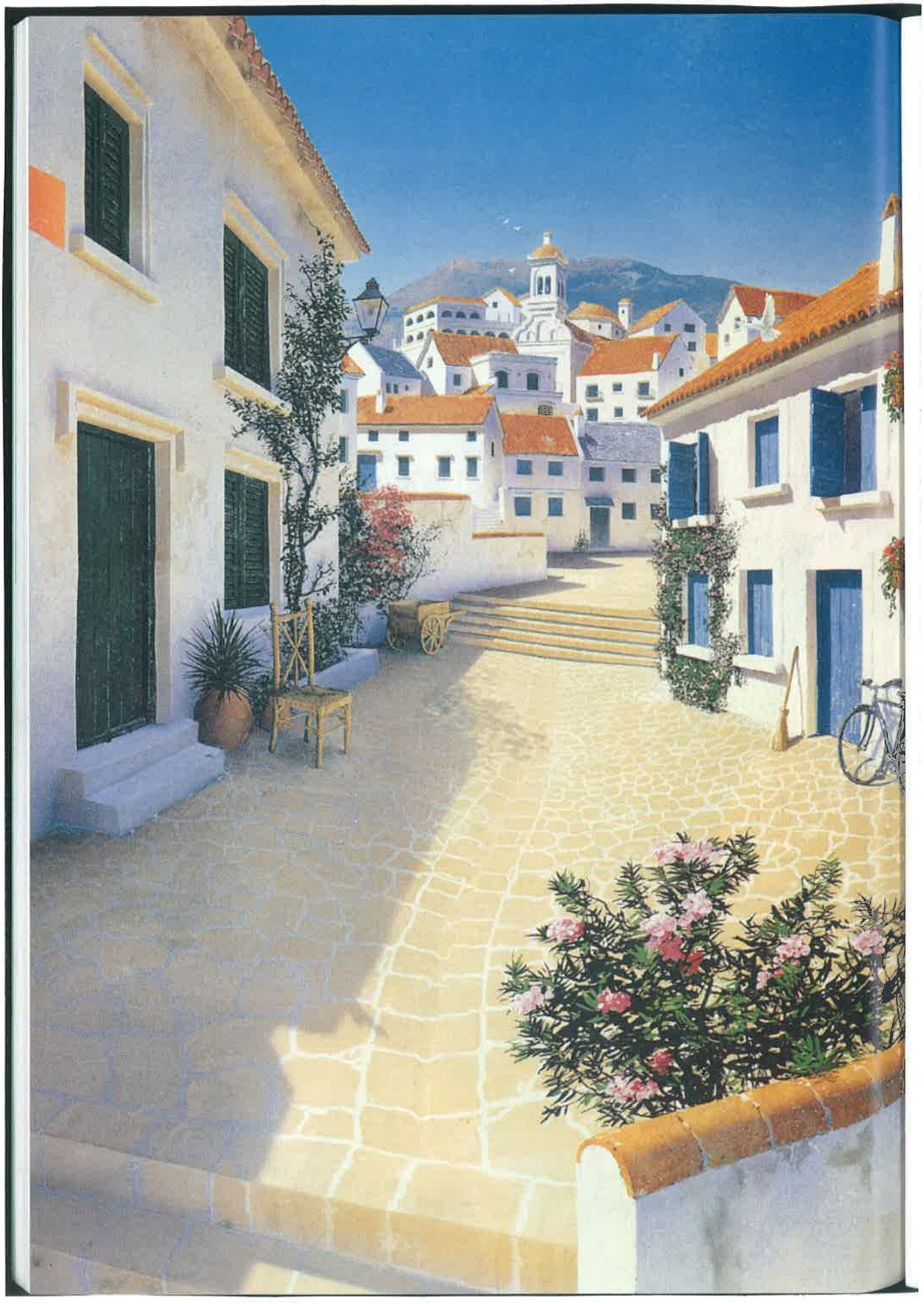

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Imigran (sumatriptan), the first available selective 5-HT_1 agonist, is highly effective at relieving migraine headache whether or not it is associated with aura. It also relieves associated symptoms, such as nausea, vomiting, photophobia and phonophobia, and is effective against migraine headache whether taken early in the attack (<4 hours), or later.[1]

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