The Singapore Family Physician

The College of General Practitioners Singapore
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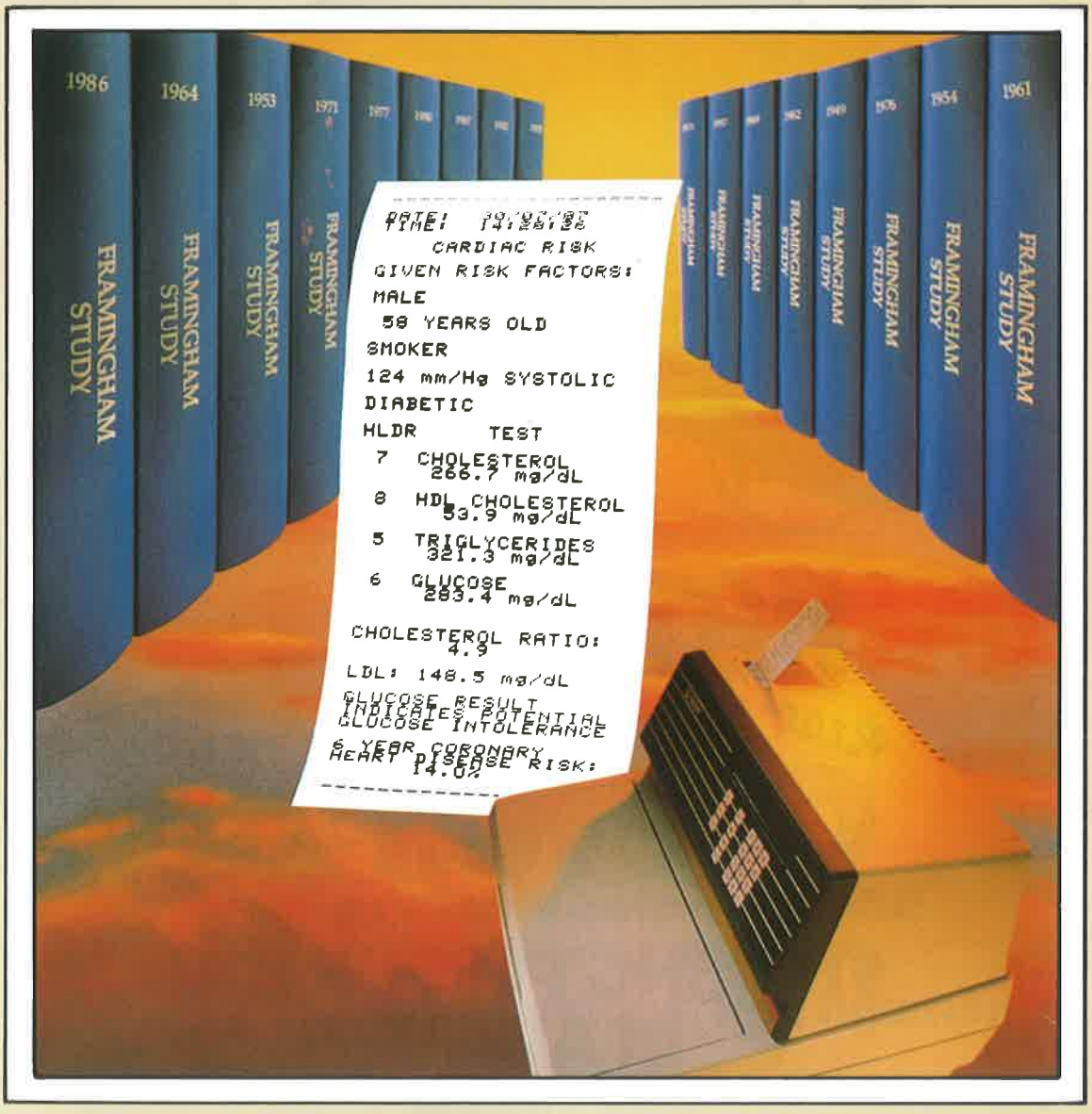

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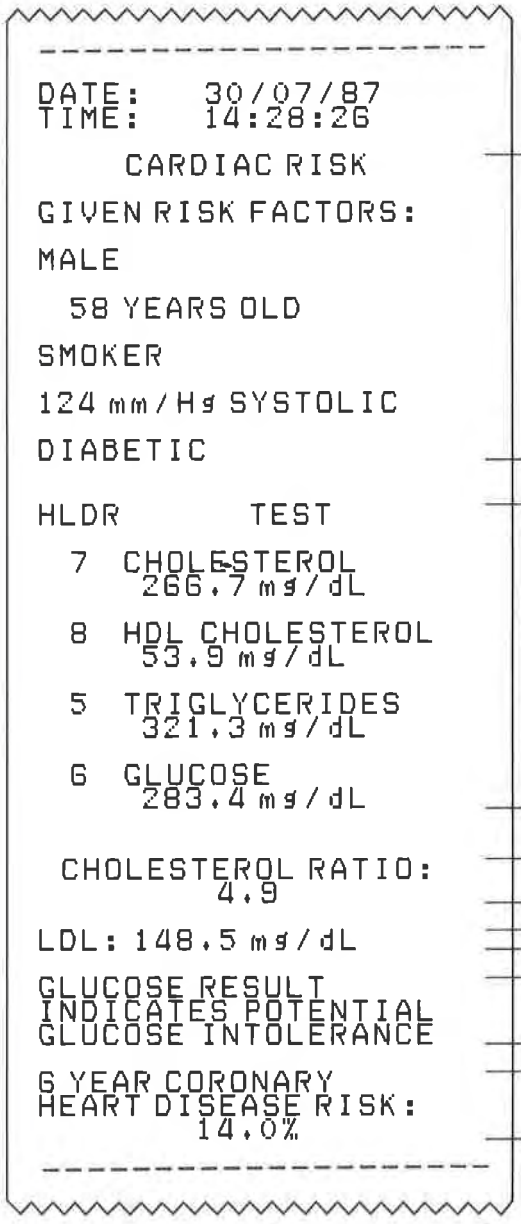

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EDITORIAL

THE PREVENTION OF LIVER CANCER

With the availability of the plasma vaccine in 1982 and the development of the new recombinant DNA hepatitis B vaccine together with the establishment of the causal relationship between hepatitis B and hepatic carcinoma, the prospects of eradicating hepatitis B and reducing the incidence of liver cancer are promising.

The management of hepatitis B is no longer limited to diagnosis and supportive treatment. The general practitioners now have very important roles to play in the war against hepatitis B and hepatic carcinoma. As doctors of first contact, general practitioners have a very important responsibility in advising and educating patients with regard to vaccination against hepatitis B.

The control of hepatitis B is of special importance to us as the incidence of hepatitis B carrier is about 6% of the general population in Singapore and we also have the accompanying high prevalence of the serious sequelae of hepatitis B (HBV) infection such as chronic active hepatitis, cirrhosis and primary liver cancer.[1]

Unfortunately, the extensive publicity in the press have given rise to serious misconceptions about the disease and hepatitis B vaccination as well as generating unnecessary anxiety amongst the public. Many people are still confused about the mode of spread of the disease, and tend to focus on the relatively unimportant method of spread by saliva through food instead of the more infected needles used in acupuncture and tattooing. Many also choose to minimise the importance of the spread via promiscous sexual intercourse.

Another misconception is that hepatitis B is an extremely serious disease which will inevitably lead to liver cancer. Dr. C. Lau-Ling rightly pointed out that on the individual level, one should stress that MOST carriers DO NOT develop complications. For example, in Taiwan, the incidence of liver cancer was actually only 527 out of 100,000 carriers. However, in terms of relative risk, this is 217 times greater than normal as the incidence of liver cancer in non-carriers is 2.6 out of 100,000. Dr. Lau-Ling also found that 6.2% of the mothers who were HBsAg +ve lost their antigenaemia, 15.2% of the mothers who were HBsAg +ve/HBeAg +ve lost their HBeAg antigenaemia and 0.8% lost both HBsAg and HBeAg.[2]

A great many patients also take the vaccination for hepatitis B with the impression that this will protect them from contracting hepatitis A and therefore they can eat cockles and clams with impunity after the vaccination. It would be a disaster if we have an epidemic of hepatitis A resulting from such an erroneous belief. It is very sad that there are doctors who are capitalising on such fears and misconceptions to enhance their income instead of clearing up these misconceptions.

We need to conduct a sensible and effective war against hepatitis B with a rational plan for immunisation. It is an utter waste of money to immunise elderly women who are not at risk and who will least benefit from the vaccination. On the other hand, the campaign would be an exercise in futility if close contacts of the carriers and others who are at risk are not immunised because they are unable to afford the high cost of the vaccination.

In the United States, the incidence of hepatitis B in 1981 was 9.2/100,000. Although the plasma derived hepatitis B vaccine was available in 1982, the incidence of hepatitis B rose to 11.5/100,000 in 1985. This was due to the fact that the initial vaccination programme was directed at three high risk groups — health care professionals exposed to blood, staff and clients of institutions for the developmentally disabled, and staff and patients in haemodialysis units. However, it

was found that recent cases of hepatitis B in the United States come from three groups — male homosexuals, parenteral drug abusers, and persons acquiring disease through heterosexual exposure.[3]

For the immunisation programme to be effective, it is therefore important to identify the major sources of infection. It has been postulated that one of the principal reasons for the high rate of HBV endemicity in the region is the vertical transmission from mother to infant which is responsible for about half of all cases. It has also been found that HBe antigenaemia tends to persist in these infected children. In her study, Dr Lau-Ling found that the spontaneous clearance of HBeAg among infected children was only 2%.[2]

Horizontal transmission of the virus between infected and non-infected members of a family may occur through the sharing of razor blades, toothbrushes as well as through sexual intercourse between the marital partners. Professor Somsak Loleka thinks that transmission of the virus through saliva may be common in Thailand. He observed that gingivitis is common there resulting in bleeding gums. He also noticed that mothers often apply saliva on to their children's cuts and abrasions and there was common sharing of spoons and chopsticks among parents and children. He also noted that the incidence of horizontal transmission declines as the socio-economic status rises[1]

Neonatal vaccination is therefore the key to the control of hepatitis B. With the high cost of the vaccine, it is imperative that the most cost-effective method of mass vaccination be adopted. A strategy of mass vaccination based on the patients' ability to pay will not ensure that those who need the vaccination most will be vaccinated.

The control of tuberculosis was successful in Singapore as all tuberculous patients were given free treatment and chest X-rays for their contacts. It is therefore essential that financial obstacles towards screening for hepatitis B and vaccination be removed from the family contacts of hepatitis carriers.

The second responsibility of general practitioners in the prevention of liver cancer is the follow-up of hepatitis carriers for the early detection of hepatoma. The cost of six-monthly tests for alpha foeto-protein and abdominal ultrasound is high. Furthermore, we need to be aware of the potential psychological damage of such tests in patients who are intrinsically anxious. The anxiety induced in such patients may be more crippling than we realise. We need to find more definitive answers as to who, when and how to screen hepatitis carriers for early hepatomas which are still in the resectable stage.

Finally, in our enthusiasm to eradicate liver cancer, it is so easy to focus all our attention on the disease and to forget that we are dealing with people who have feelings and fears which must be addressed. Our responsibility is to promote health and not to inculcate fears and emotional distress. We now have some tools to combat liver cancer. Let us use them wisely, not simply to line our pockets, but to promote health among our fellow citizens, whilst reducing the costs of health care.

PK

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FROM THE TEACHING SEMINAR

DIABETES MELLITUS – A PRACTICAL REVIEW FOR THE GENERAL PRACTITIONER

Dr Chan Cheow Ju, *MBBS (S), FRACGP, MCGP (S)*

INTRODUCTION

DIABETES AND THE GENERAL PRACTITIONER

Diabetes, together with hypertension, form the bulk of consultations for chronic illness in general practice. In Singapore, 5% of the population over age 40 years have diabetes. About 50% of diabetic are asymptomatic and detected only on screening the population.[1]

Diabetes may cause acute complications like diabetic ketoacidosis, hyperosmolar coma and chronic complications like retinopathy, nephropathy and diabetic foot gangrene. These can be prevented by early diagnosis and good control of the diabetic state.

The general practitioner has a very important role in early diagnosis, early and continuing treatment and early detection of complications of the illness. Patient education and motivation has emerged as one of the most important pillars in the management of diabetes. It is particularly important to achieve tight control of blood sugar which is crucial to prevent long term complications. The family doctor, by providing personal and continuing care, is well placed for this important task.

This article will discuss some pointers and problems in areas of interest to the general practitioner. It is based on a presentation and discussion at the teaching seminar on "Continuing Care — Diabetes Mellitus" organised by the College of General Practitioners Singapore.

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SCREENING OF AT-RISK GROUPS

The General Practitioner should screen the following groups of people who are at risk of developing diabetes:

1. Age over 40 years.
2. Family history of diabetes.
3. Obese adults.
4. All pregnant women.
5. History of subfertility, big babies, miscarriages and stillbirths.
6. Related diseases: hypertension, pulmonary tuberculosis, atherosclerotic vascular diseases.
7. History of glucose intolerance.

THE WHO DIAGNOSTIC CRITERIA FOR DIABETES MELLITUS

The latest WHO recommendations (1985)[2] and the practical problems and implications for the general practitioner are discussed below.

Random blood glucose (RBG)

In the individual presenting with classical symptoms of polyuria, polydipsia, loss of weight, and even diabetic coma in severe cases, a single raised RBG above 200mg/dl is diagnostic.

In the asymptomatic individuals, interpretation of the result as recommended by the WHO study group is shown in figure 1.

In practice, this may be translated into a flow chart as in figure 2. Essentially,

(a) a RBG $<$ 80mg/dl excludes the diagnosis of diabetes;

(b) a RBG $>$ 200mg/dl on 2 occasions confirms the diagnosis of diabetes;

(c) a RBG 80-200mg/dl is an equivocal result.

FIG. 1: UNSTANDARDIZED (CASUAL, RANDOM) BLOOD GLUCOSE VALUES

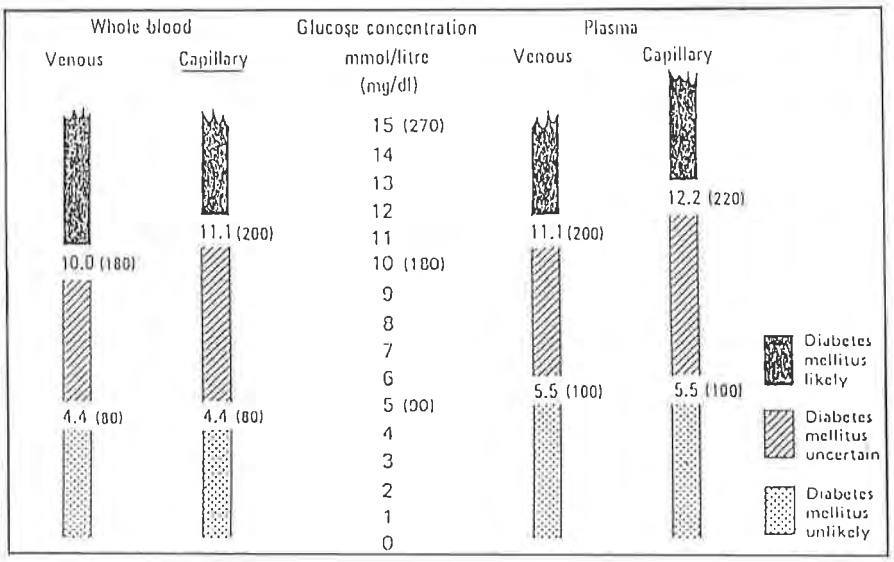


FIG. 2: FLOW DIAGRAM TO ESTABLISH DIAGNOSIS OF DIABETES MELLITUS

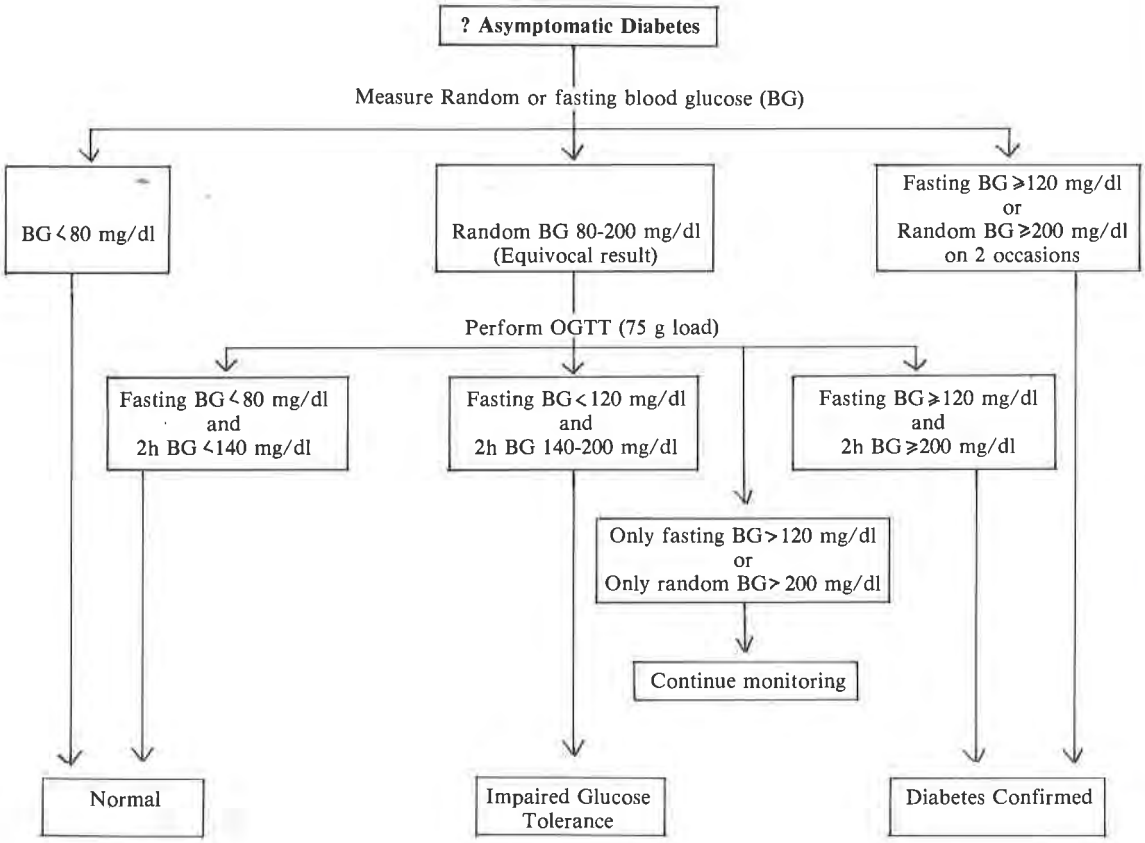


TABLE 1: DIAGNOSTIC VALUES FOR THE ORAL GLUCOSE TOLERANCE TEST

	Glucose concentration, mmol/litre (mg/dl)			
	Whole blood		Plasma	
	Venous	Capillary	Venous	Capillary
Diabetes mellitus				
Fasting value	≥6.7 (≥120)	≥6.7 (≥120)	≥7.8 (≥140)	≥7.8 (≥140)
2 hrs after glucose load	≥10.0 (≥180)	≥11.1 (≥200)	≥11.1 (≥200)	≥12.2 (≥200)
Impaired glucose tolerance				
Fasting value	<6.7 (<120)	<6.7 (<120)	<7.8 (<140)	<7.8 (<140)
2 hrs after glucose load	6.7-10.0 (120-180)	7.8-11.1 (140-200)	7.8-11.1 (140-200)	8.9-12.2 (160-220)

Oral glucose tolerance test (OGTT)

The latest diagnostic criteria as recommended by the WHO study group is shown in table 1.

In relation to the recommendations of the WHO study group, some interesting pointers and problems in general practice are discussed below.

Pointers and Problems

1. Most local general practitioners use test-strips and simple glucometers to measure capillary blood glucose to establish the diagnosis of diabetes. The WHO study group report says that "the method gives reasonably quantitative results", and "can have a coefficient of variation of less than 8%", "Diabetes may be strongly suspected from the results of the estimation, but the diagnosis cannot be confidently excluded. Confirmation of diagnosis requires estimation by chemical methods." In practice, for borderline results, it would seem wise to repeat the blood glucose estimation and seek confirmation with standard laboratory methods.

2. When collecting samples for laboratory determination of blood glucose for the OGTT, it is best to send the patient to the laboratory directly for the performance of the test, because blood glucose values fall rapidly after collection.

3. The confusion of differing blood glucose results from whole blood, plasma, venous and capillary blood samples, in the fasting and post-prandial states, is obvious from table 1. In practice it is useful to stick to one type of blood sample, and know the important demarcating values, e.g. for capillary blood glucose:

   80mg/dl : RGB < 80 mg/dl, diabetes very unlikely

   120mg/dl : Fasting BG > 120mg/dl is abnormal

   140mg/dl : 2h post-glucose load value of 140-200mg/dl implies impaired glucose tolerance

   200mg/dl : 2h post-glucose load value over 200mg/dl implies diabetes

4. In asymptomatic subjects, 2 abnormal blood glucose values in the diabetic range is desirable to establish the diagnosis. This is to guard against spurious results due to technical errors.

5. Problems with borderline cases may arise in the following instances:

   (a) An abnormal 2h post-glucose load > 200mg/dl, but a fasting blood glucose < 120 mg/dl. Here, the WHO says that "for epidemiological or population screening purposes, the 2-hour value may be used alone or with the lasting value." The finding of a raised glycosylated haemoglobin (HbA_1 C) values would also support the diagnosis of diabetes.

   (b) An abnormal RBG > 200mg/dl in an asymptomatic subject not confirmed by a repeat RBG. In practice, it would appear sensible to proceed with the more standardised modified OGTT rather than repeat the RBG; alternatively, with borderline results e.g. RBG 210mg/dl, it may be better to send the patient to a standard laboratory for the OGTT.

(c) An abnormal RBG > 200mg/dl in a subject with an acute illness e.g. a viral fever. Here, an OGTT, even if abnormal during the illness, may not be clearly abnormal after convalescence. This is a subject at risk, but non-diabetic; generally, an OGTT should not be done during episodes of stress.

The point is that the clinician must always feel confident that the diagnosis of diabetes is fully established since the physical and psycho-social consequences for the patient are considerable and life long. With borderline cases, it is best to maintain periodic surveillance until the diagnostic situation becomes clearer. For risk reducing intervention, the clinician should also take into consideration the family history, age and presence of obesity; the therapeutic course of action should emphasize general measures of patient education and dietary advice.

6. Impaired glucose tolerance (IGT) is diagnosed from the OGTT results, where fasting capillary BG <120mg/dl, and 2h post-load BG is 140-200mg/dl. The most important feature of IGT subjects is that these patients have increased risk of macrovascular disease (i.e. atherosclerotic cerebro-vascular, coronary artery and peripheral vascular disease). Although only a small percentage subsequently develop diabetes, it is prudent to aim for risk reduction by maintaining ideal body weight and encouraging a diet with high complex carbohydrates, low refined sugar, low saturated fats and high fibre.

7. For the diagnosis of gestational diabetes, the WHO study group stands by the standard 75g glucose load recommendation. In practice, many local pregnant women have difficulty tolerating the 75g load in the early stages of pregnancy. The fasting and 2 hour post-prandial blood glucose values gives some indication of gross gestational diabetes, until the 75g glucose load can be tolerated, and the modified OGTT performed.

MANAGEMENT OF THE DIABETIC PATIENT: OVERALL CONSIDERATIONS

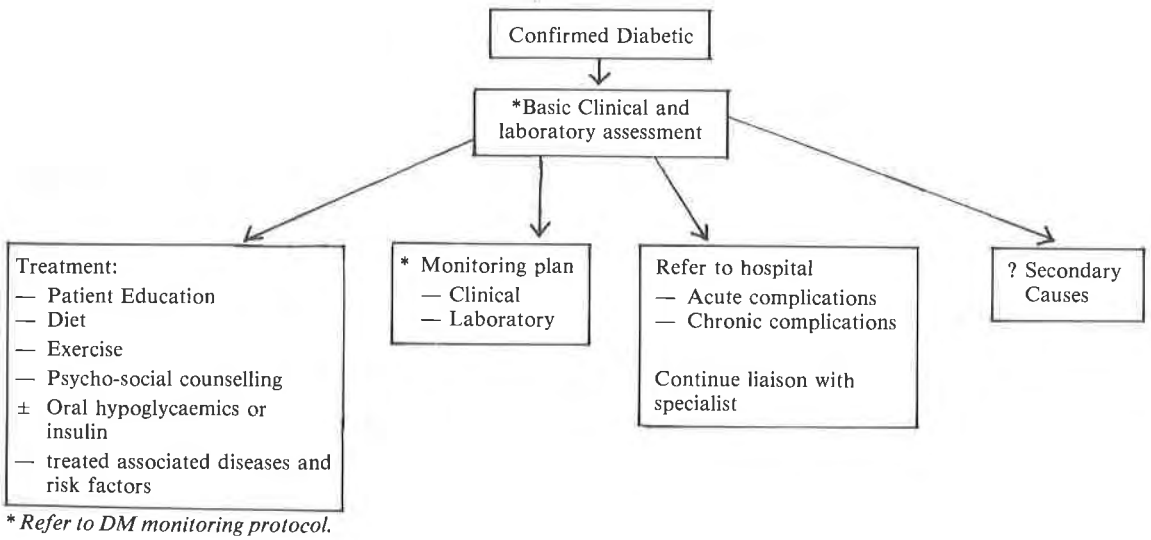
For every new diabetic, the family doctor will need to have an overview of his management plan. One such plan is shown in figure 2. The aims of long-term management would be:

1) Good biochemical control
2) Prevention and early diagnosis of long-term complications
3) Attention to various psycho-social factors.

The therapeutic modalities to consider are:

1) Patient Education
2) Psychosocial management

FIG. 2: MANAGEMENT PLAN FOR THE NEW DIABETIC PATIENT



** Refer to DM monitoring protocol.*

3) Oral hypoglycaemics
4) Insulin
5) Treatment of associated diseases and risk factors.

PATIENT EDUCATION — A CHECKLIST AND GENERAL POINTERS:

A useful checklist of topics for patient education is given in table 3. To start with, the doctor will have to establish a good doctor-patient rapport, by showing empathy and attending to various psycho-social concerns of the newly diagnosed patient. He would then be well placed to motivate the patient (and his family) to participate actively to achieve good blood sugar control and aim towards a normal life.

While different items of the checklist may be highlighted to different individuals, the areas of meaning of diabetes, diet and exercise are always important.

The busy practitioner will usually take several sessions to complete the checklist; in addition dietary advice will often have to be reinforced again and again, and specific advice on matters like fasting, hypoglycaemia and sick-day advice will be raised again when appropriate.

TABLE 3: PATIENT EDUCATION: CHECKLIST

TOPIC	DATE	COMMENTS
Meaning of Diabetes		
Diet		
Tablets		
Insulin		
Insulin Injection		
Technique		
Urine Testing		
Blood Sugar Monitoring		
Record Keeping		
Hypoglycaemia		
Hyper-glycaemia		
Exercise		
Foot Care		
Sick Days		
Travelling		
Complications		
Referral to Diabetic Society		
Patient education handouts		

The task of patient education may be partly delegated to a trained nurse (as in government polyclinics), reinforced by patient education handouts, or even by organising group viewing of videos and slide shows on diabetes. Patient education aids are readily available from the Training and Health Education Department, Ministry of Health, various pharmaceutical firms and the Diabetic Society of Singapore.

The patient will also benefit from participation in various programmes organised by the Diabetic Society of Singapore. One such programme is shown in fig. 4.

FIG. 4: SELF-CARE IN DIABETES — PROGRAMME OF A ONE-DAY COURSE BY THE DIABETIC SOCIETY OF SINGAPORE

7.30 am	Registration, Blood and Urine sugar Tests
8.00 am	Breakfast with the diabetic team *(Doctor, Nurse & Dietitian)*
8.45 am	The Meaning of Diabetes *(Doctor)*
9.15 am	Principles of Diet *(Dietitian)*
9.45 am	Technique of Insulin Injection *(Nurse)*
10.15 am	Coffee Break
10.45 am	Insulin Therapy *(Doctor)*
11.15 am	Blood sugar Tests
11.45 am	Food Exchanges and Sick Days *(Nurse)*
12.30 pm	Lunch with Diabetic Team
1.30 pm	Oral Hypoglycaemic Agents *(Doctor)*
2.00 pm	Foot Care *(Nurse)* and Exercise *(Physiotherapist)*
2.30 pm	Blood sugar Tests
3.30 pm	Tea Break
4.00 pm	Discussion and Rap Session with Diabetic Team

Diet is the cornerstone of treatment for all diabetics. Diet alone can control about one third of new diabetics. While the details of a diabetic diet and food exchange portions may appear too complicated for practical instruction by the general practitioner, the basic principles of a diabetic diet as outlined below can be clearly and emphatically communicated to the patient:

1) It is basically a normal healthy diet (beneficial to all in the family). Special attention would be in areas given below.
2) Prohibit all simple sugars.
3) Control weight and total calories — aim towards ideal body weight. A useful for-

mula to estimate this by American standards would be:

female : 100 lb for first 5 feet of height, plus 5 lb for each additional inch.

male : 106 lb for first 5 feet of height, plus 6 lb for each additional inch.

Our local standards would probably be about 10% less.

4) Spread out meals (small to moderate amounts) and snacks.

5) Control saturated fat intake.

6) Increase fibre intake.

Complicated instructions should be avoided; simple handouts are more useful. An example of such handout is shown in fig. 5.

A useful practical strategy is to ask the patient to chart his diet over a one week period (e.g. just before or just after consultation), monitor the patient's weight regularly, point out the deviations and excesses in the patient's diet and recommend suitable alternatives (e.g. artificial sweeteners for refined sugar, skimmed milk for condensed milk, soup kuay teow for fried kuay teow).

For the elderly, a permissive diet (apart from simple sugars and curbing excesses in daily intake) is kinder and may be the only practical diet. On the other hand, for the insulin dependent diabetic and the young diabetic, attention must be paid to the spread of meals and snacks, and expert dietary advice on food portions and exchanges would be worthwhile.

Exercise. Every diabetic subject should be encouraged to keep active. Exercise can enhance the sense of physical and psychosocial health; many patients would enjoy taking on exercise as another active role in the management of their disease. Exercise can help to reduce weight, improve glucose intolerance and hyperglycaemia (by enhancing the action of insulin), improve cardiovascular fitness and decrease cardiovascular risk factors (obesity, hypertension and hyperlipaedaemia.)

Smoking must be strictly prohibited as it increases tremendously the cardiovascular risks of the diabetic.

PSYCHOSOCIAL MANAGEMENT

The family doctor needs to be aware of the psychosocial problems in this chronic disease. Initially, the diagnosis of diabetes mellitus may spark fear, anxiety, and even a panic reaction. The manner in which it is handled will influence the patient's response to further management. Tact and reassurance should be combined with clear, accurate information

FIG. 5: HANDOUT ON DIET FOR PATIENT WITH DIABETES

DIET IN DIABETES MELLITUS			
FOODS NOT ALLOWED		FOODS ALLOWED IN RESTRICTED QUANTITIES	
Sugar, Jam, Honey, Condensed milk, Kaya, Glucose Chocolates, Sweets & Cakes Ice-cream, bobo-chacha, Chendol, Ice-kachang, Jelly Soft drinks, Syrups Sweet wines, Beer, Stout & Cider Dried Fruit, Fruit-cocktail, Tinned fruits		Rice, Mee, Beehoon, Kuay-teow, Macaroni, Bee Tai Bak Breakfast cereals, Bread, Chappatis Fruits, except lemons, lime, grapefruit Topioca, sweet potatoes, potatoes, yam	
FOODS ALLOWED FREELY			
Fish & Seafood Poultry & Eggs Meat Cheese	Leafy vegetables (chives, kai-lan etc) Cabbage, Cauliflower Ginger, onion, garlic chillies	Gourds Beans Tofu	Coffe (without sugar) Tea (without sugar) Lemonade (without sugar)

Source: Local Diet in Diseases: A sourcebook for patient health education, 1982, published by Association of Private Medical Practitioners of Singapore.

about the meaning of diabetes. Putting across a clear plan of management with the patient's active participation and emphasizing the prospect of a quality normal life with good blood sugar control will help the patient develop a positive approach to his illness.

The family should be involved early in patient (and family) education. The family is a major source of psychological support, technical support (in cooking and serving medications), detection of emergencies, and in many cases, is also a crucial determinant of financial support and continuing consultation. Work environment and schedule would influence the patient's meals, schedule and contents; this should be attended to specifically, with appropriate advice and adjustment of medications, e.g. a night shift worker (with his main meals at night) may need his insulin dose at night and healthy home cooked meals and packet milk drinks would be preferable to sweet snacks in the canteen.

The doctor should also take cognisance of cultural occasions like the fasting month of Ramadan, and the feasting with the Chinese New Year season.

In follow-up, the doctor must continue to attend to any psychological problems arising, e.g. relationship problems with partners arising from the stigmata of diabetes.

USE OF ORAL HYPOGLYCAEMICS

Table 3 gives an indication of the range of common oral hypoglycaemics used in Singapore. Chlorpropamide has been widely used because of the convenience of once daily dosing, and its low cost. However, it is particularly prone to produce prolonged hypoglycaemia, especially in the elderly, and has therefore lost much popularity. The newer generation oral hypoglycaemia like glibenclamide maintains the advantage of convenient dosing with lesser risk of prolonged hypoglycaemia.

Other common problems in practice include:

1) Starting oral hypoglycaemics too early, and insufficient emphasis on the importance of dietary control. About one third of diabetics can be controlled on diet alone; excessive use of oral hypoglycaemics can give rise to hunger pangs, increased meals and increasing weight, aggravating the diabetes. Diet is also the first aspect of treatment to attend to in cases of poor control, before increasing the dosage of oral hypoglycaemics.

2) Inadequate monitoring while on medication. Many patients are lulled into forgetting about diet and blood glucose monitoring, and simply replenish their supplies of tablets. They are most liable to develop chronic complications like atherosclerotic vascular disease, diabetic retinopathy and diabetic nephropathy. The attending doctor must insist on regular monitoring. The aim should be to normalise blood glucose.

One criteria for biochemical control is shown in Table 4.

TABLE 3: ORAL HYPOGLYCAEMICS

Name (and dosage available)	Duration of Action (hr)	Frequency of dosing/day	Total daily dose	Cost per tab/ per 4 weeks	Remarks
Tolbutamide (Rastinon) 500 mg	6-12	2-3	0.5-3 g (1-6 tabs)	20c $6 $36	OPS Supply at $1 per item medication per week
Chlorpropamide (Diabinase) 250 mg	60	1	125-500 mg (½-2 tabs)	30c $4 $16	— Danger of prolonged hypoglycaemia
Glibenclamide (Daonil, Euglucon) 5 mg	12-24	1-2	2.5-20 mg (½-4 tabs)	30c $4 $32	
Glipizide (Minidiab) 5 mg	12-24	1-2	2.5-30 mg (½6 tabs)	30c $4 $48	
Metformin (Glucophage) 250 mg	6-10	2-3	1-3 g (2-6 tabls)	13c $8 $24	

TABLE 4: CRITERIA FOR BIOCHEMICAL CONTROL*

Normalization of BG (Ideal)	: Fasting BG<120 mg/dl 2h PP BG<140 m/dl
Acceptable (Good)	: Fasting BG<140 mg/dl 2h PP BG<200 mg/dl
Fair	: Fasting BG 140-200 mg/dl 2h PP BG 200-250 mg/dl
Poor	: Fasting >200 2h PP>250 mg/dl HB A_1 C 8%

**Modified from S. S. Fajans[3]*

3) In the elderly, where strict changes of diet is impractical and unkind, and the danger of hypoglycaemia outweigh the risk of developing long term complications in the fewer remaining years of life, it is wiser to accept the category of 'fair' control.

4) The situation of "failed oral hypoglycaemics" can occur even after an initial period of control on diet and oral tablets. These patients may need insulin therapy (after checking on dietary and medication compliance).

USE OF INSULIN

Although few insulin requiring diabetics are under the care of the general practitioner, the interested general practitioner with a basic understanding of insulin regimes can actually supervise the patient, either alone or in consultation with specialist colleagues.

The types of insulin may be grouped by

(a) duration of action — short, intermediate or long acting insulins.

(b) purity — beef, pork monocomponents, and human insulins Genetically engineered human insulins are least immunogenic and overcomes the problem of lipodystrophy. They have gradually replaced the older insulins which are now available mainly at government pharmacies.

Some basic considerations of simple insulin regimes are illustrated in table 5 and table 6. Many patients will require an initial total daily dose of 20 to 30 units; adjustments are generally made by 2 to 4 units each time (evey few days if necessary).

TABLE 6: ADJUSTING INSULIN THERAPY

Regime of IZS before breakfast and dinner, with SI before breakfast and dinner

Blood Glucose		Insulin adjustment recommended	
pre-breakfast	BG↑	↑ evening	IZS
pre-lunch	BG↑	↑ morning	SI
pre-dinner	BG↑	↑ morning	IZS
post-dinner	BG↑	↑ evening	SI

Initial total daily insulin usually 20-30 u
Adjustments generally by 2-4 u insulin.

It is interesting to note some basic expenses of the diabetic patient on insulin e.g. the patient using 30 units per day will use about 1 vial of insulin a month, at a cost of $32 per vial (or only $3 at government pharmacies); self monitoring cost will be glucometer cost of about $500 plus test strip cost of about $45 per month (for $1.50/test/day.) Special discounts for glucometer and test strip costs are available for members of the Diabetic Society of Singapore.

In the young insulin dependent diabetic who has many long years to live and who tends to develop complications, home glucose monitoring is well worthwhile, and feasible,

TABLE 5: INSULIN REGIMES: BASIC CONSIDERATIONS

Duration of action	Daily frequency	Diabetic control
• Short, eg soluble	• Multiple	• Good
• Medium, eg insophane	• Twice	• Average
• Long, eg lente	• Once	• Poor

• Commonly used regimen: short-acting (eg soluble) + medium-acting (eg. insophane), twice daily.

to help achieve normalization of blood glucose and minimise complications. The interested general practitioner can help motivate the patient to start such a programme. Help is also readily available through membership of the Diabetic Society of Singapore.

EMERGENCIES IN DIABETIC PATIENTS

The general practitioner needs to be alert to the following emergencies:

1) hypoglycaemia
2) hypersmolar hyperglycaemic coma (and pre-coma)
3) diabetic ketoacidosis and
4) intercurrent illness (see later discussion).

Hypoglycaemia may occur because of excess insulin, unexpected exercise and missed meals. Oral hypoglycaemics can also cause hypoglycaemia. A known diabetic with confused or unusual behaviour should be considered hypoglycaemica and treated as such until proven otherwise because persistent hypoglycaemia can lead to permanent brain damage.

Hypersmolar coma (and pre-coma) usually develops slowly over weeks, either in new or neglected adult diabetic patients, preceded by symptoms of thirst, polyuria, lassitude and weight loss.

Diabetic ketoacidosis (DKA) may develop slowly (over days and weeks) in undiagnosed insulin — dependent diabetics, or more commonly can develop rapidly (over hours to a few days) as a result of a sudden increase in demand for insulin as in intercurrent illnesses like severe infections and acute myocardial infarction. This is compounded by the loss of appetite, nausea and vomiting, and failure to take medications or insulin injections accompanying the precipitating factors and the complication itself.

Differentiating features of hypoglycaemic and hyperglycaemic emergencies are usually apparent from the patient profile, history, physical signs and simple urine and blood tests. (See table 7).

The general practitioner should be prepared to initiate emergency resuscitative measures, namely oral or intravenous glucose for hypoglycaemia, and intravenous fluids and hospitalization for hyperglycaemic comas.

TABLE 7: FEATURES OF HYPOGLYCAEMIC AND HYPERGLYCAEMIC COMAS

hypoglycaemia	Hyperglycaemia
Known diabetics on treatment, sudden onset, feeling funny, confused or odd behaviour	Undiagnosed diabetics, Intercurrent illness, slower onset, polyuria, polydipsia; nausea, vomitting and abdominal pain (in DKA)
Skin pale and sweaty	Skin dry, may be flushed
Pulse bounding, normal or weak	Pulse weak
Breathing rapid and shallow	Breathing deep and slow (in DKA)
BP normal or low	BP low
Blood sugar less than 60 mg/dl	Blood sugar over 400mg/dl
Urine ketones absent	Urine ketones in large amounts (in DKA)

INTERCURRENT ILLNESS

Some practical points may be noted.

1) Intercurrent illness may rapidly cause a deterioration in diabetic control.
2) Patients with type 2 (non-insulin-dependent) diabetes may require temporary insulin treatment.
3) Patients with type 1 (insulin-dependent) diabetes should never have their insulin stopped, even if they have missed a meal.
4) Watch for danger signals (the features of hyperglycaemia discussed earlier).
5) Major intercurrent illness will usually merit hospitalization. In particular, diabetics tolerate infections poorly, and any evidence of systemic toxicity should prompt early referral for intensive hospital management.
6) Early hospital admission is also recommended if diabetic control deteriorates or if in doubt.

A guideline for management of diabetes in the presence of minor intercurrent illness is shown in Table 8.

LOSS OF BIOCHEMICAL CONTROL

When faced with this situation, it is worth reviewing the following checklist:

TABLE 8: MANAGEMENT OF DIABETES IN THE PRESENCE OF MINOR INTERCURRENT ILLNESS

Usual treatment	Diabetic control	Action
• Diet only	Blood glucose<250mg/dl	Ensure hydration and oral fluids
	Blood glucose>250mg/dl	Consider oral tablets or temporary insulin
• Diet + Oral hypoglycaemics	Blood glucose<250mg/dl Blood glucose>250mg/dl	Ensure hydration Increase sulphonylurea or consider temporary insulin
• Insulin	Blood glucose>250mg/dl	Ensure hydration and temporarily increase insulin by 2-4 unit steps

1) Is the patient following a diabetic diet? Failure to do so is the most common cause.
2) Is the patient compliant with medications?
3) Is there any intercurrent illnes? Urinary tract infectious and pulmonary tuberculosis are prominent causes.
4) Are there psychosocial problems — emotional stress, loss of motivation to maintain health, family support problems, work problems?

LONG TERM COMPLICATIONS OF DIABETES MELLITUS

Ensuring tight blood glucose control can prevent long term complications.

Diabetic retinopathy, a major cause of blindness, is eminently treatable by laser photocoagulation. Regular check of visual acuity (e.g. 6 monthly) and expert fundoscopy (yearly, and whenever symptomatic) should pick up diabetic maculopathy (exudates, haemorrhages and oedema around the macule) and diabetic pre-proliferative retinopathy (exudates, haemorrhages, micro-aneurysms) at an early treatable stage. Proliferative retinopathy with new vessel formation can lead rapidly to vitreous haemorrhage, fibrosis, retinal detachment and blindness. Any patient with vision less than 6/9 (not correctable by pinhole or the optician's refraction) and any sign of retinopathy should be referred to an ophthalmologist.

Atherosclerotic vascular complications of coronary artery disease, strokes and peripheral vascular disease are more common in diabetics. In prevention, besides tight control of blood sugar, attention to other risk factors like hyperlipaedaemia, hypertension and smoking must be emphasized.

Diabetic nephropathy develops insidiously. The deterioration of renal function is said to be irreversible once albuminuria is present (an earlier stage of micro-albuminuria may be detected in specialist laboratories). Caution is necessary with the use of insulin and oral hypoglycaemics once nephropathy sets in (the kidneys may not clear these substances well). Aggressive control of hypertension is the most useful measure to slow the decline of renal function. Besides conservative management, referral to a nephrologist should be considered for dialysis and assessment for renal transplant.

Diabetic foot gangrene can arise from a combination of peripheral vascular disease, micro vascular disease, neuropathy and infection. In prevention, the patient should be given clear instruction on care of the feet (Table 9). Rest, daily dressings, antibiotics, antifungel agents, attention to footwear for relief of pressure, and where necesary, surgery, should be considered in the treatment of foot lesions.

Autonomic neuropathy resulting in postural hypotension and impotence can be distressing. Tactfully pointing out the possibility of erectile impotence may encourage diabetics to aim for tight blood glucose control and give up smoking.

TABLE 9: INSTRUCTIONS ON CARE OF THE FEET

INSTRUCTIONS ON CARE OF THE FEET:
1. Inspect the feet daily;
2. Keep the feet scrupulously clean;
3. Wear soft socks and pliable shoes;
4. Do not walk barefoot;
5. Cut the nails carefully;
6. Avoid extremes of temperature;
7. Report to your doctor any ulcers, or swelling in the feet

INDICATIONS FOR SPECIALIST/ HOSPITAL REFERRAL

The following summarizes the common situations for referral:

1. Diabetic hyperglycaemic coma and pre-coma.
2. Severe intercurrent illness
   e.g. pneumonias, pyelonephritis, cellulitis
3. Long-term complications of retinopathy, nephropathy, diabetic foot.
4. Poorly controlled diabetes.

The specialist and general practitioner should continue to liaise on subsequent care of the patient.

ORGANIZATION OF CONTINUING CARE OF THE DIABETIC PATIENT

The IPA Diabetes mellitus monitoring protocol is a useful checklist and recording aid in continuing care of the diabetic (fig. 6). The major components in the protocol are:

A) General: Body weight, blood pressure (obesity and hypertension are important associated conditions to attend to for risk reduction).

B) Blood glucose monitoring.

C) Clinical examination:
   — cardiovascular
   — the limbs: skin, pulses, pin prick, proprioception and tendon reflexes (check 6 monthly).
   — the eyes: visual acuity (check 6 monthly) and fundoscopy (check yearly).

D) Investigations:
   — HbA_1C to be done 3 to 6 monthly
   — other tests to be considered yearly.

E) Medications.

To the above may be added one more category:

F) Patient education: reinforce advice on diet exercise and smpking.

A useful checklist for practice audit of diabetic care is shown in table 10.

TABLE 10: PERFORMANCE REVIEW CHECK LIST

General

Does the practice have a policy for the detection and general management of diabetes?

Patients on treatment

1. Is it clear from the records on what basis the diagnosis was made?
2. Is there any record of the patient's ideas or the information given to the patient?
3. Is there a patient education checklist?
4. Are the current drugs and dosage clearly recorded?
5. What proportion of patients on treatment have fasting blood glucose levels above 40mg/dl or random levels above 200mg/dl?
6. How many patients are members of the Diabetic Association?

Emergencies

1. Does the practice have a policy for the management of hyperglycaemic or hypoglycaemic coma?
2. Is there a record of patient instructions on what may make their diabetes go out of control and what to do?

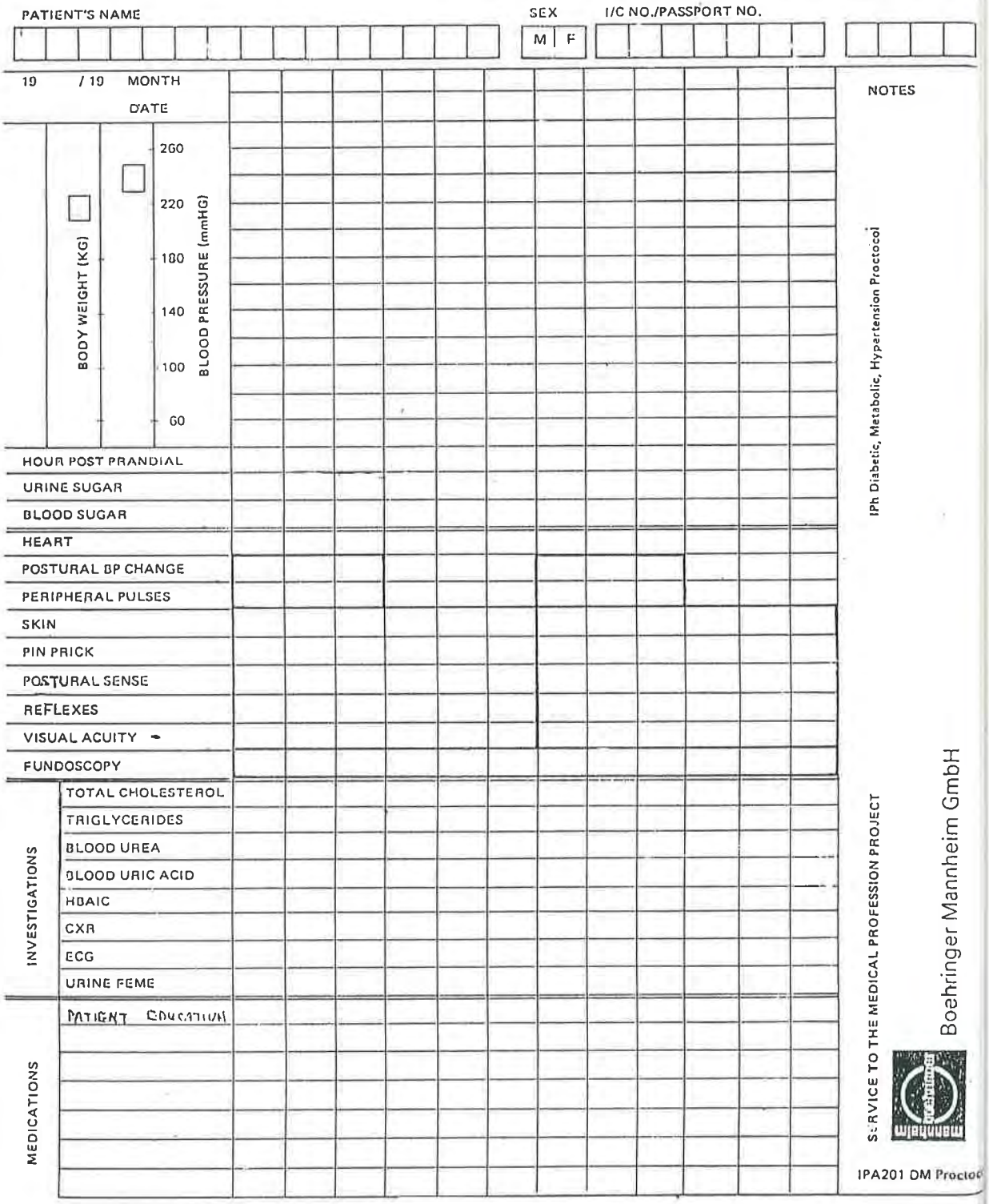
RESEARCH

Many general practitioners have participated actively in a recent survey on diabetic control conducted by the college research committee. (The results will soon be published). As the majority of diabetics and patients with glucose intolerance are diagnosed and followed-up by general practitioners, the latter can look forward to active participation in research in the areas of early diagnosis, patient education, degree of control and the development of long term complications.

CONCLUSION: CHALLENGE TO THE GENERAL PRACTITIONER

Recent scientific advances have brought into reality the possibility of a quality normal life for diabetes. What is required is for these

FIG. 6: FLOW CHART FOR DIABETES MELLITUS



ideas and methods to be translated into benefits for the patient. The family practitioner should be well placed to do this. He will need to:

1) continue providing personal and continuing care for the patient;
2) continue keeping up with recent advances on diabetes; and
3) continue auditing and organising his practice for long term management.

The challenge is for the general practitioner to do as good a job, if not better, than the hospital diabetic clinic.

ACKNOWLEDGEMENTS

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3. S.S. Fajans, Chapter on The Adult Diabetic Patient in Current Therapy in Internal Medicine, Ed. 1987. Chapter on The Adult Diabetic Patient Pg 535-544.

Recommended Reading

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Editorial, Journal of the Royal College of General Practitioner September, 1987.

— Diabetes and Diabetology in Singapore.
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— Clinical Manifestations of Diabetes Mellitus.
Dr Chua Wan Tiong, Family Practitioner, Vol. 5 No. 2 August, 1982.

— Management of Diabetes Mellitus in General Practice.
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— Diabetes and intercurrent illness in Geneal Practice.
H.J. Bodansky, Update September 1986, Pg 429-436.

Other Recommended Materials

— Patient education materials from:
  — Training and Health Education Department, MOH.
  — Diabetic Association of Singapore.
  — Pharmaceutical Firms.

Audio Digest Casette tapes on Diabetes.

— Faimly Practice Vol. 34 No. 36 and Vol. 35 No. 26.

UPDATE ON GASTROENTEROLOGY — 1

A Lecture series on 'Common Gastroenterological Symptoms in General Practice' was organised by the Division of Gastroenterology, Department of Medicine, National University Hospital in November 1987. We publish 3 lectures of the series.

EVALUATION OF CHRONIC UPPER ABDOMINAL PAIN

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INTRODUCTION

Abdominal pain is one of the commonest problems encountered in clinical practice. This article focuses on chronic upper abdominal pain. A series of patients seen at a gastroenterological clinic with this complaint was analysed to determine the relative frequency of underlying diseases and the diagnostic procedures required. The value of various associated symptoms in the differential diagnosis was also studied.

DESIGN OF STUDY

A consecutive series of patients referred from general practitioners to the author during the year 1986 was analysed. Only patients who had chronic upper abdominal pain as the only complaint, or as one of the main complaints were included.

There were one hundred and eleven patients in all. Of these, eleven were not fully investigated. Exactly one hundred patients were therefore available for study. Of these 53 were males and 47 females. Their ages were 47 ± 14 years (mean ± standard deviation) for females and 52 ± 14 years for males.

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RESULTS

The final diagnoses of the patients are listed in table 1. Two-thirds of the whole series turned out to have functional disease, arbitrarily divided into functional dyspepsia and irritable colon. Only one-third of patients had structural diseases. Duodenal ulcer accounted for half of this latter group, other diagnoses encountered being gastric ulcer, gallstone and gastrointestinal carcinoma. One patient turned out to have a cardiac problem.

TABLE 1: FINAL DIAGNOSES OF CHRONIC UPPER ABDOMINAL PAIN

	Female	Male	All
Irritable colon	24	23	47
Functional dyspepsia	10	11	21
Duodenal ulcer	9	7	16
Gastric ulcer	1	5	6
Gallstones	2	3	5
Carcinoma	0	4	4
Cardiac pain	1	0	1
Total	47	53	100

Diagnostic tests perform on these patients are shown in table 2. Some of these tests were in fact carried out prior to referral. Gastroduodenoscopy was performed on most patients referred for upper abdominal pain, this being the reason for referral in many cases as this was the only investigation required for most patients with peptic ulcer and gastric carcinoma. Abdominal ultrasound was the investigation of choice for the diagnosis or exclusion of gallstones. More investigations were required before the diagnosis of func-

TABLE 2: DIAGNOSTIC TESTS PERFORMED

	Irritable colon	Functional dyspepsia	Peptic ulcer	Gallstone	Carcinoma
Haemoglobin	64	48	9	20	0
ESR	57	33	0	20	0
Gastroscopy	96	95	95	60	100
Barium meal	13	5	14	0	25
Colonoscopy	30	0	0	20	25
Sigmoidoscopy	40	0	0	0	0
Barium enema	4	0	0	0	0
Ultra sound	32	52	0	100	25

All values given are percentages
n = 100

tional bowel disease could be made. These include blood count, erythrocyte sedimentation rate, abdominal ultrasound examination, sigmoidoscopy, and barium enema or colonoscopy.

Table 3 shows the age and duration of abdominal pain in patients with various disease. Patients with organic disorders tended to be older than those with functional disease. Patients with organic disease, with the exception of those with duodenal ulcer, also tended to have a shorter history compared especially to patients with irritable colon.

Various associated symptoms were studied to see whether they could contribute to a positive diagnosis without the necessity for specialised investigations. The results are shown in table 4. Although duodenal ulcer patients invariably stated that their pains were related to food in some way (either excerbated by hunger, improved by food, or excerbated by food), this feature was also reported by a large proportion of patients with other diseases. It therefore had little or no discriminative value. However, if a patient reported that his or her pain was relieved by defecation, the cause of the pain usually turned out to be irritable colon.

Severe pain, defined as pain requiring an injection, occurred relatively commonly in patients with gallstone and functional disorders, but uncommonly in peptic ulcer. Nocturnal pain waking the patient up was more likely to occur in organic rather than functional disorders. However, because a third of patients with functional disorders also reported this symptom, it could not be used for differentiating organic from functional disorders.

If a patient had both abdominal pain and jaundice, biliary tract disorders like gallstones would be the likely diagnosis. Anorexia and weight loss occured more frequently in organic rather than functional disease. However, because a significant proportion of patients with non-organic disorders also complained of these symptoms, their discriminative value was low.

Many patients, regardless of diagnosis, complained of constipation. The combination of abdominal pain and diarrhoea, however, usually indicated that the patients suffered from irritable colon. Abdominal distension was also more common amongst irritable colon patients than patients with other diagnoses. A history of smoking or analgesic ingestion was of no value in differentiating between the various causes of abdominal pain. If a patient recognised that his/her symptoms were related to stress, however, irritable colon became the most likely diagnosis.

CONCLUSION

At least two-thirds of patients presenting with chronic upper abdominal pain to a gastroenterological clinic have functional rather than organic disease. With the exception of duodenal ulcer patients, patients with organic disease tended to be older and to have a shorter duration of symptoms when compared to patients with functional disorder.

TABLE 3: AGE AND PAIN DURATION FOR VARIOUS CAUSES OF ABDOMINAL PAIN

	Irritable colon	Functional dyspepsia	Duodenal ulcer	Gastric ulcer	Gallstone	Carcinoma
*Age	38 ± 12	37 ± 13	44 ± 13	48 ± 20	34 ± 9	57 ± 17
† Duration of pain	73 ± 71	25 ± 30	87 ± 120	24 ± 38	6 ± 8	10 ± 13

*All values given as years, mean ± standard deviation

† All values given as months ± standard deviation

TABLE 4: DISCRIMINATIVE VALUE OF VARIOUS SYSMPTOMS

	Irritable colon	Functional dyspepsia	Duodenal ulcer	Gastric ulcer	Gallstone	Carcinoma
Pain related to food	77	67	100	67	40	75
Pain relieved by defecation	21	5	13	0	0	0
Severe pain	21	19	0	17	40	25
Nocturnal pain	30	33	69	33	60	50
Jaundice	0	0	0	0	40	0
Anorexia	17	14	13	50	40	0
Weight loss	19	19	19	50	50	25
Vomiting	17	10	13	33	0	0
Diarrhoea	66	10	0	0	88	0
Constipation	34	14	25	0	20	50
Distention	60	29	6	16	0	0
Smokers	15	5	25	33	20	25
Analgesics	0	5	6	0	0	0
Stress	13	0	0	0	0	0

All values given are percentages
n = 100

Patients who had associated diarrhoea or abdominal distension, and those whose pains were relieved by defecation, usually turned out to have irritable colon.

Comments

The large proportion of patients in the present series with functional rather than organic disease is in keeping with previously published data.[1] Indeed, patients referred for gastroenterological assessment form a highly selected group. If all patients abdominal pain general practitioners with abdominal pain were to be considered, the proportion of patients with functional disease would probably be much higher. The low prevalence of anorexia and weight loss in our carcinoma patients emphasized that this was a highly selected group of patients. Typical cancer patients would probably have been directly referred to a surgical clinic.

Patients in whom no structural lesion could be found are said to have functional dyspepsia if their symptoms are thought to originate from upper gut, or irritable colon if the symptoms are thought to arise from the colon. Patients with functional dyspepsia therefore have abdominal discomfort or pain related in some way to food ingestion. How these symptoms arise is unclear. Patients are diagnosed to have irritable colon if they have abdominal pain and alteration of bowel habit which cannot be attributed to structural disease. In formal studies of irritable colon patients, abdominal distension and pain relieved by defecation are found to be useful symptoms to differentiate functional from organic disease.[2,3]

It is obviously incumbent upon the physician who diagnoses functional disease not to miss a treatable organic lesion. One

would be especially worried about missing a cancer. A careful history and physical examination is required. Specialised investigations including blood count, erythrocyte sedementation rate and gastroscopy or barium meal are mandatory in patients over 40, especially those who have a short history. For patients who also report a change in bowel habit, sigmoidoscopy is valuable not only to exclude rectal or perianal lesion, but also for performing the air insufflation test. In this test air is pump into a patient's colon. The test is said to be positive if the patients develop this typical pain with air insufflation and if the is relieved by aspirating the air at the end of the procedure. A positive test indicates that the pain is colonic in origin. A barium enema or colonoscopy may also be indicated in patients with a change of bowel habit if they are over 40 years of age, or if their symptoms have been recent in onset. Rectal bleeding or anemia associated with change of bowel habit would of course, make full colonic assessment essential.

Since not every patient with funçtional disease was exhaustively investigated, it is possible that patients with organic diseases may have been missed in this series. Other patients may harbour organic disease while suffering from functional symptoms. It is however reassuring to note that in follow-up studies on patients with functional gastrointestinal orders as diagnosed conventionally, very little organic disease was in fact missed.[4,5]

Thorough investigation in patients with functional bowel disease is of considerable therapeutic value. Many patients will feel much happier if they can be reassurred that their symptoms are not due to any serious disease. On the other hand, over-investigation results in unnecessary discomfort and financial loss for the patients. Clinical judgement is required to achieve a happy medium.

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EVALUATION OF DIARRHOEA – A PATHO-PHYSIOLOGICAL APPROACH

Dr I Yap, *MBBS, M Med (Int Med) AM*

INTRODUCTION

The pathophysiology of diarrhoea is discussed. A series of patients with diarrhoea presenting to a gastroenterological clinic was studied to determine the common causes of diarrhoea in Singapore. A pathophysiological approach to the investigation of diarrhoea is suggested.

Definitions

Diarrhoea is defined as an increase in stool frequency from the arbitary upper limit of normal of 3 times daily. It is usually associated with an increase in stool volume as well as a looser consistency of the stools. Diarrhoea is said to be chronic when it has lasted for more than three weeks.

Pathophysiology of Diarrhoea

The average individual ingests about 2 litres of fluids daily. A further 7 litres of fluids is delivered into the small intestine in the form of saliva, gastric, biliary and pancreatic secretions (Figure 1). Most of this fluid is absorbed in the small intestine. Less than two litres enter the large intestine which absorbs 95% of this amount so that only about 100 ml eventually comes out in the faeces.

Diarrhoea can result from one or more of the following mechanisms (Fig. 2):

(1) Increased secretion (secretory diarrhoea)

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FIG. 1
FLUID EXCHANGE IN NORMAL GIT PER 24 HOURS

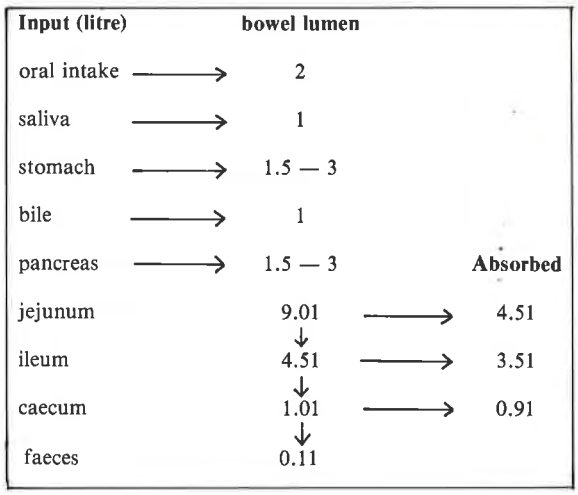


FIG 2
CLASSIFICATIONS OF DIARRHOEA ACCORDING TO MECHANISMS OF DIARRHOEA

1) **Increased osmotic load**
   - osmotic laxatives
   - lactose in lactase insufficiency
   - malabsorption

2) **Increased secretion**
   - bacterial enterotoxins as in bacterial food poisoning
   - gastrointestinal hormones as in vipomas and gastrinomas
   - chemical laxatives
   - villous adenoma
   - mucosal lesion as in coeliac disease, infalmmatory bowel disease, carcinoma
   - bile acids as in bile acid malabsorption causing colonic secretion in terminal ileal disease

3) **Decreased absorption**
   - malabsorption due to various causes

4) **Disturbed motility**
   - irritable bowel syndrome
   - Diabetes mellitus
   - post-vagotomy
   - Inflammatory bowel disease
   - laxatives

(2) Increased osmotic load (osmotic diarrhoea)
(3) Decreased absorption
(4) Abnormal motility
(5) Exudation

Two or more mechanisms may operate together to produce diarrhoea in a particular patient. Secretory diarrhoea is characterised by watery and voluminous stools without pus, blood or fat. Typically, secretory diarrhoea persists even when the patient has fasted. In contrast, osmotic diarrhoea is characterised by improvement of the diarrhoea when the patient is totally fasted. Decreased absorption and abnormal motility are probably relevant in some patients but these mechanisms have not been well studied. Disruption of mucosal integrity, for example when there is extensive ulceration, causes exudative diarrhoea. The stools may then consist largely of blood and mucus.

Diarrhoea most commonly results from disease of the colon or the small intestine. However, other parts of the gastrointestinal tract as well as extra-intestinal diseases can also be the cause (Fig. 3). It is often helpful to determine the site of disease in a particular patient. Diseases of the rectum and the distal colon frequently results in small volume diarrhoe. The stools often contain a large amount of blood and mucus and there is accompanying urgency and tenesmus. Lower abdominal pain may occur. In contrast, diseases of the small intestine often cause large volume diarrhoea. Stools volumes may be up to several litres, the stools may be watery or have features of steatorrhoea, and abdominal pain, if it occurs, is commonly central abdominal.

Another approach to diarrhoea is to look at its underlying cause. Infection, malignancy, inflammatory bowel disease, and increased osmotic load due either to excessive intake of osmotically active substances or to malabsorption are important causes of diarrhoea. Diarrhoea also frequently occurs in the irritable bowel syndrome, diabetes mellitus, and after gastric surgery. Disturbances in gastrointestinal motility may be relevant in the latter conditions.

FIG 3
CAUSES OF DIARRHOEA CATEGORISED BY ANATOMICAL SITE

1) **Gastric causes:**
surgery

2) **Pancreatic causes:**
chronic pancreatitis
Carcinoma

3) **Small intestinal causes:**
infection/food poisoning/intolerance
surgery
drug and alcoho
Crohn's disease
coeliac disease
bacterial overgrowth

4) **Colonic causes:**
infection
irritable bowel syndrome
laxatives
diverticular disease
inflammatory bowel disease
surgery
bile salt diarrhoea

5) **Extraintestinal causes:**
thyrotoxicosis
diabetes mellitus

Clinical Study

Over an eight-month period (September 1986 to May 1987), one hundred patients presented with diarrhoea to the Division of Gastroenterology, National Unviersity Hospital. Of these, eighty-eight patients had chronic diarrhoea as previously defined while 12 presented with acute diarrhoea (Tables 1 & 2). The age of these patients ranged from 17 to

TABLE 1: CAUSES OF ACUTE DIARRHOEA IN NUH (12 CASES)

	No	%
Drugs	5	42
Infection	3	25
Inflammatory bowel disease	2	17
Irritable bowel syndrome	1	8
Unknown	1	8
Total	12	100%

TABLE 2: CAUSES OF CHRONIC DIARRHOEA IN NUH (88 CAUSES)

	No	%
Irritable bowel syndrome	61	70
Bile salt diarrhoea	5	6
Carcinoma of colon	4	5
Polyps	3	3
Inflammatory bowel disease	3	3
Infective	2	2
Spurious	2	2
Others (laxative, antibiotic, diverticuli)	3	3
Unknown	5	6
Total	88	100

87 years. There were approximately equal numbers of males and females. The cause of the diarrhoea could be ascertained in all but six patients. The present series probably included more patients at the severe and chronic end of the spectrum and would hence be unrepresentative of patients seen in general practice.

Acute diarrhoea was usually due to either infection or drugs. Antibiotics were incriminated in 4 patients and mefenamic acid one. Irritable bowel syndrome was by far the commonest cause of chronic diarrhoea in this series while organic causes were more common amongst the older patients. Diverticular disease of the colon and small polyps were frequently found but they were felt usually to be incidental findings rather than the cause of the diarrhoea.

In our experience the most useful points in duration of symptoms, loss of weight, and organic causes of chronic diarrhoea were age, duration of symptoms, loss of weight, and occurrence of overt blood in the stools. Patients with functional disease were in general younger and the duration of symptoms longer when compared to patients with organic diarrhoea. Loss of weight is uncommon in functional diarrhoea and so was the occurrence of overt blood in the stools (Table 3).

TABLE 3: DISCRIMINATIVE FACTORS TO DETERMINE THE CAUSES OF CHRONIC DIARRHOEA

	Functional	Organic
Mean Age	40 yrs	51.8 yrs
Mean Duration of symptoms	3 yrs	1.2 yrs
Associated loss of weight	20% (12/61)	53% (9/17)*
Blood in stool	11% (7/61)	64% (11/17)**

* $p < 0.02$
** $p < 0.001$

Suggested Clinical Approach to the Diagnosis of Diarrhoea

(I) History

We should confirm the actual presence of diarrhoea. Spurious diarrhoea secondary to constipation must be excluded. Patients with irritable bowel syndrome usually give a long history of diarrhoea, sometimes following a gastroenteritis-like illness and the diarrhoea often alternates with constipation. Often, small amount of stools are passed frequently. If the stools have the characteristics of steatorrhoea, small intestinal or pancreatic disease has to be considered. Passage of blood and mucus usually indicates an inflammatory or neoplastic lesion in the colon. Nocturnal diarrhoea is usually a feature of organic disease but may sometimes occur in the irritable bowel syndrome. Food intolerance or hypersensitivity is frequently a feature of the irritable bowel syndrome. A history of contact with other patients with diarrhoea, travel to foreign countries and a positive family history points towards an infective cause. Marked weight loss suggests carcinoma. A detailed drugs and alcohol history is essential: antibiotics are an important cause of diarrhoea while excessive alcohol ingestion is sometimes the problem.

(II) Examination

Usually few abnormal signs are found in acute diarrhoea. Weight loss, anaemia and an abdominal mass would suggest carcinoma. Clubbing and pyoderma gangrenosum are associated with inflammatory bowel disease. Perianal inspection is essential in cases of Crohn's disease. A rectal examination is mandatory.

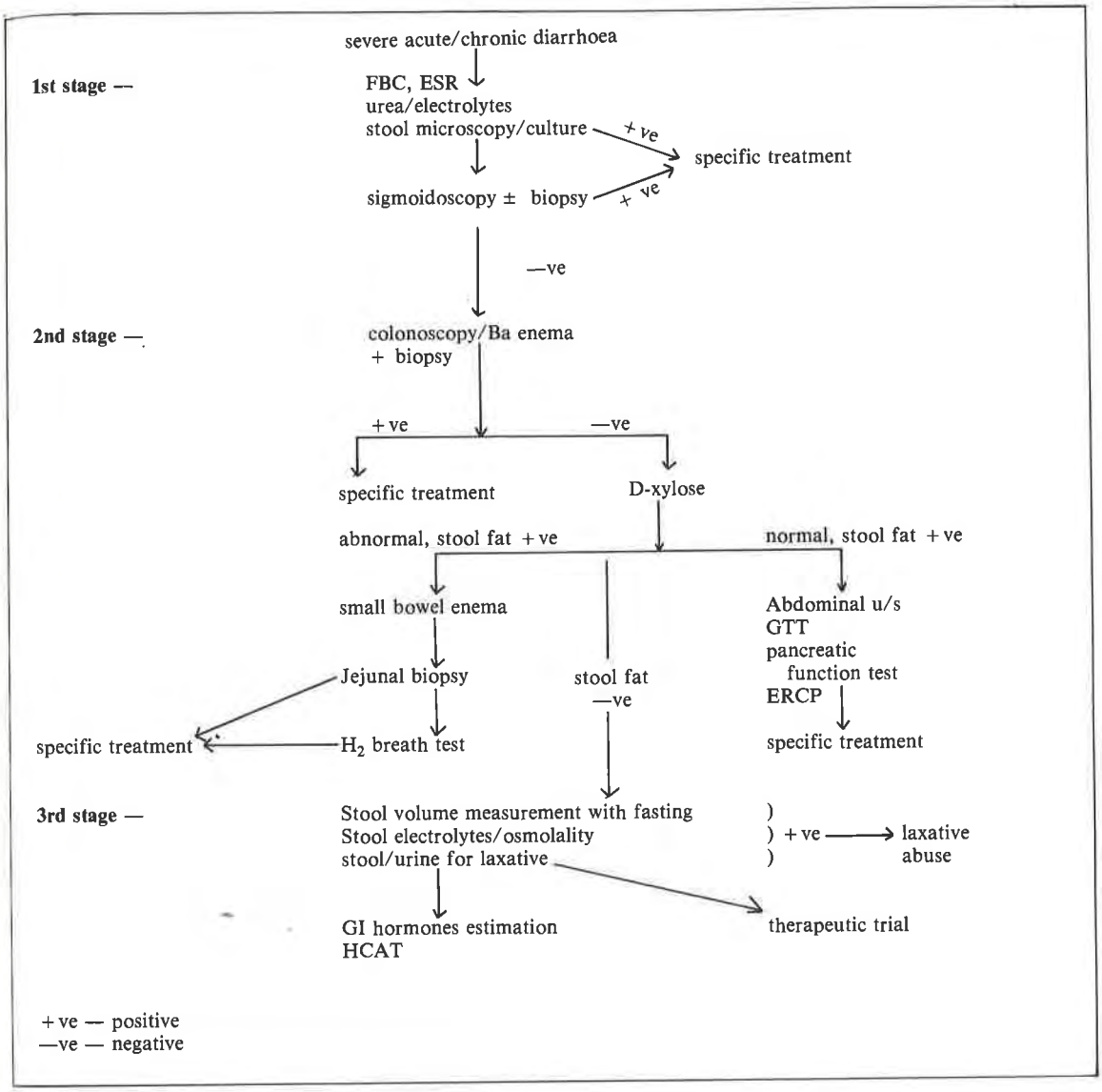
(III) Investigations

Most patients with mild to moderate acute diarrhoea need only have infection confirmed or excluded by stool microscopy and culture. However, patients with severe acute diarrhoea and chronic diarrhoea require further investigations. These can be performed in 3 stages (Fig. 4):

1st Stage

Full blood count, Erythocyte sedimentation rate, urea and electrolytes, stool microscopy and culture, as well as sigmoidoscopy and biopsy are performed. Iron deficiency anaemia with increased platelet count indicates gastrointestinal blood loss. Hypokalemia suggests a secretory diarrhoea. Sigmoidoscopy is a simple bedside procedure and should be done without preparation. It may reveal proctitis, diffuse ulceration, pseudomembranes, melanosis coli, blood, pus, mucus or focal lesions such as carcinoma

TABLE 4: INVESTIGATION OF DIARRHOEA



or adenoma. Abnormal mucosa or focal lesions must be biopsied. Sometimes a routine biopsy of normal-looking mucosa may reveal diseases like amyloidosis, Crohn's or Whipple's disease.

2nd Stage

The above investigations are adequate in the majority of cases. If the diagnosis is still in doubt after the basic workup, the following additional tests could be carried out.

A barium enema is used to diagnose or exclude large bowel disease. Colonoscopy with biopsy may reveal colitis, neoplasm or polyp even when a barium enema is normal. In suspected small bowel diseases, barium follow-through or small bowel enema may be helpful.

Qualitative estimation of stool fat can be done since a quantitative 72 hours stool fat estimation is not routinely available in Singapore. A small quantity of stool is applied on a glass slide and mixed with a few drops of glacial acetic acid and Sudan III solution, gently heated to boiling point and examined under the microscope. More than 100 fat globules/high power field indicates steatorrhoea. A D-xylose absorption test will dif-

ferentiate between pancreatic and intestinal cause since it is only abnormal in small bowel disease. If the D-xylose test is normal, abdominal ultrasound, glucose tolerance test, pancreatic function tests and endoscopic retrograde cholangio-pancreatography (ERCP) will help to establish the diagnosis of pancreatic disease and to determine its cause. Small intestinal mucosal lesions can be confirmed by jejunal biopsy while the hydrogen breath test utilising lactulose helps in the diagnosis of bacteria overgrowth.

3rd Stage

These tests are reserved for a small number of patients with chronic or intractable diarrhoea whose cause remains unknown despite the above tests. Daily stool volume and electrolytes measurements before and during fasting should be done. Testings for laxatives in urine or stool may reveal the diagnosis of laxative abuse. A decreased uptake of radiolabelled bile salt (Selenium Homocholic acid Taurine-HCAT) indicates the presence of bile salt malabsorption. Rare neuro-endocrine tumours can only be diagnosed by gastrointestinal hormone estimations (Vasoactive intestinal polypeptide, Gastrin, Prostaglandins etc). Therapeutic trials of anti-tuberculous agents, antibiotics, pancreatic enzymes and cholestyramine could be useful depending on the degree of clinical suspicion if no definitive diagnosis is made.

Problems in Diagnosis

The majority of patients with acute diarrhoea will settle spontaneously even if a precise diagnosis cannot be made. With chronic diarrhoea, specific investigations are required to diagnose or exclude inflammatory and neoplastic conditions. Most patients with chronic diarrhoea will in the end be labelled as suffering from irritable bowel syndrome. However, the occasional patient will be encountered in whom no satisfactory diagnosis can be made.

JAUNDICE IN CLINICAL PRACTICE A DIAGNOSTIC APPROACH

R Guan, *MB, MRCP*

SYNOPSIS

80 consecutive cases of jaundice admitted to the Singapore General Hospital during a 6 month period were analysed. Hepatitis, obstructive jaundice and primary liver cancer were common causes of jaundice. Simple blood tests including routine liver function tests were usually adequate for the diagnosis of hepatocellular jaundice while more sophisticated investigations like abdominal ultrasound, computerised tomography and endoscopic cholangiography were needed to determine the cause of cholestasis. A clinical approach to the diagnosis of jaundice was discussed and recent advances in the management of the more common causes were mentioned.

INTRODUCTION

Jaundice is a common medical problem and is due to excessive levels of bilirubin (both conjugated and non-conjugated) in the blood. Accumulation of bilirubin can result from excessive haemolysis, from liver damage due to virus infection, drugs and other causes, or from obstruction to bile flow due to stones or malignancy.

A combination of the above factors is often present. The commonest causes of jaundice are hepatitis and obstruction to the biliary system. These two diseases are easily identified by simple laboratory investigations. Ancillary investigations like ultrasound, computerised tomography and endoscopic retrograde cholangiography help in the more difficult cases.

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Patients and Methods

The records of consecutive patients admitted for jaundice (total bilirubin 1.6 g/dl) to the adult medical and surgical wards of the Singapore General Hospital during the 6 month period from December 86 to May 87 were reviewed. The different causes of this condition were determined and the different investigations done towards their diagnoses were noted. The modes of presentation of jaundice due to biliary tract obstruction were also determined.

Results

There were altogether 70 patients (51 males and 19 females) admitted for jaundice during the above period. Their ages ranged from 10 years to 91 years (mean: 55 years). One 2-year old female with biliary atresia was admitted to an adult surgical ward and was included in the analysis.

CAUSES OF JAUNDICE

Hepatitis accounted for 29% of jaundice admitted during the study period (Table 1). Acute viral hepatitis was responsible for 75% of cases of hepatitis. 40% of acute viral hepatitis were due to hepatitis A, 27% due to hepatitis B and 33% due to hepatitis non-A non-B (NANB). Acute hepatitis A and B were seen in the younger age group while acute NANB hepatitis seem to affect older patients. 15% of hepatitis were drug induced and this was found in the elderly (Table 2). Rifampicin was the drug implicated in all our patients. Alcohol was responsible for 5% hepatitis.

In patients above 60 years of age, jaundice

TABLE 1: ADMISSION TO SGH FOR JAUNDICE DECEMBER 1986 — MAY 1987 70 CASES

Types of Jaundice	No of cases	(%)	M: F	Mean Age (Range)
Hepatitis	20	(29)	15: 5	38 (10-72)
Hepatocellular Carcinoma	20	(29)	17: 3	62 (37-80)
Obstructive Jaundice	16	(23)	10: 6	65 (2-86)
Liver Secondaries	5	(7)	5: 0	57 (37-68)
Cirrhosis	4	(6)	2: 2	77 (63-91)
Haemolytic	3	(4)	1: 2	45 (29-62)
Gilbert's Syndrome	1	(1)	1: 0	27
Septicaemia	1	(1)	0: 1	77

TABLE 2: ADMISSION TO SGH FOR HEPATITIS DECEMBER 1986 — MAY 1987 20 CASES

Types of Hepatitis	No of cases	(%)	M: F	Mean Age (Range)
Hepatitis A	6	(30)	4: 2	24 (10-36)
Hepatitis B	4	(20)	3: 1	26 (25-30)
Non A Non B	5	(25)	4: 1	52 (16-72)
Drug Induced	3	(15)	2: 1	49 (36-65)
Alcoholic	1	(5)	1: 0	49
Others	1	(5)	1: 0	50

was either due to hepatocellular carcinoma (29%) or extrahepatic biliary obstruction (23%). Of the 16 cases of obstructive jaundice 10 (63%) were due to stones and 5 (31%) due to malignancy (Table 3). Pain and septicaemia were the presenting symptoms in half of the patients in this group (7 of 10 with stones and 2 of 5 with malignancies). Endoscopic retrograde cholangio-pancreatography (ERCP) was performed in 7 of 10 patients with choledocholithiasis. Endoscopic removal of stones were effected in 3 patients while 2 patients had their stones removed surgically. Four others had spontaneous expulsion of their common bile duct (CBD) stones. One patient refused further investigations and was discharged. He came back later with septicaemia and died.

Liver secondaries were responsible for 7% of jaundice. Liver infiltration and biliary obstruction were probably the mechanisms involved.

Jaundice was due to cirrhosis in 4 (6%) patients. Chronic hepatitis B virus infection was responsible in 2 patients and alcohol in the other 2.

Excessive haemolysis is an uncommon (4%) but important cause of 'acholuric' (absence of bile in the urine) jaundice in adults. The causes of haemolysis were leptospirosis, drugs and systemic lupus erythromatosis.

One patient had Gilbert's disease. Septicaemia secondary to bronchiectasis was the cause of jaundice in one other patient. Liver damage and haemolysis were amongst the mechanisms involved.

INVESTIGATIONS

Patients with acute hepatitis had normal haemoglobin levels (Table 4). Low haemoglobin levels were seen in haemolysis (mean: 7.2 g/dl), cirrhosis (mean: 8.75 g/dl) and liver secondaries (mean: 8.9 g/dl). Recticulocyte count was increased in haemolysis (mean: 9%)

A raised bilirubin with normal liver function tests was present in the patient with Gilbert's disease. The highest bilirubin levels were seen in patients with hepatitis and biliary obstruction although this was not reflected in the mean values calculated for our patients. Serum alkaline phosphatase (SAP) was raised in patients with hepatocellular carcinoma (mean: 445 IU/L), biliary obstruction (mean: 483 IU/L), and liver secondaries (mean: 476 IU/L). Very high levels of serum glutamate

TABLE 3: ADMISSION TO SGH FOR OBSTRUCTIVE JAUNDICE DECEMBER 1986 — MAY 1987 16 CASES

Cause of Obstruction	No	(%)	M: F	Mean Age (range)	Septicaemia (%)	Pancreatitis (%)	ERCP
Choledocholithiasis	10	(63)	7: 3	66 (43-86)	7 (43)	2 (13)	7
Malignancy	5	(31)	3: 2	75 (58-82)	2 (13)	0	0
Others	1	(6)	0: 1	2	0	0	0

TABLE 4: 70 CASES OF JAUNDICE ADMITTED TO SGH DECEMBER 1986 — MAY 1987 (CLINICAL & LABORATORY FINDINGS)

Types	No of Pat	Liver	Spleen	Haemoglobin g/dl Mean (Range)	Retic (%)	Albumen g/dl Mean (Range)	T. Bil mg/dl Mean (Range)	SAP IU/L Mean (Range)	SGPT IU/L Mean (Range)	SGPT IU/L Mean (Range)	HBs/Ag +ve	AFP +ve
Hepatitis	20	10	0	13.8 (11.2-17.6)	—	3.6 (2.4-4.5)	7.9 (1.6-26.3)	169 (82-274)	777.2 (19-3438)	469.7 (34-2893)	4	—
Hepatocellular Carcinoma	20	18	1	11.5 (9.0-14.8)	—	2.9 (2.0-3.8)	6.9 (1.6-21.3)	444.5 (105-1265)	188.2 (48-1085)	380 (58-2354)	14	12
Obstructive Jaundice	16	11	1	11.5 (5.2-16.1)	—	3.18 (2.5-4.2)	7.7 (1.7-25.7)	483 (114-1832)	201.5 (49-649)	153.2 (40-330)	2	0
Liver Secondaries	5	4	0	8.9 (9.9-10.7)	—	2.42 (2.2-2.6)	14.7 (3.2-21.9)	476.5 (208-955)	112 (75-177)	290 (41-2354)	0	0
Cirrhosis	4	0	0	8.75 (6.9-10.6)	—	2.45 (2.4-2.5)	6.7 (6.0- 7.4)	134 (124-145)	92 (82-102)	180 (168-193)	1	0
Haemolytic Jaundice	3	2	1	7.2 (4.3- 9.0)	9 (7-10)	3.1 (2.1-3.7)	6.96 (1.9-15.2)	153.6 (58-272)	27.3 (18-45)	28.3 (23-37)	1	—
Gilbert's Syndrome	1	0	0	15.7	—	4.5	3.6	83	19	34	0	—
Septicaemia	1	0	0	14.7		4	1.9	134	58	117	0	0

pyruvate transaminase (SGPT) and serum glutamate oxaloacetate transaminase (SGOT) (mean SGPT level: 777 IU/L, mean SGOT level: 469 IU/L) were seen in hepatitis. Low albumin levels were seen in cirrhosis (mean: 2.4 gm/dl), hepatocellular carcinoma (mean: 2.9 gm/dl) and liver secondaries (mean: 2.42 gm/dl).

Patients with hepatocellular carcinoma 12/17 (71%) had high levels of alpha-foetoproteins while 14/16 (88%) had hepatitis B surface antigen (HBsAg) in their serum.

Blood investigations were adequate for the diagnosis of hepatitis and haemolysis. Abdominal ultrasound was used in the diagnosis of 13/20 (65%) patients with hepatocellular carcinoma and 14/16 (88%) patients with extrahepatic biliary obstruction. Computerised tomography (CT) was used in 13/20 (65%) patients with hepatocellular carcinoma and in 4/16 (25%) patients with cholestasis. 6/20 (30%) patients with hepatocellular carcinoma had hepatic angiography (Table 5). ERCP was used in the diagnosis of the cause of the obstruction in 7/16 (44%) patients.

DISCUSSION

Hepatitis, hepatocellular carcinoma and extrahepatic biliary obstruction are common causes of jaundice locally. Viral hepatitis is by far the commonest cause of hepatitis and the incidence was probably higher than observed

TABLE 5: 70 CASES OF JAUNDICE ADMITTED TO SGH DECEMBER 1986 — MAY 1987 (RADIOLOGICAL INVESTIGATIONS)

Types	No of Patients	U/S	CAT	Hepatic ERCP	Angiography
Hepatocellular Carcinoma	20	13	13	ND	6
Obstructive Jaundice	16	14	4	7	ND
Liver Secondaries	5	2	3	ND	ND
Cirrhosis	4	3	ND	ND	ND
Hepatitis	20	ND	ND	ND	ND
Haemolytic Jaundice	3	ND	ND	ND	ND
Gilbert's Syndrome	1	ND	ND	ND	ND
Septicaemia	1	ND	ND	ND	ND
Total	70	32	20	7	6

as most patients were not admitted. Hepatitis A (infective hepatitis) is seasonal in incidence and has a short incubation period of 4 to 6 weeks. It is usually spread through the oro-faecal route and does not become chronic. Hepatitis B (serum hepatitis) has a longer incubation period (6-8 weeks) and can be spread by both parenteral and non parenteral route. Hepatitis B carries a real risk of chronic infection and cirrhosis. Non-A non-B (NANB) hepatitis is the commonest form of post transfusion hepatitis in Singapore and other countries where donor blood is screened routinely for HBsAg. This type of hepatitis has been seen in epidemics in Africa and Asia and the epideomology resembles that of hepatitis A, although the two viruses are serologically unrelated. Some forms of NANB hepatitis are waterborne. 33% of viral hepatitis seen during the study period were due to hepatitis NANB. This was higher than the 16% reported by Chan and his colleagues in 1982.[1] Viral hepatitis can also occur with non-specific viral infections and when this happens, the liver transaminases are usually only mildly raised. Of note here is glandular fever due to the Epstein Barr virus. Drug induced hepatitis is relatively common among the elderly and this is very often overlooked.

Jaundice is a late sign of hepatocellular carcinoma. Non-specific symptoms like loss of weight and appetite usually heralds the disease and the diagnosis is usually made on abdominal examinatioin. The figure of 29% for hepatocellular carcinoma is biased as the University department of Medicine was involved in the treatment of patients with hepatocellular carcinoma during the study period.

Jaundice is a cardinal feature of extra-hepatic biliary obstruction. This is commonly due to an impacted stone or a carcinoma of the head of the pancreas (Table 3). Pain and septicaemia were the presenting symptoms in 9/16 (56%) of patients with extra hepatic biliary obstruction.

Very mild jaundice is usually seen in Gilbert's disease. This is a relatively common but benign condition affecting about 3-6% of the population.[2] The clinical picture is that of recurrent mild jaundice especially during an intercurrent infection. The liver function tests are completely normal except for a mildly raised bilirubin (less than 3 mg/dl). Bile is absent in the urine and there is no evidence of haemolysis. This condition is easy to diagnose and very satisfactory to manage as all the patient needs would be reassurance. A rise in bilirubin after a two day fast or nicotinic acid administration (the other parameters of the liver function tests remaining normal) confirms the diagnosis in doubtful cases.

Jaundice is rarely an emergency except when a patient is having associated septicaemia. A careful history, detailed clinical examination and some simple blood tests are usually adequate to diagnose a majority of patients. More sophisticated investigations are required in about 20% cases.

History

Patients with hepatitis B often but not always give a family history of jaundice and hepatoma.[3] A family history of jaundice or liver disfunction would otherwise suggest a hereditary disorder like Wilson's disease, thalessemia OR red cell enzyme defects. Diagnosis in these patients are usually made early on in life.

A previous history of gall bladder surgery should alert the clinician to the possibility of retained stones.

Hepatitis A infection is suggested by a history of shellfish ingestion. Contact history may be difficult to obtain. Forgotten needle stick injury is especially important amongst medical personnel. NANB hepatitis should be suspected if jaundice follows recent blood transfusion. Workers in plastic factories (where exposure to vinyl chloride monomers can be high) have an increased risk of liver damage.

The presence of prodromal symptoms of fever, nausea and anorexia followed by jaundice when the fever subsides strongly suggest viral hepatitis.

The pattern of onset of the jaundice can also give diagnostic clues and avoid superfluous investigations. Haemolytic jaundice is usually abrupt. Progressive jaundice associated with fever, chills and rigors (Charcot's triad) would suggest cholangitis secondary to stone obstruction. Associated pruritis

and xanthelasmia would point to primary biliary cirrhosis and less often a pancreatic carcinoma. Associated diarrhoea especially with foul smelling stools suggests a pancreatic pathology. Substantial weight loss is usually associated with malignancy.

A detailed drug history is sometimes impossible locally especially when medicinal herbs have been ingested. Elderly patients are usually on multiple drug therapy and it is helpful to remember the common drugs that can cause hepatitis either by a direct toxic effect or through a hypersensitivity reaction (Table 6). Jaundice usually occurs within 6 weeks of the start of therapy. In this context, it is useful to remember that inhalation of naphthelene vapour and ingestion of broad beans can cause jaundice in Chinese male patients with glucose-6-phosphatase (G-6-P) deficiency in their red blood cells. Viral hepatitis can also precipitate destruction of G-6-P deficient cells and cause acute haemolysis.[4]

TABLE 6: SOME OF THE COMMON DRUGS THAT CAUSE LIVER DAMAGE

Direct toxic effects
Paracetomol overdosage
Ferrous sulphate overdosage
Tetracycline
Carbon tetrachloride
Anabolic steroids (cholestasis)
Azathioprime
Hypersensitivity reaction
Methyl dopa (can also cause haemolysis)
Anti-tuberculous drugs
Oxyphenisatin (in some laxatives)
Phenylbutazone
Nitrofurantoin
Chlorpromazine
Danazol

Recording alcoholic consumption during the previous seven days is preferable to asking the patient to estimate for an average day.[5] As a rough measure, a 0.25 litre glass of beer, a glass of wine and a measure of spirit all contain the equivalent of 10 grams of alcohol. The risk of liver disease increases if the daily consumption is more than 50 grams in a male and more than 30 grams in a female.[6]

Examination

The diagnosis may be apparent when the patient walks into the surgery. A young person is likely to have hepatitis or haemolysis. Obstructive jaundice, drug jaundice and malignancy are usually seen in the older patient. A grossly discoloured patient who is otherwise well has hepatitis especially when a prodromal history is present. A cachexic old man probably has malignancy.

Associated pallor indicates either a haemolytic or malignant process or cirrhosis of the liver with gastrointestinal bleeding. The presence of anaemia and supraclavicular lymphadenopathy strongly suggests a pancreatic carcinoma.

Spider naevi, male gynaecomastia, testicular atrophy, ascites and other signs of liver insufficiency indicates a chronic process and usually but not always signify a relatively bad prognosis. Chronic liver disease is very often due to HBV infection if an alcoholic history is absent. Less common causes include Wilson's disease and lupoid hepatitis.[7] Hepatosplenomegaly is often present. Liver insufficiency is a rather late presentation of hepatocellular carcinoma.[8]

The characteristics of the liver enlargement will help plan further investigations — alphafoetoprotein estimation and liver ultrasonography would be more useful than a biochemical LFT if an irregularly enlarged, hard liver with nodular surface is present. An enlarged, tender liver with an auscultatory bruit and perhaps a friction rub usually indicates underlying malignancy. The very tender liver of liver abscess is usually associated with toxicity and a mild jaundice.

Couvoisier's law has stood the test of time. A jaundiced patient with a palpable (distended) gall bladder and enlarged liver probably has a carcinoma of the head of the pancreas or an impacted stone at the lower end of the common bile duct or in the hartman's pouch. Associated fever and tenderness of the right hypochondrium signifies biliary tract sepsis.

The presence of other abdominal masses would usually result in a hospital/specialist referral as liver secondaries would be implicated. A rectal examination might prevent a delay in diagnosing liver secondaries from a rectal carcinoma.

Blood Investigations

Haematology and biochemical liver func-

tion tests usually help to differentiate hepatocellular from obstructive jaundice. Sometimes, an 'obstructive' pattern with raised SAP levels is seen in patients with viral hepatitis.[9] These patients generally look well. Patients with haemolytic anaemias can also show an 'obstructive' and sometimes a mild 'hepatitis' picture, especially if they are seen a few days after a haemolytic episode. At least one patient with auto-immune haemolytic anaemia has presented in this way. Although low albumin levels are indicative of chronic liver disease, many patients with cirrhosis have normal albumin levels.[10] The prothrombin time provides a quick helpful guide in the assessment of the severity of liver involvement when hepatocellular jaundice is suspected.

Should acute viral hepatitis be suspected IgM antibodies to hepatitis A virus (anti-HAV IgM) and IgM antibodies to hepatitis B core antigen (anti-HBc IgM) are useful tests to differentiate the two major types. HBsAg, though usually done because of its relative ease and low cost is not a helpful test. It's presence does not necessarily mean that the hepatitis event is a result of acute hepatitis B infection locally. The chronic carriage rate for hepatitis B in Singapore is 6-10%.[11] Up to 20% of patients with chronic hepatitis B infection undergo conversion hepatitis reactions annually when they lose their 'e' antigen and develop antibodies to the 'e' antigen. Reactivation hepatitis reaction occur in about 8% of patients with chronic hepatitis B infection annually. During reactivation the 'e' antigen reappear in the serum indicating resurgence of viral replication.[12] A hepatitis reaction in a known hepatitis B carrier can also be due to superinfection by the A, NANB or delta viruses.

The presence of HBsAg in a patient with an enlarged, hard liver should make a diagnosis of hepatocellular carcinoma suspect as 88% of patients with this condition had this antigen in their serum. This finding was in concurrence with a report from Taiwan which stated that 80% of liver cancer patients were positive for HBsAg.[13] High levels of alpha-foetoprotein (AFP) are also diagnostic of liver cancer. Moderate increase in AFP levels, however, can occur in chronic hepatitis especially when there is excessive regeneration of liver cells following destruction.

Specialised Investigations

Further investigations are best done in a hospital as specialist interpretation would be needed. Table 7 shows some of the more useful laboratory investigations in the diagnosis of hepatocellular jaundice.

TABLE 7: FURTHER INVESTIGATIONS HELPFUL IN ESTABLISHING THE CAUSE OF HEPATOCELLULAR JAUNDICE

Blood	Urine
Iron and iron binding capacity	Electrolytes
Ferritin	24 hour copper
Copper & ceruloplasmin	
Auto antibody screen	
Protein electrophoresis	
Viral antibodies (e.g. cytomegalovirus epstein barr virus)	
Cholesterol & triglycerides	

A hepatobiliary ultrasound examination is cheap and convenient and in the hands of a skilled radiologist, cirrhosis, tumours as well as stones can be diagnosed with confidence. Its real usefulness however is in the diagnosis of cholestasis.

Very occasionally, an abdominal ultrasound examination would fail to detect the cause of a dilated biliary system when the pathology is in the distal common bile duct where duodenal gas can prevent a good view. In such cases more invasive methods are necessary to visualise the biliary tree. Percutaneous transhepatic cholangiogram (PTC) and endoscopic retrograde cholangiogram (ERC) have largely superceded intravenous cholangiogram. A PTC examination is preferable for lesions of the proximal biliary system whereas an ERC is more sensitive for distal lesions [Figs 1 (a) and (b)].

More sophisticated and increasingly expensive investigations like CT scan and arteriography have largely replaced isotope scans in the diagnosis of tumours and other solid hepatic lesions.

Needle liver biopsy is useful in the patient who presents with diagnostic difficulty in spite of having all the above investigations or if a histological diagnosis is needed. Among the many techniques available, the Menghini aspiration technique is the safest.

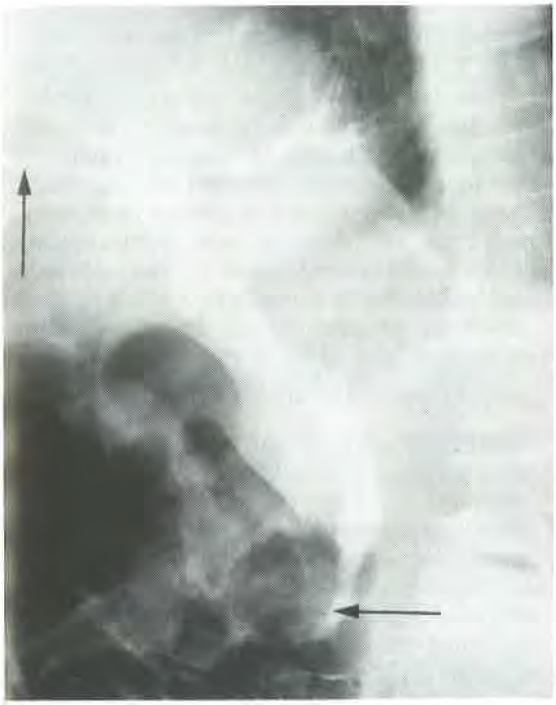


Figure 1(a): Percutaneous cholangiogram in a patient with obstructive jaundice: vertical arrow shows the position of cholangiogram tube and horizontal arrow shows site of obstruction with irregular 'rat tail' appearance suggestive of malignancy.

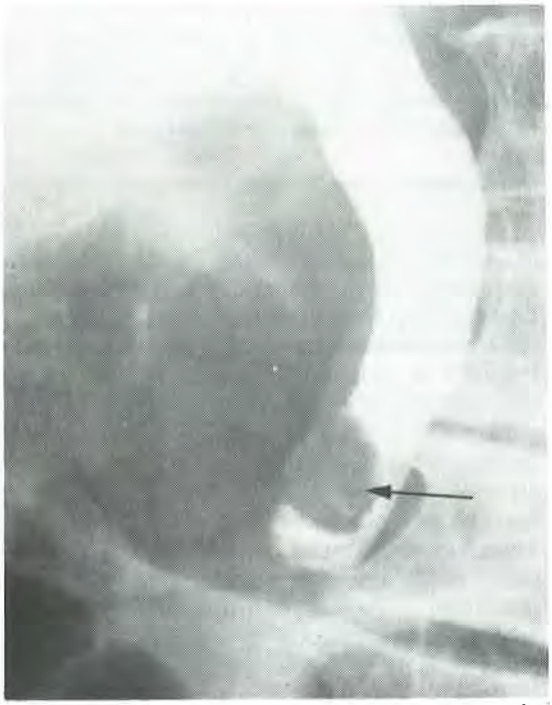
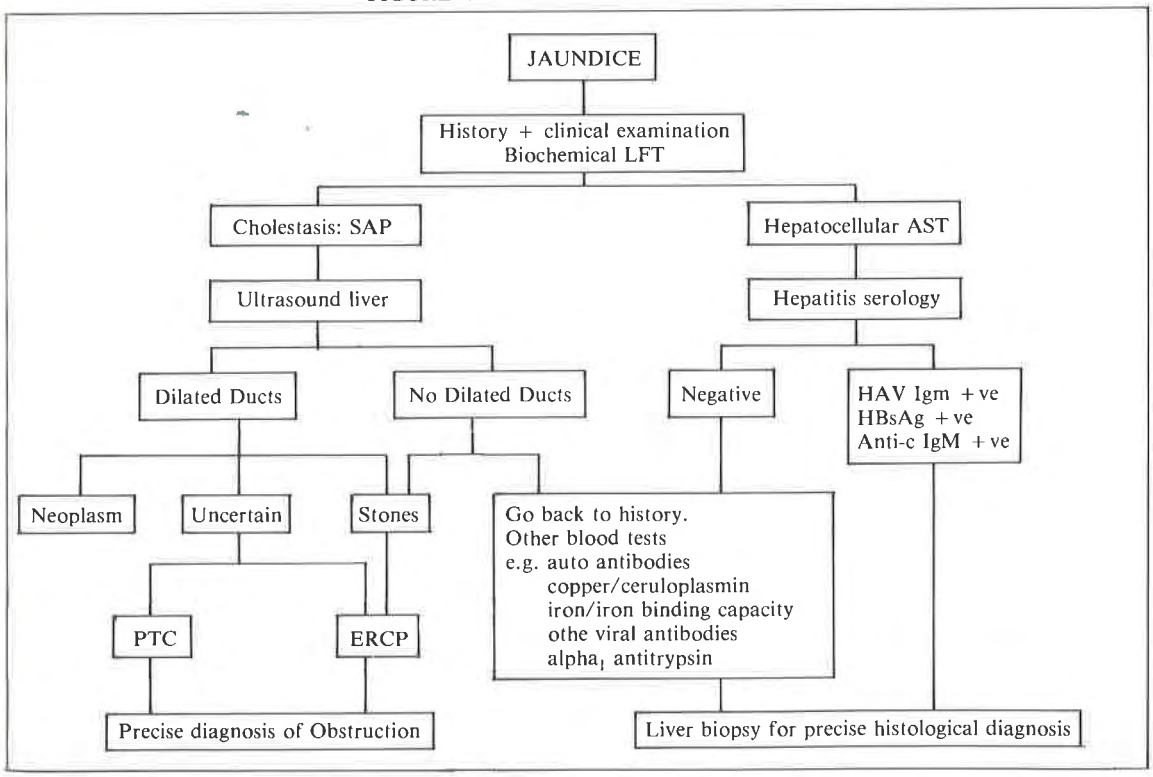


Figure 1(b): Endoscopic retrograde cholangiogram of same patient showing actual cause of obstruction (arrowed) at lower end of common bile duct to be due to a stone. (Picture taken after sphincterotomy and removal of duodenoscope).

FIGURE 2: INVESTIGATION OF JAUNDICE



A flow chart to help in the investigations of a jaundiced patient is given in Figure 2. With all the above diagnostic armamentarium, exploratory laparatomy is seldom necessary in the diagnosis of jaundice.

Management

Most cases of viral hepatitis can be treated at home and no dietary restrictions are necessary.[14] The majority of patients recover uneventfully. Serial liver function tests (LFTs) are recommended to monitor progress and if the LFT is still abnormal after 6 months, a liver biopsy is recommended. Isolation of patients with viral hepatitis is not only unnecessary but is costly. Family members are already exposed by the time the patient presents and advice like 'no sex please' is unnecessary. Patients with Type A hepatitis cease to be infectious soon after becoming jaundiced.[15] Type B hepatitis patients tend to carry HBsAg for 6 to 12 weeks but are probably infectious only in the first few weeks of clinical illness when 'e' is present. Although saliva and semen have been shown to contain HBsAg, blood is the most consistently efficient vehicle for Hepatitis B transmission. Unvaccinated victims of hepatitis B needlestick injuries should receive gamma-globulin injections (H-BIG; Abbot Laboratories — 0.06 mls/kg body weight) within 12 hours of innoculation. A course of vaccination should also be started straight away.[16]

Reassurance is the only treatment needed in Gilbert's syndrome. Phenobarbitone can be tried to reduce serum bilirubin levels by inducing glucoronyl transferase.[17] 'Sufferers' should be warned that jaundice can follow an intercurrent infection.

The prognosis in alcoholic liver diseases is greatly improved if the patient can be persuaded to abstain from alcohol. Very often treatment is disappointing as the patient usually goes back to his honey of solace. Sedation should be avoided and diuretics can be used if ascites is present.

No specific treatment is indicated for drug jaundice but the offending drug should be withdrawn as soon as possible. Jaundice usually takes a while to subside.

Autoimmune (lupoid) chronic hepatitis usually responds well to moderate doses of prednisolone (20 mg/day). The rare patient with Wilson's disease can be treated with copper chelating agents like penicillamine. Zinc has been found to be as effective recently.[18] The mainstay of the treatment of haemochromatosis is removal of excess iron by venesection. There is at present no effective treatment for patients with HBV chronic hepatitis. Antiviral agents like adenine-arabinoside[19] and the interferons[20] have been shown to be effective in suppressing viral replication and have been used with some success in patients in this phase of infection.

Although surgery still play a major role in the curative and palliative management of resectable hepatocellular carcinoma, ampullary carcinoma and stones in the common bile duct, the endoscopists and radiologists are playing an increasing role in palliation. Endoscopic removal of common bile duct stones have spared many surgeons the nightmare of post operative complications in high risk patients. Emergency nasobiliary drainage of the biliary tree in cases of cholangitis consequent upon cholelithiasis has also helped prevent emergency surgery in acutely ill patients.[21] Endoscopic stenting as well as percutaneous drainage of the dilated biliary system has improved the quality of life in many patients with inoperable pancreatic carcinoma.

ACKNOWLEDGEMENTS

The author would like to thank Drs Ong Yong Yau, Ng Han Seong, R Nambiar and Abu Rauff for allowing their cases to be studied, Mr Fung for Figs 1 and Miss Ang Ee Lin for stenographical help.

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HOME STUDY SECTION

THE TREATMENT OF HYPERURICAEMIA AND GOUT

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Hyperuricaemia is said to be present when the serum uric acid level is above 7.0 mg% in the adult male and 6.0 mg% in the adult female. It is not a disease in itself but is often an indicator of important underlying conditions [table below]. Before considering drugs to lower serum uric acid level, possible aetiological factors should be sought and a plan for their correction instituted.

Factors which contribute to hyperuricaemia:

(1) Increased production of urate
a. Genetic
b. Aquired
myeloproliferative disorders — polychemotherapy, radiotherapy, psoriasis
(2) Decreased urate excretion
a. Genetic
b. Acquired
* acute or chronic renal disease
* hypertension
* lacic acidosis, ketoacidosis
* drugs — thiazides, low dose aspirin, pyrazinamide
* hypercalcaemia — hyperparathyroidism, sarcoidosis
* low urine volume

It is important to appreciate that hyperuricaemia can be associated with several of the risk factors for cardiovascular disease [hypertension, obesity, hyperlipidaemia] and it might be necessary to address these. Correction of these risk factors which could demand a change in lifestyle, will often correct hyperuricaemia.

Asymptomatic hyperuricaemia requires no treatment provided the blood pressure, weight and renal function remain normal. Treatment is necessary when the patient.

* becomes symptomatic
* has a strong family history of gout, nephrolithiasis or renal failure
* is excreting large quantities of urates in the urine [greater than 1100 mg in 24 hours]

Gout is the result of the accumulation of urate in the body and its deposition in joint tissues where the crystals provoke an acute inflammatory arthritis. Other manifestations include tophi [the bulk deposit of urates in tissues] and renal stones.

Episodes of gout can be spontaneous or provoked by stress such as illness, injury, surgery, severe exercise or dieting, or by alcohol, rich foods or drugs such as thiazides. It may also follow drug allergy or initiation of Vitamin B12 therapy in pernicious anaemia.

The goal in the treatment of acute gout is the control of acute inflammation with subsequent relief of pain. Lowering of the serum uric acid level should be avoided in the presence of acute gouty arthritis. Drugs which lower the serum urate concentration usually have no anti-inflammatory action and could aggravate and prolong the acute episode.

The first line of treatment is a non-steroidal anti-inflammatory drug [NSAID]. Indomethacin is usually preferred — 50 mg orally may be given 3 to 4 times a day until the acute attack subsides and then tailed over one or two weeks. Any other NSAID may be used, but phenylbutazone, although highly effective, should not be used because it has an unacceptably high incidence of serious and often fatal blood dyserasias.

Colchicine is also effective in the treatment of acute gout and as prophylaxis against further attacks, but it has been largely superseded because it causes dose-dependent vomiting and diarrhoea. Colchicine may be given orally

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as 1 mg stat and 0.5 mg every one or two hours until the attack subsides or diarrhoea occurs. It is probably less effective than NSAIDs but may have advantages if gastric irritation or sodium retention is a problem.

The timing of treatment may be more important than the drug used. Doses much smaller than those usually recommended may be effective if treatment is started early; established attacks may require high doses and for a longer duration.

Most attacks respond quickly. The most common reasons for an acute episode not responding to treatment is the concomitant starting of allopurinol which can turn a mild attack into a severe one affecting a number of joints. But other drugs including alcohol, low dose asprin, uricosurics and diuretics may also be implicated.

In rare instances where oral therapy is not possible or contraindicated indomethacin suppository or intramuscular hydocortisone or corticotrophin gel injections or intravenuous colchicine may be used.

Long-term control of hyperuricaemia is indicated in the following circumstances:

* recurrent attacks of acute gout
* the development of tophi
* uric acid calculi
* gouty erosions on X-ray
* persistent severe asymptomatic hyperuricaemia [serum uric acid level more than 10.0 mg%].

All urate lowering drugs have a tendency to provoke acute gout and should

* not be started during or soon after an acute attack
* be started at a low dose and
* be given with an appropriate NSAID, or colchicine, for the first 3 to 4 months.

Drugs used to lower the serum uric acid level are either inhibitors of xanthine oxidase or act by inhibiting the reabsorption of urates within the renal tubules [uricosuric agents].

Allopurinol, a xanthine oxidase inhibitor, reduces the formation of uric acid. It is well absorbed in the upper gastro-intestinal tract and although its half-life is only 2 to 3 hours, its active metabolite, oxipurinol, has a long half-life of 18 to 30 hours. This makes once daily therapy with allopurinol feasible and assists compliance. The usual dose is 300mg, but this should be reduced in patients with significant renal impairment because both allopurinol and oxipurinol are excreted by the kidneys.

The common side effect of allopurinol are gastrointestinal intolerance and an erythymatous maculo-papular pruritic skin rash, which usually subsides on withdrawing the drug. Less common side effects include vasculitis, toxic epidermal necrolysis, alopecia, hepatitis and bone-marrow depression.

The purine analogues azathioprine and mercaptopurine depend on xanthine oxidase for their metabolism, and their doses should be reduced in patients treated concomitantly with allopurinol. Allopurinol also potentiates the pharmacological effects of cyclophosphamide and warfarin.

Uricosuric agents — probenecid and sulphinpyrazone — are prescribed less often than allopurinol but may be useful in some cases of allopurinol sensitivity. They are generally less effective than allopurinol and are not effective in renal failure or in patients who overproduce uric acid and they are contraindicated in patients with renal stones. Uricosuric agents have the potential to cause uric acid crystalluria and it is therefore important that patients be instructed to maintain a high fluid intake to reduce nephrolithiasis.

These drugs are generally well tolerated, but gastrointestinal intolerance is often a problem. Skin rashes occasionally occur with both drugs and bone marrow suppression has been reported with sulphinpyrazone. The uricosuric effect of both probenecid and sulphinpyrazone is inhibited by concurrent aspirin or thiazide therapy.

Chronic tophaceous gout may be prevented or reduced by lowering the serum uric acid level to normal. Allopurinol is the drug of choice. Tophi may begin to resolve after several weeks of therapy. Again an appropriate NSAID, or colchicine must be given currently because mobilisation of urate deposits may trigger an acute attack of gout. Concurrent therapy of allopurinol with pro-

benecid is sometimes used in some patients with chronic tophaceous gout to hasten the regression of urate deposits and in resistant cases.

Gout should be excluded in all cases of renal colic. Nephrolithaiasis with hyperuricaemia may pose some problems. Is gout the primary cause of the renal stone or is hyperuricaemia secondary to renal impairment caused by the damaged kidney? In either case treatment with allopurinol is required. Reduction of dose is essential in the setting of renal insufficiency because of the risk of a fulminant toxicity syndrome (rash, fever, hepatitis, eosinophilia and worsening renal failure).

Summary

The primary physician should be able to promptly diagnose and treat acute gout, prevent recurrences and minimise the risks of complications such as chronic gouty arthritis and nephrolithiasis.

Although treatment of gout depends on the application of biochemical and pharmacological principles, the importance of reduction of excessive weight (without fasting), avoidance of excessive alcohol intake and maintenance of good hydration need to be emphasized. Severe dietary restrictions are unnecessary; only 10% of circulating purines in the body is derived from dietary resources.

Many treatment failures are the result of failure to follow therapeutic regimens; instruction and education of patients is often the most important factor in the management of gout and hyperuricaemia. The reluctance of otherwise healthy people to take long-term medication makes their management interesting and challenging.

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MULTIPLE CHOICE QUESTIONS

1. Recognised precipitating causes of acute gouty arthritis include
   - A trauma
   - B initiation of allopurinol therapy
   - C excess alcohol intake
   - D fasting
   - E following drug allergy

2. Drugs which might be indicated in an acute attack of gout include
   - A indomethacin
   - B probenecid
   - C colchicine
   - D sulphinpyrazone
   - E hydrocortisone

3. An acute attack of gout
   - A responds well to a combination of aspirin and probenecid
   - B is best treated with allopurinol
   - C may be precipitated by diuretic treatment
   - D typically responds within 48 hours to treatment with indomethacin
   - E is always associated with hyperuricaemia

4. In the long term managment of gout
   - A the drug of choice is allopurinol
   - B the patient should be advised to abstain from alcohol permanently
   - C probenecid should be avoided in patients with urolithiasis
   - D if allopurinol is to be used, administration should be started as soon as possible after an acute attack
   - E allopurinol therapy should be accompanied by a non-steroidal anti-inflammatory drug or colchicine for the first 3 months

5. Allopurinol therapy
   - A should not be used with probenecid
   - B increases serum cholesterol
   - C is useful in gout resulting from renal failure
   - D should be decreased in dosage when given with azathioprine therapy
   - E may be associated with a skin rash

ANSWERS

1. A B C D E
2. A C E
3. C D
4. A C E
5. C E

CASE REPORT

REPORT OF A CASE OF NASOPHARYNGEAL CARCINOMA PRESENTING WITH SUPERIOR ORBITAL FISSURE SYNDROME

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INTRODUCTION

Superior Orbital Fissure Syndrome (SOFS) is a rare condition caused by a lesion in the region of the superior orbital fissure with involvement of the IIIrd, IVth, VIth, and of the ophthalmic division of the Vth cranial nerves. IIIrd nerve involvement may selectively affect only the superior division (which supplies the levator palpebral and superior rectus muscles, causing an external ophthalmoplegia) or the inferior division (which supplies the medial and inferior recti, inferior oblique, the ciliary muscle and the sphincter of the pupil, resulting in an internal ophthalmoplegia as well).

SOFS may also be associated with fractures of the zygomaticomaxillary complex, and of the Le Fort II and III types fractures. Haematoma in the orbital muscle cone, cavernous sinus infection/haematoma, and infection of the meninges, CNS and retrobular space can also result in the SOFS.[1]

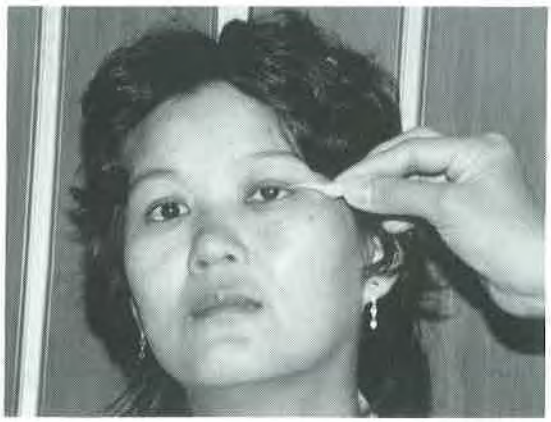
Symptoms and signs depend on the nerves initially involved, and more signs may appear as the lesion extends.

A case report on a patient with SOFS due to nasopharangeal carcinoma (NPC) is presented to highlight the lessons that may be learnt from this case.

DESCRIPTION OF CASE

PCH was a Chinese female aged 29 who presented on 18 Nov 1987 with headache, giddiness and nausea of five days duration. She gave a history of frequent headaches (mainly left temporal, non-throbbing), giddiness and nausea and left-sided facial numbness of one and a half years duration during which time she also noticed in her left eye a drooping of the upper eyelid and a squint. She also had a pain in the left side of her neck since the delivery of her only child on 14 Aug 1987 during which time she recalled a swelling on the left side of her neck.

There was no history of diplopia, tinnitus or deafness. Examination showed a normally nourished lady. In her left eye, she had a mild convergent squint and a mild ptosis. There was loss of corneal reflex and loss of pinprick sensation on the left side of her forehead and left cheek (Fig. 1). Vision was 6/6 in each eye. The swelling in the left side of her neck was an enlarged left jugulo-digastric lymphnode of about 2 cm. in diameter, firm, and slightly tender. There was no lesion in the mouth or throat to suggest a primary focus of infection.



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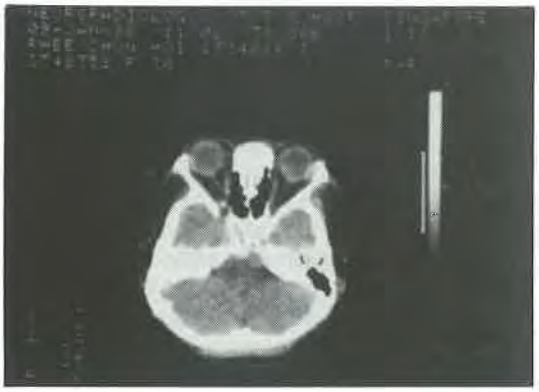
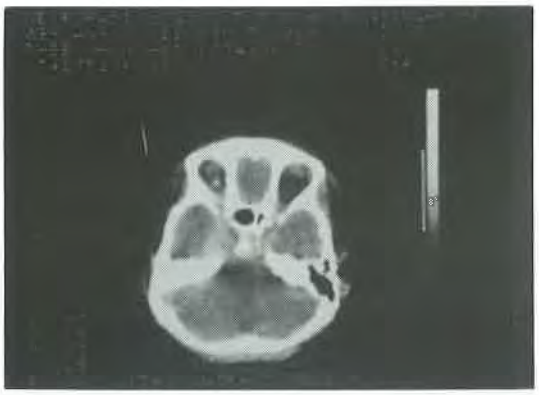
There was partial regression of the lymph node after a course of Septrin tablets.

A diagnosis of SOFS due to a space occupying lesion to explain her strabismus, ptosis, corneal anaesthesia, loss of sensation over the left side of her face (VIth, IIIrd and Vth cranial nerves) and the enlarged lymph node was made, and she was referred to a consultant ophthalmologist who concurred and referred her to a neurosurgeon.

CT scan of the head showed a left middle cranial fossa tumour (Fig. 2, 3, 4) which was thought to be a meningioma, and she was scheduled for surgery in February 1988. The operation was postponed, and she saw the author on 29 Feb with a red, painless left eye, due to a large corneal ulcer in the lower half of the cornea. Fortunately this did not involve the pupillary area. Her left eye was immediately padded and she was admitted for management of the corneal ulcer.

Craniotomy was done on 4 Mar 88. At operation, the tumour was found to be in the floor of the left middle cranial fossa between the dura and the bone, and the IIIrd nerve





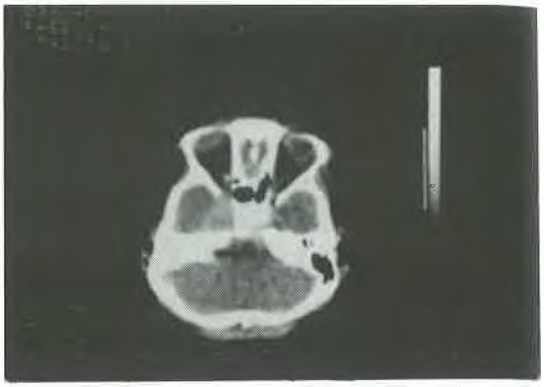


Fig. 2, 3, 4. CT scans showing left middle cranial fossa tumour.

noticed to be displaced by the tumour. The posterior aspect of the tumour was found to be adherent to the midbrain and its blood supply, making dissection very difficult and hence was incompletely excised (Fig. 5 — post-craniotomy CT scan). The intra-operative diagnosis was that of a left cavernous sinus and petrous apex meningioma.

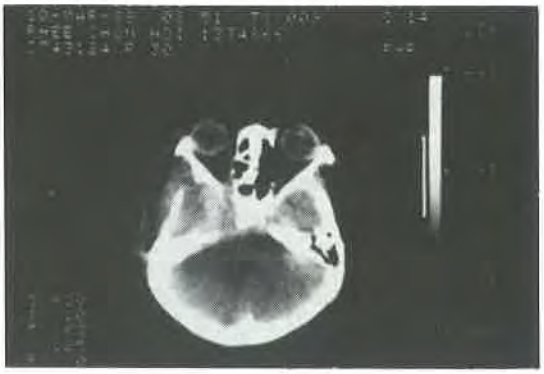


Fig. 5. Post-craniotomy CT scan showing hematoma in left cranial fossa.

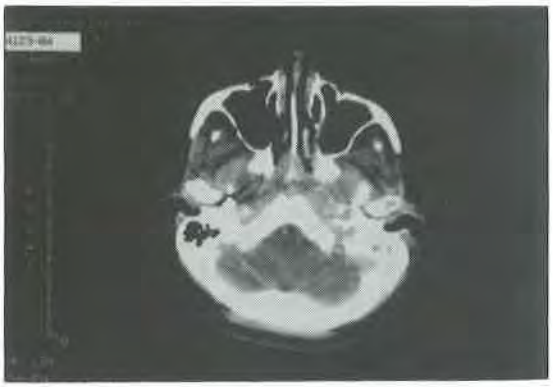
Cerebrospinal fluid (slightly blood-stained) examination post-operatively on 19 Mar 88 showed:

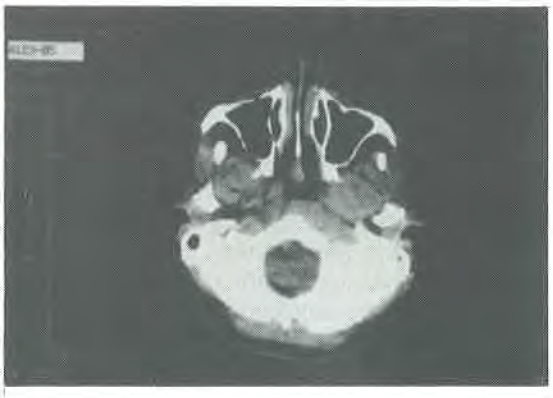
glucose 62mg.% chloride 714mg.%
total protein 40mg.% globulin negative
cell count 32 (mostly lymphocytes, few polymorphs)

Histology showed undifferentiated carcinoma. The ENT surgeon who was called in found the left side of the PNS to be bulging. Biopsy of this area on 16 Mar 88 was inconclusive (atypical cells). A repeat PNS biopsy on 23 Mar 88 showed undifferentiated carcinoma. Meanwhile, a scan of the PNS on 23 Mar 88 showed a mass involving the left medial pterygoid muscle that was causing flat-

tening of the left lateral wall of the nasopharynx. The left parapharyngeal space was pushed laterally, and the tumour appeared to have extended backwards out into the antero-lateral side of C1 and C2 vertebrae. There was a widening of the orbital fissure on the left side where the tumour had extended through (Fig. 6, 7, 8). On 5 Apr 88, blood for Ebstein Barr virus (EBV)-related antibodies was done:

Anti-EBV-Ea/IgA: positive 1: 40
(VCA — Viral Capsid Ag)





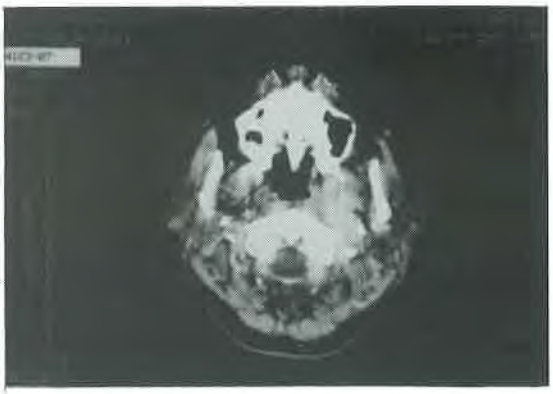


Fig. 6, 7, 8. Post-craniotomy CT scan of post-nasal space showing tumour in the left nasopharynx extending backwards out into the antero-lateral side of C1 & C2 vertebrae.

Anti-EBV-Ea/IgA: positive 1: 160
(Ea — Early Ag)

The diagnosis then was clear: Nasopharyngeal carcinoma with extension into the left middle cranial fossa. She was then referred for radiotherapy.

DISCUSSION

The orbit communicates with the middle cranial fossa through the superior orbital fissure formed by the greater and lesser wings of the sphenoid. Within the orbit, its posterior extension is confluent with the inferior orbital fissure. Through it passes the motor nerves III, IV and VI, the sensory nerve V^1 (ophthalmic branch), sympathetic fibres, and the ophthalmic veins (Fig. 9). Through the inferior orbital fissure pass branches from V^2 (infra-orbital and zygomatic nerves), the infra-orbital artery, and a communicating vein from the inferior ophthalmic vein to the

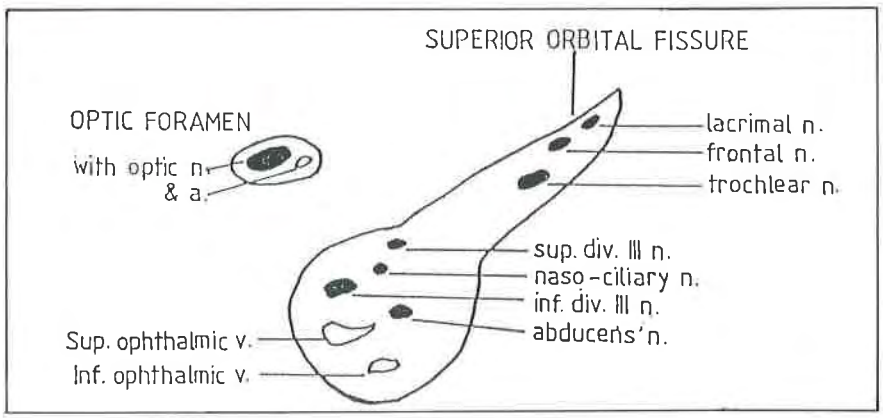


Fig. 9 Nerves & vessels passing through the superior orbital fissure (SOF) and the optic foramen.

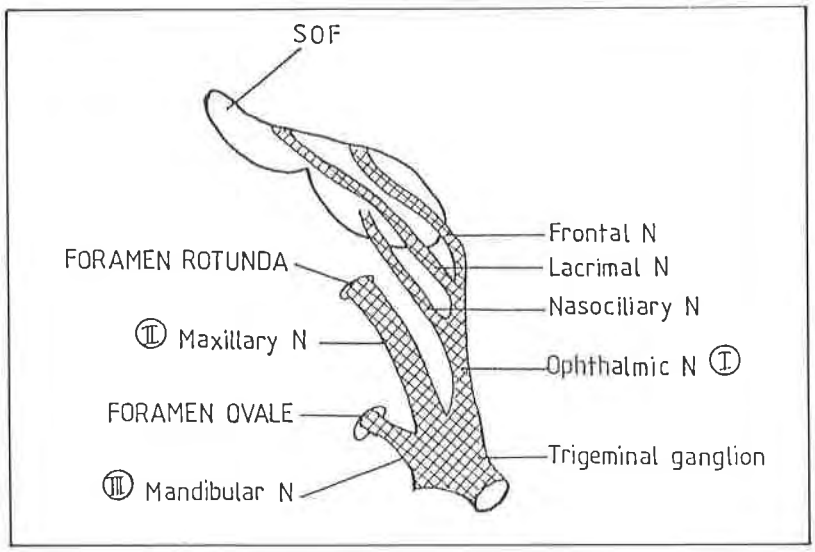


Fig. 10 Divisions of the Trigeminal Nerve in relation to the superior orbital fissures (SOF).

pterygoid plexus. Thus, extension of a lesion within this little area may involve V^2, as was seen in this patient (Fig. 10).

Involvement of the nerves in the superior orbital fissure gives rise to the signs and symptoms of the SOFS. The full blown picture is described as consisting of exophthalmos, ophthalmoplegia, retro-orbital pain, a fixed, dilated pupil, ptosis, and anaesthesia over the ophthalmic division of the trigeminal nerve, including loss of corneal reflex. Exophthalmos results from paresis of the ocular muscles, which normally exert a small backward pull on the globe. Other contributory factors to exophthalmos may be from tumour, aneurysm, haemorrhage or infection. The fixed dilated pupil results from damage to the autonomic supply to the iris, which travels with the branch of the oculomotor nerve that supplies the inferior oblique muscle, with parasympathetic disruption predominating. Incomplete forms of the SOFS often occcur, such as III and VI nerve palsy with affection of the nasociliary nerve, resulting in neuralgic pain and anaesthesia of the cornea and the skin and conjunctiva of the medial canthus. Involvement of the optic foramen and optic nerve results in retrobulbar neuritis, papilloedema and blindness — the orbital apex syndrome. In this patient, only the superior division of IIIrd nerve was involved, causing an external ophthalmoplegia, the pupil remaining normal. The nasociliary nerve was affected, causing corneal anaesthesia. By extension of the tumour, V (facial) nerve damage here involves also the maxillary division, resulting in loss of sensation of the left side of the face (forehead and cheek).

This case is uncommon, interesting as well as instructive. It points to pitfalls in diagnosis which on hindsight appear to be straightforward. The patient consulted various doctors, including her obstetrician (who also doubles as a GP) who had just delivered her child three months prior to consulting the author, and was uniformly advised that her eye problem was congenital. No squint or ptosis should be dismissed as congenital until a simple but extremely important question is asked, namely, the duration of the symptom, which, in this case, was about one and a half years. This led the author to the suspicion of a possible space occupying lesion, which suspicion was heightened by the discovery of localising signs (IIIrd, Vth and VIth cranial nerve palsies) and enlargement of the left jugulo-digastric node.

When she returned to see the author on 12 Jan 88 after the first head scan to request for an earlier operation, the author thought that the lesion was probably benign in view of its relatively long history, although the presence of the lymph node was noted, and so did not press the point with the neurosurgeon. The red herring here was that the lymph node partially resolved with antibiotic. But the

diagnosis of a meningioma could not adequately explain the presence of the node. The axiom that in this part of the world a cervical node plus localising signs means NPC till proved otherwise was overlooked. One other misleading fact is that the tumour was found to be in a common site for meningiomas. The tumour was unusual in that, except for widening of the orbital fissure, CT scan showed that the bones were unusually well preserved for a NPC of this extension, though unfortunately no bone settings were done to allow for detailed studies of the bones. (See Fig. 11 — lateral skull x-ray).

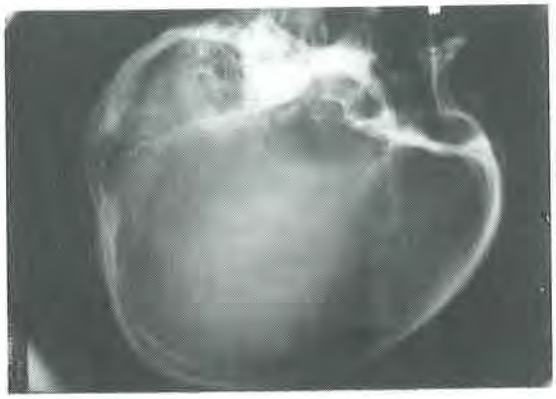


Fig. 11. No bone involvement seen on lateral skull x-ray.

Khor T H et al[2] in his detrospective review of the presenting picture of nasopharngeal tumours in Singapore patients between 1969 and 1971 found in 369 patients (362 of whom had nasopharyngeal cancer) that neck swelling was the commonest presenting symptom followed by epistaxis, nasal obstruction, tinnitus, headache, nasal discharge and deafness. Diplopia, dysphagia, impaired vision and numbness were less common presenting symptoms (see table I). Tinnitus was the commonest associated symptom, followed by epistaxis, nasal obstruction, headache and neck swelling.

Of 190 cases with skull X-rays available, 66 had involvement of the base of the skull

TABLE I
NASOPHARYNGEAL TUMOURS — SINGAPORE 1969-1971
PRESENTING AND ASSOCIATED SYMPTOMS

Symptom	No. with Presenting Symptom	Frequency of Presenting Symptom	No. with Associated Symptom	Frequency of Associated Symptom	Total Presenting and Associated	Overall Frequency
Neck Swelling	174 (1)† (1)°	47.15	52	14.13	226	61.41
Epistaxis	82 (2)x (1)M	22.22	91	24.73	173	37.01
Nasal Obstruction	33 (1)x (1)M	8.94	85 (1)x (1)M	23.10	118	31.99
Tinnitus	21	5.69	122	33.15	143	38.86
Headache	15	4.06	70 (1)x (1)M (1)°	19.02	85	23.1
Nasal Discharge	8	2.17	33	8.94	41	11.11
Deafness	8	2.17	79	21.47	87	23.64
Diplopia	5	1.35	7	1.90	12	3.26
Dysphagia	5 (1)*	1.35	8	2.17	13	3.53
Blood stained saliva	3	0.81	19	5.16	22	5.98
Impaired Vision	2	0.54	12	3.26	14	3.8
Numbness	2	0.54	13	3.53	15	4.08
Hoarseness	3	0.81	5	1.36	8	2.17
Loss of weight	—	—	15 (1)†	4.08	15	4.08
Others	7	1.90	43 (1)* (1)°	11.68	49	13.31
Unknown	1	0.27	4	1.09	5	1.36

Distribution figures refer to all tumours
Figures in brackets denote Non-Carcinomatous tumours except for the one case without biopsy

( )x = Angiofibroma
( )* = Chordoma
( )† = Lymphoma
( )M = Malignant Melanoma
( )° = Clinically NPC — No biopsy done.

Source: Khor et al. SMJ 1975 Dec: 16(4)

TABLE II
NASOPHARYNGEAL CANCER — SINGAPORE 1969-1971
RADIOLOGICAL EXTENT OF SKULL INVOLVEMENT

	No.	% Total involved
Skull base including Sphenoid Sinuses	66	88.0
Maxillary Antrum	3	4.0
Ethmoid Sinuses	—	—
Skull base & Antrum	2	2.67
Skull base & Ethmoids	—	—
Antrum & Ethmoids	1	1.33
Other combinations	3	4.0

Source: Khor et al. SMJ 1975 Dec: 16(4)

(including the sphenoid sinus), making it the most frequently involved site, accounting for 88% of all cases with definite bony involvement radiologically (table II). 78 of 366 cases of NPC showed cranial nerve palsies, the most frequently involved being the VI and the IX and X, which were invariably involved together (table III). 47 of the 78 cases (60.3%) had multiple cranial nerve palsies, the commonest grouping being the nerves to the ocular muscles (III, IV and VI). Unfortunately, there was no report of such involvement in isolation as the chief presenting feature.

The unusual presentation of isolated nerve lesions by extension of the NPC without nasal symptoms or auditory symptoms in this patient may be explained by the fact that the tumour did not ulcerate into the nasopharynx, though CT scan did show distortion of the left eustachian tube. Again, one would have expected more prominent cervical nodes by this time.

At operation, the tumour was thought to be a meningioma, sitting between the dura and the bone; no bone erosion was noted, and again invasive cancer was not suspected.

From the management point of view, it is worth noting was that she was not warned of the possible consequences of an anaesthetic cornea and the corneal ulcer presented late. The ulcer could have perforated painlessly with more serious consequences. She needed tarsorrhaphy in the management of her corneal ulcer which fortunately healed uneventfully.

EBV-related antibody titres are of prognostic value in patients with NPC. Chan S. H. et al[3] reported that anti-EBV-Ea/Iga had the best prognostic value, followed by the VCA titre. Patients with lower titres (Ea = 160, VCA = 640) survived longer than those with higher titres (Ea = 320, VCA = 1280). This patient had low titres. Tsong C.L. et al[4] also found such antibodies to have predictive value in the early diagnosis of recurrence or distant metastasis.

Still on the subject of prognosis, Fedder[5] found that the extent of the primary tumour in the nasopharynx and the presence of cranial nerve involvement were not significant determinants, but that bone invasion was a more significant prognosticator, being associated with a five-year survival rate of 14%.

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TABLE III
NASOPHARYNGEAL CANCERS — SINGAPORE 1969-1971
CRANIAL NERVE INVOLVEMENT

	VI	IX & X	V	VII	III	IV	VII	XI	VIII	II	I
Patients with Palsy	30	30	25	20	19	14	11	4	3	2	—
Frequency of Palsy %	38.5	38.5	32	25.6	24.4	17.9	14.1	5.1	3.8	2.6	—

No. Patients with Palsy/Palsies = 78
% Total Cancer Patients = 21.31%

Source: Khor et al. SMJ 1975 Dec: 16(4)

Community, Occupational and Family Medicine, NUS, for his encouragement and help in reporting this case and Mrs Ruby Koh for drawing Figure 9 and Figure 10.

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NEWS FROM THE COUNCIL

1) Continuing Medical Education

The CME Committee conducted a lunchtime "Paediatrics Course" from April-May 1988. The programme included the following:

Date	Topics	Lecturer
06.4.88	The Child with a painful joint Case Demonstration	Dr Saw Aik Hin
13.4.88	The Infant with a rash Case Demonstration	Dr Leong Yuet Yow
20.4.88	The Problem of Childhood Malignancy Case Demonstration	Prof Tan Cheng Lim
23.4.88	Bedside Teaching	Departmental Staff, Paediatric Unit SGH
27.4.88	The Child with a Persistent Cough Case Demonstration	Dr Ho Lai Yun
04.5.88	Congenital Heart Disease Case Demonstration	Dr Ng Moi Pen & Dr Wong Keng Yean
11.5.88	Failure to Thrive Case Demonstration	Dr Phua Kong Boo
14.5.88	Bedside Teaching	Department Staff, Paediatric Dept SGH

The Committee also conducted a "Geriatrics, Psychiatric and Family Medicine" Update Module in April. The programme is as follows:

Date	Topics	Lecturer	Moderator
06.5.88	Health Screening for Elderly	Dr Rilly Ray	Dr Ho Gien Chiew
13.5.88	Management of Stress	Mr Anthony Yeo	Dr Lee Suan Yew
20.5.88	Health Education in Office Practice	Dr S Wan	Dr Soh Cheow Beng
27.5.88	Approach to the Patient with Dysfunction	Dr V Atputharajah	Dr Hia Kwee Yang
03.6.88	Common Skin Diseases in the Elderly	Dr Goh Chee Leok	Dr Henry Yeo
10.6.88	Care of the Terminally Ill at Hospices and at Home	Dr Anne Merriman	Dr Chan Cheow Ju
17.6.88	Psychological and Psychiatric Problems in Adolescents	Dr Ang Peng Chye	Dr Goh Lee Gan
24.6.88	School Refusal and Phobia in Children	Dr Goh Choo Woon	Dr Hia Kwee Yang

2) Seminar

The Singapore Cancer Society and the College of General Practitioners Singapore jointly organised a special intensive course on "Management of the Terminally Ill Patient at Home" over four Sundays in May.

3) New Members

Dr Chua Sui Leng	Ordinary Member
Dr Tan Kong Chong	Associate Member
Dr Chee See Guan	Associate Member
Dr Tommy Tan Kay Seng	Associate Member
Dr Linda Leong Wai Hin	Associate Member

4) Mailing Address

With effect from 1 July 1988, the P O Box service will be terminated. Kindly mail all correspondence to:

The College of General Practitioners Singapore
College of Medicine Building
16 College Road
Singapore 0316

5) Family Medicine Training Programme 1988/90

The Ministry of Health has offered a 2-year Family Medicine Vocational Training Programme for the first time to government medical officers who wish to pursue a career in Family Medicine/Family Practice. The programme consists of eight 3 monthly rotating hospital postings and eight modules of Teaching Seminars in Family Medicine.

The Steering Committee on Family Medicine Training consists of two representatives each from the College of General Practitioners, the Ministry of Health and the Department of Community, Occupational and Family Medicine. The members are:

Chairman	:	DR CHEE YAM CHENG
Members	:	DR CHEN AI JU [alternate Dr Lam Sian Lian]
		DR LEE SUAN YEW [alternate Dr Koh Eng Kheng]
		DR LIM KIM LEONG [alternate Dr Alfred W T Loh]
		PROFESSOR LEE HIN PENG [alternate Dr Chan Cheow Ju]
		DR GOH LEE GAN

Eleven medical officers have taken up the Family Medicine traineeship for the 1988/1990 programme.

6) Family Medicine Teaching Programme

The Family Medicine Teaching sessions in the Family Medicine Teaching Programme 1988/1990 are also opened to General Practitioners and Primary Care Medical Officers of the Ministry of Health. Registration for the first two modules is now open. The first module will begin on 2 July 1988. The programme is given on the next page.

FAMILY MEDICINE TEACHING PROGRAMME JULY 88 — NOV 88

— Saturday, 2.30 – 5.30 pm

Module	1			2		
Period	July 88 — Aug 88			Sep 88 — Nov 88		
Submodule	**1A**	**PRACTICE SKILLS**		**2A**	**THE CHILD AND ADOLECSCENT**	
Concepts in FM 4 sessions per term	— 1	2 Jul	Family Medicine: Discipline & Practice; MCGP Examination	— 1	17 Sep	Common problems in childhood
	— 2	23 Jul	Consultation & communication in general practice	— 2	1 Oct	Normal & abnormal development; the handicapped child
	— 3	6 Aug	Counselling in general practice	— 3	15 Oct	Problems of the adolescent
	— 4	20 Aug	Towards earlier diagnosis	— 4	29 Oct	Behavioural problems in the child and adolescent
Submodule	**1B**	**RESP DISORDERS: CVS DISORDERS**		**2B**	**GASTROINTESTINAL DISORDERS**	
Case Discussions/ Journal club 3 sessions per term	— 1	9 Jul	Acute resp problems in general practice	— 1	24 Sep	Upper GIT problems in general practice
	— 2	30 Jul	Chronic resp problems in general practice	— 2	8 Oct	Lower GIT problems in general practice
	— 3	13 Aug	Ischaemic heart disease	— 3	22 Oct	Jaundice in general practice
Submodule	**1C**	**PRACTICE MANAGEMENT**		**2C**	**PRACTICE MANAGEMENT**	
Practice management medico-legal & ethical topic — 1 session/term		27 Aug	Medical record keeping Confidentiality of records		5 Nov	The GP's responsibility in — Notification — Certification — Dispensing Medical negligence

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