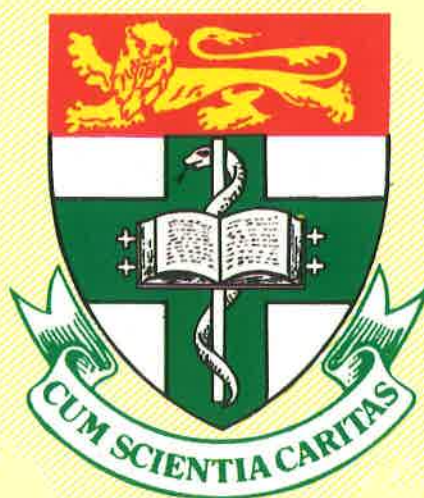


The Singapore Family Physician



ISSN 0377-5305

**The College of General
Practitioners Singapore
Vol. XV No. 4
Oct/Dec 1989**

USM Haller — A furniture system most suited for your medical practice



WHY? Because Haller's Modular design is so carefully thought out it guarantees to equip custom made and individual solutions. Later additions or changes are possible at anytime.

As a doctor, you spend a great deal of your life in your practice, so it's obvious that you will want to make your working environment as pleasant as possible.

Haller's attractive design and high quality will undoubtedly create a most soothing environment for you to work in.

For the "Haller" experience, you are invited to view our display units at:

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

If you need further information please contact: Mr James Quek/Miss Christina Lim at 4748228.



DIETHELM SINGAPORE PTE. LTD.
INSTRUMENTATION DIVISION
4, LENG KEE ROAD #02-03
THYE HONG CENTRE SINGAPORE 0315

JAF

2-17, k
tel: 08;



JAPAN MEDICAL SUPPLY

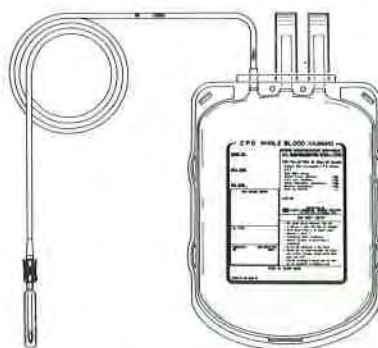
Medical technology.....new advances are being made every day.

Good medical supplies are crucial in making full use of new medical technology to provide better health care. **JMS** recognizes that it has a responsible part in advanced medical systems through disposable medical products and it continues to make efforts to accelerate the progress of health care.

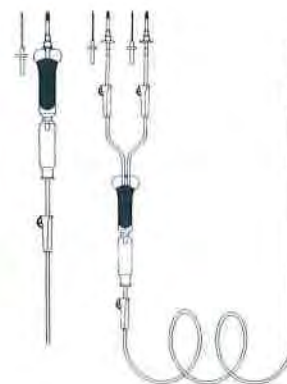
THE WORLD'S BEST QUALITY MEDICAL DISPOSABLE PRODUCTS

Our Range of Products:

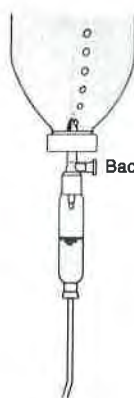
- I.V. Administration Systems
- Blood Collection & Accessories
- Blood Administration Systems
- Syringes & Needles
- Dialysis Products
- I.V. Accessories
- Feeding Systems
- Drainage Systems
- Clinical Examination Products
- Gloves & Surgical Products
- I.V. Hyperalimentation
- Laboratories Products



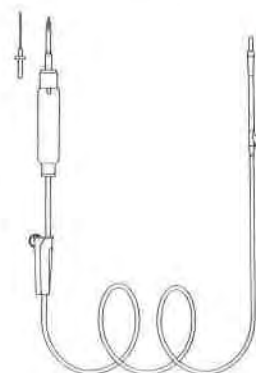
Blood Bag



Transfusion Set



Bacterial Filter



Infusion Set

JAPAN MEDICAL SUPPLY CO., LTD.

2-17, Kako-machi, Naka-ku, Hiroshima, Japan.
Tel: 082-243-1120. Facsimile: 082-246-9079. Telex: 652930 JMSJ.



JAPAN MEDICAL SUPPLY (S) PTE. LTD.

440, Ang Mo Kio Industrial Park 1, Singapore 2056.
Tel: 4571144. Facsimile: 4599564. Telex: RS 36747 JMSSIN.

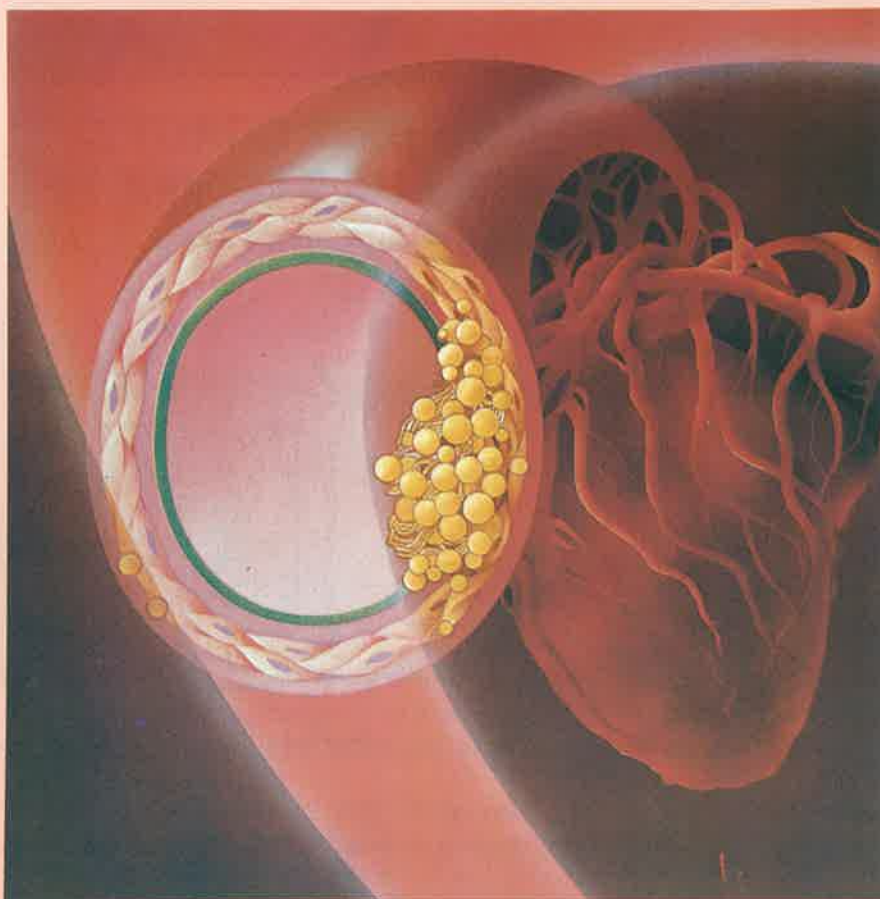
NEW

Olbetam[®]

acipimox

FOCUSSING ON THE PROBLEM OF HYPERLIPIDAEMIA

EXCELLENT LONG-TERM SAFETY PROFILE MAKES OLBETAM
SUITED FOR CHRONIC USE



**25% reduction of total
and LDL cholesterol**

20% increase of HDL cholesterol

50% reduction of triglycerides

**reduction of a major
risk factor for CHD**

Further information can be obtained from:
HONG KONG: Farmitalia Carlo Erba (HK) Ltd (Tel: H-8652292)
SINGAPORE: Apex Pharmacy International Pte Ltd (Tel: 2941355)



The Original Research From:

FARMITALIA CARLO ERBA

ERBAMONT GROUP
MONTEDISON HEALTH CARE

THE SINGAPORE FAMILY PHYSICIAN

The College of General Practitioners Singapore
College of Medicine Building, Level 1 (Right Wing)
16 College Road, Singapore 0316

Vol. XV, No 4 Oct/Dec 1989 Price to Non-Members S\$5.00 M.C.I(P) No. 9/3/88

CONTENTS

The Twelfth Council 1989/91	165
-----------------------------------	-----

EDITORIAL

Absenteeism and Sick Certification Dr Goh Lee Gan	167
A One-Day Morbidity Survey of Outpatients Dr Shañta C Emmanuel, Dr Tan Bee Yian & Dr Paul Chan Swee Mong	171
Poorly-Controlled Type 2 Diabetes Mellitus — Are they any Solutions? Dr Tan Khai Tong	198
Duodenal Ulcer — Dilemmas in Aethiology and Management Dr Roland S E Chong	203

HOME STUDY SECTION

Epilepsy and Anti-epileptic Drugs Dr Omar B S T	207
ECG Quiz Dr Baldev Singh	218
Answer to ECG Quiz	218

NEW BOOK ANNOUNCEMENT

Minor and Trace Elements in Breast Milk (World Health Organization Publications)	219
42nd International Congress on General Practice Societas Internationalis Medicinae Generalis, Austria	220

WONCA REGIONAL CONFERENCE ASIA PACIFIC REGION

June 24-28, 1990, Bali-Indonesia	221
--	-----

ONE PIECE COMPLETES THE PICTURE



Blood test results on your desk in minutes

Today's modern family clinic isn't complete without Reflotron.

Using just one drop of whole blood, Reflotron provides confirmation of your clinical judgement in minutes, right there on your desktop, giving you the power to make immediate and important treatment decisions. The most common tests are currently available, with more on the way.

Reflotron uses highly accurate dry chemistry technology coupled with advanced reflectance photometry, so there are no handling and stability problems. Its robust construction, backed by a responsive service team, ensures years of reliable use.

With Reflotron, your clinic is complete. Your patients can place their confidence in a doctor who

is in complete control of their treatment.

If you would like to see exactly how Reflotron fits into your picture, contact Boehringer Mannheim and arrange for a demonstration of the system's power and ease of use.

Reflotron®

Completes the modern family clinic



Further information available upon request
Please contact your local Boehringer Mannheim representative office
Boehringer Mannheim (Far East) Pte Ltd
450-452, Alexandra Road #07-00 Inchcape House, Singapore 0511.
Your partner in disease management

The College of General Practitioners Singapore

12TH COUNCIL 1989/91

President	Dr Koh Eng Kheng
Vice President	Dr Alfred W T Loh
Censor-in-Chief	Dr Lim Kim Leong
Hon Secretary	Dr Soh Cheow Beng
Hon Treasurer	Dr Lim Lean Huat
Council Members	Dr Chan Cheow Ju Dr Huan Meng Wah Dr John Lim Khai Liang Dr Richard Ng Mong Hoo Dr Arthur Tan Chin Lock Dr Goh Lee Gan
Hon Editor College Journal	

BOARD OF CENSORS

Censor-in-Chief	Dr Lim Kim Leong
Members	Dr James Chang Ming Yu Dr Goh Lee Gan

ADMINISTRATION

Administrative	Ms Sonia Fam
Secretary	
Asst Admin	Ms Sandy Ler
Secretary	
Senior Clerk	Mdm Rose Hoon

CONTINUING MEDICAL EDUCATION COMMITTEE

Chairman	Dr Soh Cheow Beng
Secretary	Dr Richard Ng Mong Hoo
Ex-Officio	Dr Alfred W T Loh
Members	Dr Goh Lee Gan Dr Hia Kwee Yang Dr Omar bin Saleh Talib
Library	Dr Chan Cheow Ju Dr Choo Kay Wee Dr Chong Hoi Leong

UNDERGRADUATE TEACHING COMMITTEE

Chairman	Dr Wong Wee Nam
Secretary	Dr Kevin Koh
Ex-Officio	Dr Koh Eng Kheng
Members	Dr Goh Lee Gan Dr Patrick Kee Chin Wah Dr Lim Lean Huat

PUBLICATIONS COMMITTEE

Chairman	Dr Goh Lee Gan
Secretary	Dr Chan Cheow Yu
Ex-Officio	Dr Alfred W T Loh
Members	Dr Patrick Kee Chin Wah Dr Leong Vie Chung Dr Moti H Vaswani

FINANCE COMMITTEE

Chairman	Dr Lim Lean Huat
Secretary	Dr Paul Chan Swee Mong
Ex-Officio	Dr Koh Eng Kheng
Members	Dr Leong Vie Chung Dr Frederick Samuel Dr Wong Heck Sing

RESEARCH COMMITTEE

Chairman	Dr Chan Cheow Ju
Secretary	Dr Bina Kurup
Ex-Officio	Dr Alfred W T Loh
Members	Dr Paul Chan Swee Mong Dr Choo Kay Wee Dr Shanta C Emmanuel Dr Goh Lee Gan Dr Kevin Koh Dr Lee Pheng Soon Dr Lim Lean Huat

COMMITTEE ON PRACTICE MANAGEMENT

Chairman	Dr Huan Meng Wah
Secretary	Dr Henry Yeo Peng Hock
Ex-Officio	Dr Koh Eng Kheng
Members	Dr Ganesh Balasundram Dr Kwan Kah Yee Dr Tan Chek Wee



Geigy

Cataflam is an analgesic/anti-inflammatory drug – with a difference. Specifically designed for its rapid onset of action, Cataflam is particularly suited to short-term treatment of acute painful inflammatory conditions of non-rheumatic origin. In sports and other traumatic injuries, in post-operative pain or in primary dysmenorrhoea, Cataflam starts working fast – usually within 15-30 minutes!

Presentation: Diclofenac potassium: tablets of 25 mg and 50 mg. **Indications:** Post-traumatic painful inflammatory conditions, post-operative pain and swelling. Painful and/or inflammatory conditions in gynaecology. As adjuvant in severe painful inflammatory infections of the ear, nose, or throat. Painful vertebral syndrome, muscle and joint pain. **Dosage:** Depending on indications 75-150 (dysmenorrhoea: up to 200) mg/day. See full prescribing information. In ENT conditions the lower dosage range is recommended. **Contra-indications:** Peptic ulcer, known hypersensitivity to the active substance, acetylsalicylic acid, or other prostaglandin synthetase-inhibiting drug. **Precautions:** History of gastro-intestinal disease, severe hepatic, cardiac, or renal insufficiency. Pregnancy. Patients on diuretics, and after major surgical operations. **Side effects:** Gastro-intestinal effects are usually mild. Rarely reported: peptic ulcer, bleeding from gastro-intestinal tract, blood dyscrasias, abnormalities of liver and kidney function, as well as skin and sensitivity reactions such as bronchospasm or anaphylactic/anaphylactoid systemic reactions.

Packs: 500 coated tablets of 25 mg and 50 mg

Full information is available on request
CIBA-GEIGY S.E. ASIA (Pte) Ltd 4 Fourth Lok Yang Road, Singapore 2262
1. Data on file, Ciba-Geigy Limited, Basle, Switzerland

PAIN

RELIEF

FAST

[®] CATAFLAM

**Works where it hurts
in acute, painful
inflammatory conditions**

EDITORIAL

ABSENTEEISM AND SICK CERTIFICATION

Abuses need to be investigated and rectified and unions have an important role in the process.

Straits Times Editorial, December 4, 1981¹

AN EVERGREEN PROBLEM

The sick leave issue is an evergreen one. Periodically, it will sprout in the newspapers. The latest episode was in 1989. There were several letters in the Forum Page^{2,3,4} from doctors with suggestions on how to deal with this thorny issue such as "Reward hard workers, punish malingerers" (July 12, 1989)³ and "Look into sick leave problem" (July 12, 1989).⁴ The latter said, "The problem of sick leave should be thoroughly looked into by employers, unions and the National Productivity Board in consultation with the doctors. There is no simple answers and we need to find better solutions so that we will have a healthier as well as more productive nation."

This sick certification phenomenon is also creeping into schools. It is time it be curbed.

UNHAPPINESS ALL ROUND

The demands of the sick certificate by employees who are not sick puts a strain on the doctor-patient relationship as the doctor now has to become judge instead of physician — he has to decide whether a patient is malingering instead of listening to his problems. Patients also frequently question whether doctors have a quota set for medical leave.⁵ Then there is the morale problem — employees with the sick role behaviour demoralise their colleagues and may ever lower the productivity of the

company if the number of such workers are large enough.

UNDUE EMPHASIS AND FAILURE

The undue emphasis in our society on the medical certificate for all absences encourages the medicalisation of social problems and the active, though unconscious, promotion of the sick role. There is an urgent need to ratify its proper use.

The proper use of the sick certificate and discouragement of inappropriate sick role behaviour requires the exercise of responsibilities and rights of the individual, employers, and doctors in the case of absence from work.

There is also a need to address the issue of sick certificate for absence of school. In this case the proper use of the sick certificate for absence from school requires the exercise of responsibilities and rights of the parents, the school, the pupil and the doctor.

Failure of the employer, parents and the school to exercise the responsibilities related to sickness of people under their charge and the failure of employees and pupils to honour their responsibilities is seen by the medical profession to be a major causes of the sick certification problem. Consequently, a solution of the problem will require the co-operation of these different groups of people.

ABSENCE FROM SCHOOL

The problem defined

It is observed that schools, at least some of them, insist on a medical certificate before students can be excused from school, examinations, and physical exercise for all and sundry reasons of not

being able to turn up. This is unhealthy as it encourages the sick role behaviour in our school children. It also means that students have to see a doctor for every minor illness if they are to be excused from school.

Many of us doctors, have often wondered if we are unwitting accomplices of those pupils who play truant. Many pupils who turn up at the doctor's consultation room in our experience have flimsy excuse for being unable to attend school. The possibility of some pupils seeing a doctor to get a sick certificate and then skip school without their parents' knowledge seems real.

Parents are certainly better at controlling truant behaviour. Hence, the school should let them be in control of saying if their children are able to attend school. Of course there are instances where the parents feel that they need a medical opinion to help them decide.

There are instances where a doctor's opinion is necessary in defining the fitness to return to school as for example, after an operation, a bout of chicken pox, mumps or an infectious disease.

Proposals for action

There is a need for the medical profession to have a dialogue with the Ministry of Education on the problem of certification of absence from school with the view to propose the following:

- parents be encouraged to accept the responsibility of certification of absence from school of their children as a matter of routine; the school will undertake to accept such certification as being equivalent to a doctor's certificate.
- consultation by a pupil be accompanied either by a letter from the parent or the parent or nominee in person or the school. This will reduce pupils seeking a sick certificate to play truant.
- the necessity for a doctor's certification be limited to situations where a medical opinion is needed, such as, the return to school after an attack of an in-

fectious disease, after operation or a major illness.

ABSENCE FROM WORK

The problem defined

Absence from work may be grouped into three groups of causes and the doctor is effective only in one of the three, in the other two groups, the solution lies in the management. The groups of causes are:

- the patient is genuinely sick, either physically, emotionally, or mentally.

This is the area that the doctor is most able to express an opinion on the need for sick leave. Unfortunately, some doctors may have to fight with management who believe that the doctor should be "strict on MCs".

- the patient cannot work because of other commitments at home or socially. Some have children who fall sick, aged people to look after and so on.

Management could help their staff by allowing such staff to be excused on the endorsement of the personnel officer or work on "flexitime" arrangements,⁶ rather than insisting they get a sick certificate. They are not sick.

- the patient does not want to work. It is well known that certain jobs that are boring (e.g., production operators), stressful (e.g., bus drivers) or onerous (e.g., cleaners, construction workers, fitters) are related to greater sick absence. Work overload, perceived unreasonable management are other reasons. Should the problems inherent in the occupation and the work place not be given recognition?

In dealing with work absences, management have often instituted measures that either shift the responsibility or are counter-productive in the long run. Some observed examples are given below:

- Indiscriminate blasts by management against all absences from work as "absenteeism" are detrimental to employee morale, because they lump together people whose attitudes and circumstances may be significantly different. Such management may make it

difficult for their company doctors to do their work because they insist that "the doctor must be strict on sick leave".

- Some companies give financial incentives to those who do not report sick. This means, however, that those who are genuinely sick are in fact, penalised. Furthermore, the carrot of financial reward is often not attractive enough to deter those who have to take sick leave for attitudinal reasons from doing so. Also, the system may backfire in that workers who have "lost" their financial incentive, may feel justified to take their full quota of sick leave for the year in order to compensate for their loss of incentive.
- Some employers adopt the practice of reimbursing the medical fee only when sick leave is granted. Such a practice is counterproductive as it forces the worker to request for sick leave unnecessarily.

The concept of responsibilities and rights of the employer, the employee, and the doctor in the absence from work transaction may shed light on the solution.

The employer

The employer has a responsibility to ensure that the organisational health is looked after: work overload, poor morale, poor worker attitude must be sorted out before absences from work becomes entrenched.⁷

The employer has a right to demand that each worker informs the management of occasional and excused absences in advance so that work activity of his group is minimally disturbed. It has a right of getting the worker to account for his absence from work.⁶

The employee

The worker too has responsibilities and rights. He has a responsibility of informing prearranged and making arrangements for unexpected absences to be informed. An absence of which the supervisor is not immediately informed interferes with group efficiency because scheduling is made difficult. He has a moral responsi-

bility not to shirk work by absenting himself if he knows that a rush job or a difficult job is coming up.

He has a right to expect excuse from work if he is genuinely sick and a right to expect sympathetic treatment if he is unable to be at work because of family or social circumstances.

The doctor

The doctor also has responsibilities and rights. He has a responsibility to be the employee's advocate if he feels that management is not giving the worker fair recognition of the inability to work. The doctor also has a responsibility of counselling the employee towards a positive attitude towards work, to help management determine the underlying cause for the employee's absence, particularly if this is repeated.

The doctor also has certain rights: that his opinion be given due consideration by the management and, that his opinion, unless unreasonable, should be accepted by the employee. The doctor should be given sufficient autonomy to give a professional opinion without undue need to fight for the autonomy.

A point to note here is that the doctor should deal with feedback from management about sickleave positively. One needs not be aggressive because how else is the management able to let the doctor know that his patient seems to be having undue sickness absenteeism. On the other hand, one should not veer the other extreme of being apologetic. Once an MC is given one should stand by it. The best approach is to tell management that the sickness absenteeism will be noted in the record for future reference. Of course if the patient is in poor physical health and clearly requires the sickleave, the doctor can easily defend this. This is usually not problematic.

If each of the players in the sickness absence transaction follows the rules on responsibilities and rights, there will be very little need for dispute.

Proposal for action

There is a need for the profession to in-

initiate a dialogue with the Ministry of Labour and relevant bodies to discuss the following:

- the concept of the employer, employee, and the doctor having certain responsibilities that others can insist on and certain rights that could be insisted.
- that the employers as a corporate body works out action statement on how the organisational health can be monitored, on the counselling of the frequently sick and a review of incentive schemes to reward good worker attitude and performance.
- that the employers endorse and encourage the arrangement where the personnel officer is given the responsibility of granting excused absences for staff who apply for such absences reasonable on social grounds, such as having to look after a sick member of the family.
- that the union promotes the concept of responsibilities and the positive attitude to adopt towards work amongst its members.

As was pointed out in an editorial (Singapore Family Physician 1980,⁸ we

have arrived at a certain milestone in civilization when a person cannot declare himself ill without a sick certificate.

There is a need for us to initiate a redefinition of some of the responsibilities in relation to absenteeism and sick certification.

REFERENCES

1. A good idea, if workable, Straits Times Dec 4, 1981.
2. Do away with paid medical leave, Straits Times Jun 29, 1989.
3. Look into sick leave problem, Straits Times Jul 12, 1989.
4. Reward hard workers, punish malingerers, Straits Times Jul 12, 1989.
5. No quota set for medical leave: Doctors, Straits Times Dec 4, 1981.
6. Pigors P & Myers C A. Indicator of Organisational Health: Using Personnel Research in Personnel Administration, 1977. New York: McGraw-Hill: 185-6.
7. Famularo J J. Absenteeism and tardiness in Handbook of Human Resources Administration, 2nd edition, 1987. New York: McGraw-Hill: 60-15.
8. Leong V C. Medical Certificate (Editorial). Singapore Family Physician 1980; 6, 2: 33 and 34.

GLG

A ONE-DAY MORBIDITY SURVEY OF OUTPATIENTS

S C Emmanuel MBBS, MSc (PH), AM
B Y Tan BSc (Hons), MSc (Statistics)
P S M Chan MBBS, MCGP

1 INTRODUCTION

Singapore is recognised internationally to have the infrastructure and capability for reliable and comprehensive data collection, in most areas, to support strategic planning and policy formulation.

In the field of health care, a well established surveillance system has been developed for inpatients, capturing comprehensive information on the biographic and morbidity profile of patients hospitalised in both the public and private sectors.

The morbidity pattern of patients seeking outpatient care, however, suffers seriously from a lack of information. Information on the morbidity profile of patients attending primary health care clinics is available for the public sector. The sickness profile of patients receiving care from the private sector has hitherto therefore remained largely unknown. Furthermore, an earlier survey¹ on private practitioners and private clinics showed that the private sector provides at least two-thirds of the total outpatient care in the country. The morbidity profile of outpatients if available, will provide useful inputs for:

- the training of doctors both at undergraduate and postgraduate levels, to better equip them for their important role as family physicians
- the allocation of health resources within the country, and
- for epidemiological research.

Morbidity surveys are a well-tested means for obtaining information on the sickness patterns of a population. Such surveys are conducted regularly in countries such as the United Kingdom (UK)² and Japan.

In an attempt to capture outpatient morbidity data in Singapore, the professional medical bodies and the Ministry of Health (MOH) collaborated on the conduct of a one-day morbidity survey of all outpatients, seeking care from both the private and public sectors. This survey, the first of its kind in the country, was conducted on 12 July 1988.

2 MATERIALS AND METHODS

The study was conducted under the aegis of the College of General Practitioners (CGP). Overall coordination of the study and data analysis was carried out by the Research and Evaluation (R&E) Department of the MOH. Data variables captured and the survey format were designed for simplicity, so as to minimise the impingement on the time of the doctors who participated in the survey.

Prior to the conduct of the survey, an explanatory letter outlining the objectives of the survey, endorsed by the Presidents

*Director, Research & Evaluation Department
Ministry of Health HQ/
Member, Research Committee
College of General Practitioners*

*Statistician, Research & Evaluation Department
Ministry of Health HQ*

*Immediate Past Chairman, Research Committee
College of General Practitioners*

of all the professional medical bodies, and the Permanent Secretary of Health/Director of Medical Services in Singapore, together with the survey format were despatched to all of the 1434 medical practitioners registered with the Singapore Medical Council (SMC), who were practising in the private sector. The survey was also preceded by limited publicity in a few newsletters of the medical professional organisations. To maintain confidentiality and to encourage participation, no identification of clinics and patients nor follow-up of non-responders to the survey was carried out.

For the public sector, a corresponding survey was carried out on all outpatients seen at the A&E Departments, Specialist Outpatient Clinics and the Primary Health Care Clinics on the designated survey day. This was to ensure that there would be comparable data from the public sector.

Coding of the diagnosis for every patient covered in the survey was based on the International Classification of Diseases (ICD) 9th edition. A sample of each of the survey format used for private and public sectors is in Appendix A.

3 SURVEY RESULTS

3.1 Profile of Doctors

Out of a total of 1434 registered private medical practitioners to whom survey forms were despatched, 351 doctors completed and returned them. The overall response rate to the survey from the private sector was therefore 25%.

Table 3.1 shows that corresponding their responses, there was a higher representation of private general practitioners (72.1% vs 60.5% on the SMC Register) and a lower representation of specialists (20.8% vs 26.5%) in the survey. The survey response rate for the general practitioner-and-specialist group was the highest (65.4%) amongst the five categories of doctors. However, this group of doctors is the smallest (26 doctors) in the SMC Register. The survey response rate was the lowest (18.8%) for specialists in group practice. General practitioners in solo practice who comprise the largest

(35.4%) category of private medical practitioners, registered a one-third response rate, the second highest group response.

The majority (87%) of the private medical practitioners who participated in the survey were engaged mainly in family practice rather than contract practice.

TABLE 3.1 RESPONSE RATES OF PRIVATE MEDICAL PRACTITIONER GROUPS COVERED IN THE SURVEY

Type of Practice	No. of Doctors on SMC	No. of Doctors who responded to survey	Percentage Response
Total	1434	351	24.5
General Practitioner (Single)	507	180	35.5
General Practitioner (Group)	360	73	20.3
Specialist (Single)	252	49	19.4
Specialist (Group)	128	24	18.8
General Practitioner/ Specialist (Group)	26	17	65.4
Others	161	8	5.0

For the public sector, full response was obtained from all doctors working in all Primary Health Care Clinics, Specialist Outpatient Clinics, and A&E departments, on the day of the survey.

3.2 Profile of Patients

The total number of patient attendances at the private clinics manned by the 351 doctors who responded to the survey was 11716. The corresponding figure was 14,626 outpatient attendances seen at all government clinics on the day of the survey. From this, it can be estimated that the private sector provides between two-thirds to three-quarters of the outpatient medical care to the population, which is slightly higher than the two-thirds reported six years ago.¹

Of the 11716 patients reported by the private sector during the survey, 85% sought their care from general practi-

TABLE 3.2 GENERAL PROFILE OF OUTPATIENTS ATTENDING PRIVATE AND PUBLIC SECTOR CLINICS

Selected Characteristics	Private Sector Clinics		Public Sector Clinics	
	Percent	Number	Percent	Number
Total patients		11716		14626
Sex				
Male	46.0	5389	49.5	7242
Female	54.0	6327	50.5	7384
Ethnic Group				
Chinese	76.6	8976	71.2	10416
Malay	12.6	1482	16.2	2363
Indian	5.9	689	10.4	1523
Others	4.9	569	2.2	324
House-Type				
HDB/HUDC	77.0	9021	86.4	12637
Private Apartment	6.4	750	2.6	380
Landed Property	11.5	1347	6.1	892
Others	5.1	598	4.9	717
Age (Years)				
Under 1	3.1	362	8.3	1215
1-4	9.0	1054	6.3	925
5-14	11.3	1323	8.6	1256
15-29	25.7	3002	25.6	3743
30-44	26.2	3077	21.9	3203
45-59	13.6	1598	14.8	2165
60 and above	11.1	1300	14.5	2119

tioners, 11% from specialists and the remaining 4% from general practitioner-cum-specialists. For the public sector, the distribution of outpatient workload was 60%, 32% and 8% among the Primary Health Care Clinics, Specialist Outpatient Clinics, and all A&E departments respectively.

A summary of the biographic data of patients by sector is presented in Table 3.2.

3.2.1 Sex and Age Profile of Patients

The detailed distribution of patients by sex and age in the private and public sector, is contained in Appendix B1 and C1 respectively.

On overall, the distribution of young children i.e. those aged between 0-4 years (12.1%) and the elderly i.e. those aged 60 years and above (11.1%) in the private sector, were higher than their respective

7.9% and 8.3% representation in the population. For the public sector these two age groups made up 14.6% and 14.5% of attendances at government outpatient clinics. This demonstrates that outpatient care for young children and for the elderly is sought to a greater extent from the public sector. This is desirable situation and fulfils the objectives of the health care service in the public sector. There was an overall lower representation of persons in the 10-19 years age group amongst patients attending private outpatient clinics (9.2%) as well as those seeking treatment from government clinics (10.2%) compared to that in the population (16.1%). This is because this age group comprise one of the healthier segments of an individual's life.

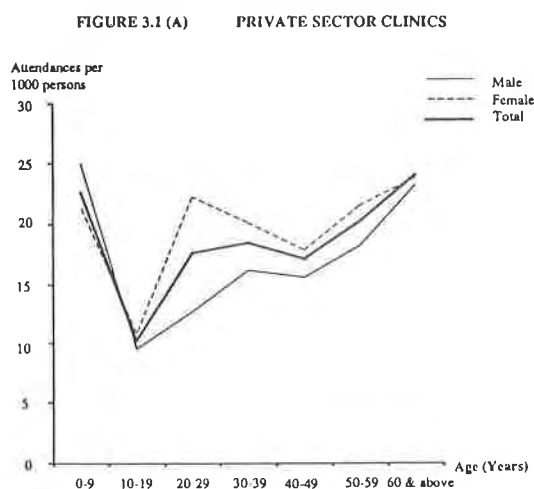
On comparison of the sexes, it was seen that the private sector had a higher proportion of female patients (54%) compared with the males (46%). Not unexpectedly,

the adjusted age-specific attendance rates* in Appendix B2, show that except for age group 0-19 years, female patients had higher attendance rates than males in every age group. This was especially so for women in the prime childbearing age of 20-29 years (22.3 vs 12.6) due to attendances for obstetric and gynaecological conditions. Men of this age group were under-represented compared to the population (16.7% vs 21.2%), due to these being the relatively healthier years of a man's life.

In the public sector, there were equal proportions of male and female patients. The patterns of age-specific attendance rates were quite similar to those of the private sector, except that elderly males had higher attendance rates at the government clinics compared with their female counterparts (Appendix C2).

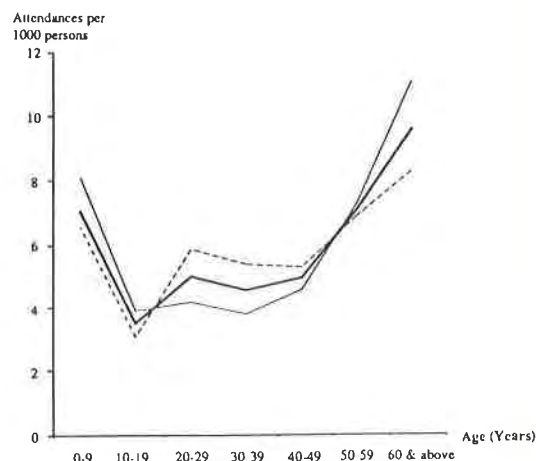
Figures 3.1(A) and 3.1(B) which depict the age-specific attendance rates per 1000 persons by sex and age, illustrate the differences in sickness load at the different ages for the two sexes, in the private and public sector respectively.

FIGURE 3.1 AGE-SPECIFIC ATTENDANCES PER 1000 PERSONS PER DAY BY AGE, SEX AND SECTOR OF ATTENDANCE



* Age-specific attendances for the private sector have been adjusted for the 75% non-response rate in the survey.

FIGURE 3.1 (B) PUBLIC SECTOR CLINICS



3.2.2 Ethnic Profile of Patients

In the private sector, the majority (77%) of patients were Chinese corroborating with their representation in the general population. Malays were slightly under-represented (12.6% vs 15%) among patients attending private clinics. There was no significant difference in the distribution of ethnic groups between the sexes.

In the public sector on the other hand, Chinese were under-represented compared to their distribution in the general population (71% vs 76%), whilst there was an over-representation of Indians (10% vs 6%) among outpatients attending government clinics.

3.2.3 Residence Type of Patients

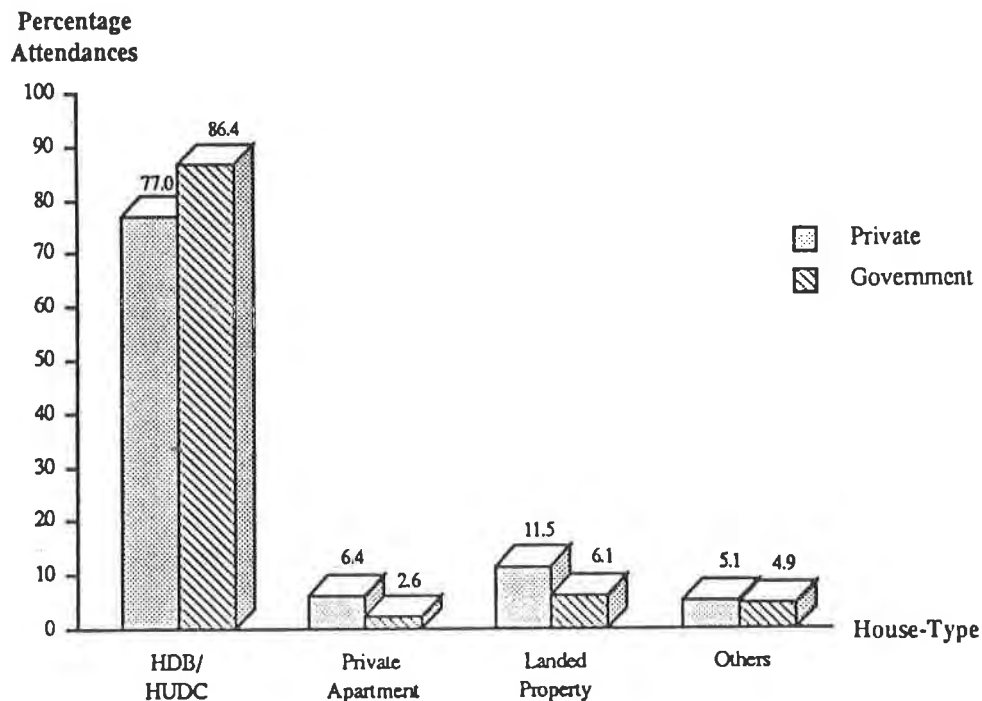
The distribution of patients by house-type was quite dissimilar between the private and public sectors (see Figure 3.2).

In the private sector, more than three quarters (77%) of the patients resided in HDB/HUDC flats. This was, not unexpectedly, lower than the 85% of the general population who are HDB dwellers. The remaining respondents comprised 6.4% residing in private apartments, 11.5% living in landed properties, and 5.1% in other types of housing.

TABLE 3.3 PERCENTAGE DISTRIBUTION OF PATIENTS OF PRIVATE AND GOVERNMENT CLINICS BY ETHNIC GROUP

Ethnic Group	Private Clinics			Government Clinics		
	Male n = (5389)	Female n = (6327)	All n = (11716)	Male n = (7242)	Female n = (7384)	All n = (14626)
Chinese	75.8	77.3	76.6	70.3	72.1	71.2
Malays	12.7	12.6	12.6	16.3	16.0	16.2
Indians	6.8	5.1	5.9	11.3	9.6	10.4
Others	4.7	5.0	4.9	2.1	2.3	2.2

FIGURE 3.2 PATIENTS OF PRIVATE AND GOVERNMENT CLINICS BY HOUSE-TYPE



Among the patients seen by general practitioners per se, 82.4% lived in HDB/ HUDC flats. This could be attributed to the fact that the majority of general practices are located within HDB housing estates. Specialists had a higher proportion (34%) of patients who lived in private apartments or landed properties.

In the public sector, a higher proportion (87%) of outpatients lived in HDB/ HUDC flats. Among the rest, about 3%

resided in private apartments and 6% in landed property.

3.2.4 Attendances at Private Clinics by Postal District

Table 3.4 shows that the sample of general practitioners who participated in the survey was generally representative of the distribution of general practitioners registered with the SMC, based on the distribution of location of their practices.

TABLE 3.4 PERCENTAGE DISTRIBUTION OF GENERAL PRACTITIONERS (GPs) AND PATIENTS BY POSTAL DISTRICT

Postal Districts	No of* Private Clinics	All GPs	GPs who responded to survey	GP Response Rate	Patients in Survey Attending Clinics	
					Outside Area of Residential PD	Within Area of Residential PD
Total Number	686	867	253	—	3861	5791
Total Percentage	100.0	100.0	100.0	—	40.0	60.0
01	45	11.3	4.3	11.2	56.4	43.6
02	16	2.9	1.2	12.0	48.3	51.7
03	51	6.7	8.3	36.2	32.9	67.1
04	11	1.6	2.4	42.9	44.3	55.7
05	31	3.8	4.3	33.3	37.9	62.1
06	7	0.8	0.4	14.3	66.7	33.3
07	39	5.2	4.0	22.2	41.2	58.8
08	19	2.7	1.6	17.4	74.8	25.2
09	36	5.8	3.6	18.0	57.0	43.0
10	32	3.7	3.6	28.1	43.4	56.6
11	7	0.9	0.8	25.0	82.9	17.1
12	48	6.8	8.0	33.9	45.6	54.4
13	16	1.5	1.6	30.8	83.7	16.3
14	37	4.3	5.1	35.1	36.7	63.3
15	22	3.7	4.3	34.4	48.6	51.4
16	48	7.3	9.1	36.5	70.3	29.7
17	1	0.3	0.4	33.3	95.2	4.6
18	23	2.1	2.4	33.0	48.2	51.8
19	41	5.2	8.7	48.9	26.8	73.2
20	40	5.5	8.3	43.8	28.3	71.7
21	11	1.7	3.2	53.3	48.5	51.5
22	38	7.8	7.2	26.5	26.5	73.5
23	27	3.0	2.0	19.2	40.5	59.5
24	2	0.2	0.0	0.0	100.0	—
25	6	0.8	0.4	14.3	93.3	6.7
26	3	0.6	0.8	40.0	80.0	20.0
27	24	3.1	3.2	29.6	39.8	60.2
28	5	0.7	0.8	33.3	64.1	35.9

* Information obtained from the Singapore Medical Council Register.

The survey also revealed that three in five patients are likely to visit general practitioners practising within their residential districts. Discounting postal districts with very few private clinics (i.e. less than 10), it was observed that a high proportion of residents (greater than 70%) living especially in the Jalan Besar, Macpherson Estate and Bedok New Town areas (postal districts 8, 13 and 16), tended to visit general practitioners located in other districts. On the other hand, patients of Bukit Merah, Clementi, Hougang, Ang Mo Kio and the Jurong East and West HDB New Towns (i.e. postal districts 3, 5, 19, 20 and 22) were more insular and largely patronised general practitioner clinics within their residential areas.

3.3 Morbidity Profile

The following section presents the morbidity profile of patients who attended private clinics and government clinics during the survey. The leading morbidity conditions, analysed by age and sex, and a detailed listing of the morbidity data grouped according to the 18 main disease categories of the ICD-9 index, are contained in Appendices B3 and B6 for the private sector and Appendices C3 and C6 for the public sector.

3.3.1 Attendances at Private General Practitioner Clinics and Primary Health Care (PHC) Clinics

a) Morbid Conditions

The following findings regarding morbidity conditions are those of patients who sought care from private general practitioners and government PHC clinics. Specialists have been analysed separately because of their specialised areas of practice.

Table 3.5 compares the leading morbidity conditions seen by general practitioners compared with those seen in the PHC clinics.

The overall 10 leading conditions attended to by general practitioners and in government PHC clinics were generally similar, though the ranking of the diseases showed some interesting differences. The overall leading conditions comprised of a good case mix of acute conditions, namely upper respiratory tract infections, gastroenteritis and conjunctivitis, and chronic medical conditions such as arthritic conditions/rheumatism, diarrhoeal diseases,

diabetes and hypertension.

Upper respiratory tract infections constituted the leading condition seen in both sectors, accounting for approximately one-third of attendances at general practitioners and 28% of attendances at government PHC clinics.

In the private sector, arthritic conditions/rheumatism were the second commonest condition for which care was sought. This was followed closely by dermatological conditions, gastroenteritis, hypertension and asthma/bronchitis. All these six conditions accounted for around 5% of total attendances each. The seventh leading condition was gastritis (3%), followed by diabetes (2.1%) and conjunctivitis (1.4%). The morbidity profile in the private sector therefore consisted of a higher load of acute conditions and a lesser load of chronic conditions.

TABLE 3.5 LEADING CONDITIONS SEEN AT PRIVATE GENERAL PRACTITIONER CLINICS AND PRIMARY HEALTH CARE CLINICS

Rank	GENERAL PRACTITIONER CLINICS		PRIMARY HEALTH CARE CLINICS	
	Condition	Percentage of all Attendances	Condition	Percentage of all Attendances
1	Upper respiratory tract infections	33.6	Upper respiratory tract infections	27.9
2	Arthritic conditions/rheumatism	6.1	Hypertensive disease	12.4
3	Dermatological disorders	5.7	Diabetes mellitus	8.0
4	Diarrhoeal diseases	5.2	Dermatological disorders	5.3
5	Hypertensive disease	5.1	Diarrhoeal diseases	3.6
6	Asthma, Bronchitis and Emphysema	4.8	Arthritic conditions/rheumatism	3.5
7	Gastritis	2.7	Asthma, Bronchitis and Emphysema	3.1
8	Diabetes mellitus	2.1	Tuberculosis	2.3
9	Conjunctivitis	1.4	Conjunctivitis	1.8
10	Neurotic disorders	1.1	Gastritis	1.2
	Ill-defined conditions	9.1	Ill-defined conditions	6.5
	Others	24.1	Others	24.3

In the public sector, on the other hand, hypertension was clearly the second leading condition (12.4%) for which care was provided. This was followed by diabetes (8%). Dermatological conditions constituted the fourth leading condition (5.3%) followed by gastroenteritis (3.6%), arthropathies (3.5%), asthma/bronchitis (3.1%) and tuberculosis (2.3%). The morbidity picture in the public sector, was thus more heavily dominated by chronic medical conditions. This difference from the private sector is probably because it is economical for patients in the long run to obtain their care and medication for long term chronic conditions from the government PHC clinics.

The other observable practice difference between the two sectors was that private practitioners were consulted more for complaints such as gastritis (2.7% vs 1.2%) and neuroses (1.1% vs 0.3%), and for ill-defined conditions (9.1% vs 6.5%). Being less well defined and perhaps related to stress, these conditions probably prompt these patients to seek the more personalised services of the private medical practitioners for care for these diseases.

The morbid conditions seen in private general practice and government PHC clinics were further analysed by age and sex (Appendices B3 and C3), ethnicity (Appendices B4 and C4), and socio-economic status (Appendices B5 and C5). Figures 3.3 (A) and 3.3 (B) illustrate the trends in the selected conditions by age group, seen in the private sector and public sector, respectively. Both sectors showed that the predominant illness seen among the younger age groups was largely upper respiratory tract infections and to a much lesser extent gastroenteritis; the older age groups, not unexpectedly, were treated more for chronic degenerative conditions such as hypertension, arthritic conditions, diabetes mellitus and a much smaller load of neuroses and tuberculosis.

When analysed by sex, both the private and government clinics showed that when the 10 leading morbid conditions were ranked for each sex, they were almost similar for both sexes except for minor variations in the ranking order. For both

FIGURE 3.3 AGE-SPECIFIC ATTENDANCES PER 1000 PERSONS PER DAY FOR SELECTED CONDITIONS

FIGURE 3.3 (A) PRIVATE GENERAL PRACTITIONER CLINICS

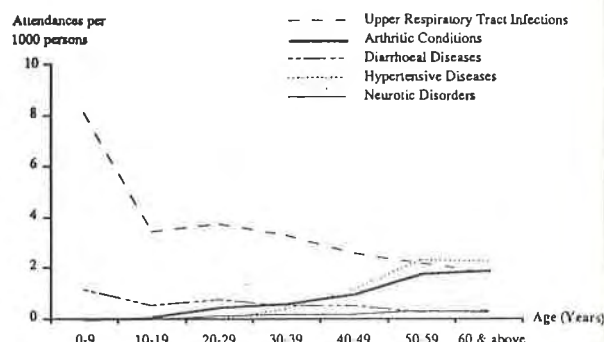
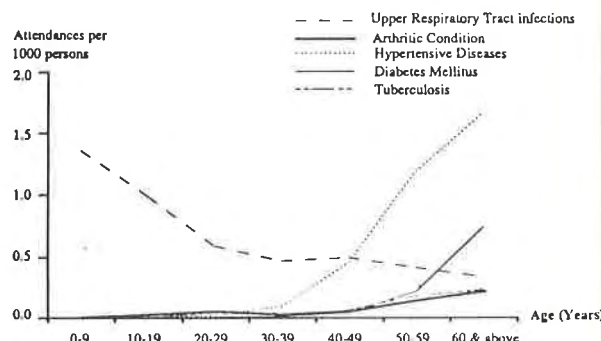


FIGURE 3.3 (B) PRIMARY HEALTH CARE CLINICS



sectors, the main differences was that females were treated more for gynaecological problems and had a higher proportion of diabetes mellitus, gastritis and ill-defined conditions. Men appeared slightly more prone to asthma and chronic obstructive lung diseases, tuberculosis and diarrhoeal disease.

The distribution of illnesses was next assessed by ethnicity. Results from both sectors showed that Chinese sought proportionally more outpatient care for hypertension, Indians for diabetes mellitus and bronchitis/asthma and Malays for conjunctivitis and other inflammation eye conditions.

For the private sector, no apparent morbidity differences could be identified for the different socio-economic groups,

using house-type as a proxy socio-economic indicator. However, in the public sector, patients residing in private apartments sought treatment for more acute conditions such as dermatological disorders, arthritic conditions, asthma/bronchitis, while patients living in HDB/HUDC apartments had a higher proportion of the more chronic conditions such as hypertension.

b) Non-Morbid Conditions

Table 3.6 presents the profile of patients attending outpatient clinics for non-morbid conditions.

Attendances during the survey, for non-morbid conditions, included consultations for prophylaxis, counselling, and for reasons such as routine medical examination (pre-employment, antenatal checks etc) family planning and immunisations. These in all, accounted for 8% of the total attendances at private general practitioners. Attendances for non-morbid conditions were more frequent among females due to the preponderance of women in the age group 20-39 receiving care during pregnancy. This discounted, the next major group of attendances was for general medical examinations, probably

pre-employment checkups. There was also a larger representation of the higher socio-economic group in this category of patients.

By comparison, in the public sector, about a quarter of all attendances at the government PHC clinics were for non-morbid conditions. Preventive health care including antenatal care, immunisations, well baby clinics, family planning and rehabilitative care made up 60%, 13% and 12% of these attendances each, for this sector.

3.3.2 Attendances at Specialist Clinics

Table 3.7 lists the 10 main conditions seen in private specialist clinics and government specialist outpatient clinics. It must be noted here that the response rate of specialists to the survey was poor.

The 1289 attendances to specialist clinics during the survey day comprised 967 sick attendances and 322 well attendances. Once again the main condition for which patients attended was upper respiratory tract infections. These was followed by attendances for cataracts, gynaecology problems, and rheumatism/

TABLE 3.7 MAJOR TYPES OF ATTENDANCES FOR MORBID CONDITIONS SEEN AT PRIVATE SPECIALIST CLINICS AND GOVERNMENT SPECIALIST OUTPATIENT CLINICS

Rank	PRIVATE SPECIALIST CLINICS		GOVERNMENT SPECIALIST OUTPATIENT CLINICS	
	Type of Attendances	Percentage of Attendances	Type of Attendances	Percentage of Attendances
1	Upper respiratory tract infections	11.3	Psychoses	9.5
2	Cataract	5.4	Skin	9.0
3	Gynaecological disorders	4.8	Cancer	7.4
4	Conjunctivitis	4.1	Urinary tract disorders	4.5
5	Arthritis/Rheumatism	3.9	Arthritis/Rheumatism	4.4
6	Hypertension	3.9	Gynaecological	3.9
7	Psychoses	3.7	Neuroses	2.4
8	Diabetes mellitus	3.5	Viral diseases	2.2
9	Refractive disorders	3.3	Upper respiratory tract infections	2.2
10	Fractures, sprain and dislocation	2.4	Cataract	2.2
	Ill-defined conditions	6.0	Ill-defined conditions	5.5
	Others	48.1	Others	46.8

arthritic conditions. These returns however, perhaps reflect the areas of speciality of the small proportion of private specialists who responded to the survey.

Of the 4639 attendances registered at all government specialist outpatient clinics on the day of the survey, there were 3928 sick attendances and 711 well attendances. The leading condition attended to by specialists in the government hospitals was psychoses, followed by dermatological disorders, malignant neoplasms, urinary tract disorders, and arthritic conditions. Once again this probably describes the distribution and patientload of the various specialities in government specialist clinics.

3.3.3 Attendances at Accident and Emergency Departments

Among the 1181 attendances recorded at the A&E Departments in all government hospitals on the survey day, the large majority (98.7%) were for morbid conditions.

Once again upper respiratory tract infections (7.5%) was the most common condition attended to in the A&E Departments. The next three leading conditions were all related to injuries namely sprains, fractures and dislocations (6.3%), contusion (5.9%), and open wounds (5.8%). In addition, about 11.5% of all morbid cases were classified as ill-defined conditions.

4 DISCUSSION

The one-day morbidity survey of outpatients was the first attempt at collecting a complete morbidity profile of all ambulatory patients in Singapore.

The survey, it must be acknowledged, received only a 25% response at this first attempt. However, an analysis of the distribution and characteristics of the private practitioners who responded showed them to be fairly representative of all private practitioners on the Singapore Medical Council Register. Furthermore, a total of more than 11,000 observations obtained during the survey provided a large enough sample size for meaningful analysis of the morbidity profile of outpatients seeking their care from the

private sector.

This survey has clearly identified for the first time, leading conditions for which ambulatory care is sought and the biographic characteristics of the patients who seek these care from the public and private sectors. This should enable:

- the teaching of medical undergraduates and family physicians to be focussed on the investigation and management of these conditions to the highest level of sophistication possible at outpatient level. Such expertise among family physicians will ensure that patients receive the best and most complete care for these conditions at ambulatory level and in so doing, cut down on referrals to secondary and tertiary institutions for management;
- enable the prioritisation of resources and facilities to be channelled to health care for these conditions at outpatient level; and
- the identification of conditions for which health education of patients should be developed to the fullest so as to prevent the disease process from progressing further, utilising measures such as by healthy practices, the early detection and management of the conditions, and patient compliance to medication where necessary.

These measures should all contribute towards minimising complications of the diseases and cut down on unnecessary hospitalisation. With the thrust of the health care policy in Singapore, as seen elsewhere the world, being geared towards cost containment, these are very important functions that the findings of this survey can serve.

The survey has shown that the leading conditions seen at the private general practices and the primary health care clinics did not vary greatly. However, doctors in the government primary health care clinics (PHC) have been shown to attend to more paediatric and elderly patients, as well as chronic cases requiring long term care e.g. diabetes mellitus and hypertensive diseases. Not unexpectedly government PHC clinics also had a much higher proportion of patients attending for preventive

health care or health screening programmes.

The conditions seen by private general practices were generally more evenly distributed favouring however the more acute conditions such as dermatological disorders and diarrhoeal diseases and for conditions such as rheumatism. This could be partly attributed to the wider range of medication offered by the private sector for these conditions. There were more visits also to general practitioners for less well defined conditions including neuroses, due perhaps to the greater confidence of the public to having more time with their doctors for such conditions and for which they may wish to have medical leave from work.

The training of medical undergraduates and family physicians whether for public or private practice should therefore be geared towards the characteristics defined by this survey.

Despite differences in survey methodology used, the morbidity profile of outpatients from this survey can be generally compared with that of the Third National Morbidity survey conducted in U.K. The U.K. survey covered one-year's recording in 48 practices in England and Wales, with a total registered population of approximately 300,000 persons, served by 146 general practitioners. The doctors were required to record each face-to-face consultation in a disease index during the survey period of July 1981 to June 1982. Based on the ICD-918 main disease groupings, the study showed that the three most common major disease groups in U.K. were:

- diseases of the respiratory system
- diseases of the nervous system and sense organs, and
- musculoskeletal system and connective tissue diseases.

This can be compared to the top three major disease groups in Singapore which showed that they were not completely similar. For Singapore, the major diseases were:

- diseases of the respiratory system
- diseases of the circulatory system, and

- infectious and parasitic diseases (covering diseases such as intestinal infections, viral diseases, mycoses etc),

For the future, it is hoped that the one-day morbidity survey can be repeated on a regular basis of say once in a few years, so as to provide medical administrators and trainers in training institutions with the latest patterns of morbidity of patients seeking outpatient care in the country. A higher participation rate from the private sector is also desirable so as to increase the comprehensiveness of the survey and to enhance the quality of the data analysed. This is important as the survey findings indicate that almost three quarters of the outpatient care to the population is provided by the private sector.

Several measures can be used to improve the response rates of private medical practitioners in future surveys. These should focus mainly on improving the preparatory phase of the survey whereby all private practitioners should have more advance notice regarding the survey and be informed of its objectives and findings and of its beneficial use to both clinicians and health planners. Extensive publicity by the professional bodies regarding the survey would also be advantageous.

Once the survey has been accepted in principle by most doctors, activities such as the follow up of non-respondents can be undertaken to ensure better coverage, without generating the suspicion of doctors. When this happens, more information could also be captured on each patient, as in done in the US annual National Ambulatory Medical Care Survey, including information on treatment offered to patients, sick-leave taking patterns, etc. This would provide additional valuable information for health care planning.

REFERENCES

1. Survey of Private Practitioners and Private Clinics, 1982. Research and Evaluation Department, Ministry of Health. (Unpublished Report).
2. Morbidity Statistics from General Practice 1981-1982. Third national study microfiche, Series MB5 no 1. London: Her Majesty's Stationery Office.

APPENDIX B1 PERCENTAGE DISTRIBUTION OF OUTPATIENT ATTENDANCES AT PRIVATE CLINICS BY AGE, SEX AND ETHNIC GROUP

Age Group (in years)	Female					Male					All				
	C	M	I	O	All Ethnic groups Total (N=100%)	C	M	I	O	All Ethnic groups Total (N=100%)	C	M	I	O	All Ethnic groups Total (N=100%)
Total number	4891	797	323	316	6327	4085	685	366	253	5389	8976	1482	689	569	11716
Total percent	77.3	12.6	5.1	5.0		75.8	12.7	6.8	4.7		76.6	12.6	5.9	4.9	
Under 1	2.7	2.3	2.5	2.2	2.6	4.0	3.1	2.2	2.0	3.7	3.3	2.7	2.3	2.1	3.1
1 - 4	7.7	9.5	4.7	8.0	7.8	10.9	9.2	7.4	10.0	10.4	9.2	9.4	6.1	8.9	9.0
5 - 9	6.4	6.0	5.0	6.4	6.3	8.8	5.8	6.6	10.7	8.4	7.5	5.9	5.8	8.3	7.3
10 - 14	3.2	4.2	2.5	2.2	3.2	5.1	4.9	4.4	4.8	5.0	4.1	4.5	3.5	3.4	4.0
15 - 19	5.7	4.7	6.6	3.9	5.6	4.7	4.6	3.9	5.6	4.7	5.2	4.7	5.2	4.7	5.2
20 - 24	10.3	13.1	12.6	12.8	10.8	6.3	9.0	9.6	6.0	6.8	8.5	11.2	11.0	9.8	9.0
25 - 29	12.2	13.5	14.2	18.3	12.8	8.9	15.0	10.7	10.3	9.9	10.7	14.2	12.3	14.7	11.5
30 - 34	11.3	10.5	13.8	14.7	11.5	9.3	11.3	12.7	11.1	9.9	10.4	10.9	13.2	13.1	10.8
35 - 39	8.4	8.9	9.1	12.5	8.7	10.1	8.4	8.8	11.1	9.8	9.2	8.7	8.9	11.9	9.2
40 - 44	6.1	5.5	7.9	7.5	6.2	6.4	6.2	6.9	6.0	6.4	6.2	5.8	7.4	6.8	6.3
45 - 49	4.9	4.0	3.5	2.2	4.6	4.9	4.9	4.7	5.2	4.9	4.9	4.4	4.1	3.5	4.7
50 - 54	4.6	6.8	6.9	2.2	4.9	4.8	4.4	4.4	6.0	4.8	4.7	5.7	5.6	3.9	4.9
55 - 59	4.1	3.6	3.8	3.2	4.0	4.2	3.7	5.8	2.8	4.1	4.1	3.6	4.9	3.0	4.0
60 - 64	4.4	3.2	2.8	1.6	4.0	3.4	4.0	3.9	2.4	3.5	3.9	3.6	3.4	2.0	3.8
65 - 69	3.1	2.4	2.8	1.3	2.9	2.9	2.5	5.2	2.4	2.9	3.0	2.4	4.1	1.8	2.9
70 & above	4.9	1.8	1.3	1.0	4.1	5.3	3.0	2.8	3.6	4.8	5.1	2.4	2.1	2.2	4.4
C - Chinese	M - Malay					I - Indian					O - Others				

APPENDIX B2 AGE-SPECIFIC ATTENDANCES AT PRIVATE CLINICS PER 1000 PERSONS PER DAY, BY SEX AND AGE GROUP

Age (Years)	Female						Male						Total		
	Population@		Attendances		Age-specific* Attendance Rate per 1000	Population		Attendances		Age-specific* Attendance Rate per 1000	Population		Attendances		
	1000s	Percent	Number	Percent	Number	Percent	1000s	Percent	Number	Percent	1000s	Percent	Number	Percent	
Total	1299.7	49.9	6327	54.0	19.5	1347.4	50.1	5389	46.0	16.0	2647.1	100.0	11716	100.0	18.1
0 - 4	100.9	7.8	656	10.4	26.1	108.5	8.1	760	14.1	28.0	209.4	7.9	1416	12.1	27.6
5 - 9	96.2	7.4	398	6.3	16.6	84.9	6.3	453	8.4	21.3	200.2	7.6	851	7.3	17.4
10 - 19	206.6	15.9	557	8.8	10.8	220.7	16.4	523	9.7	9.5	427.3	16.1	1080	9.2	10.3
20 - 29	269.3	20.7	1493	23.6	22.3	285.7	21.2	900	16.7	12.6	555.0	20.0	2393	20.4	17.6
30 - 39	253.5	19.5	1278	20.2	20.1	262.9	19.6	1062	19.7	16.2	517.4	19.5	2340	20.0	18.4
40 - 49	153.2	11.7	683	10.8	17.8	155.7	11.5	609	11.3	15.6	308.9	11.7	1292	11.0	17.1
50 - 59	103.0	8.0	563	8.9	21.6	105.0	7.8	480	8.9	18.2	208.0	7.8	1043	8.9	20.3
60 & above	117.4	9.0	696	11.0	23.9	103.5	7.7	604	11.2	23.3	220.9	9.4	1300	11.1	24.1

* Age-specific attendance rates have been adjusted for the 75% non-response rate for private medical practitioners.
i.e. age-specific attendance rate for age x = attendances for age x (100/response rate of survey)/population for age x
@ Based on 1988 population

APPENDIX B3 AGE-SPECIFIC ATTENDANCES AT PRIVATE GENERAL PRACTICES PER 1000 PERSONS PER DAY FOR SELECTED MORBID CONDITIONS, BY SEX AND AGE

Conditions	Sex	Age group						Percentage of cases	
		0-9	10-19	20-29	30-39	40-49	50-59		
Upper Respiratory Tract Infections (including influenza)	M	8.93	3.33	2.99	2.85	2.20	2.34	2.10	29.2
	F	6.11	3.57	5.91	3.85	2.92	2.03	1.41	33.6
	T	8.14	3.44	3.75	3.33	2.54	2.16	1.79	33.6
Arthropathies & Rheumatism	M	1.92	0.14	0.48	0.72	1.00	1.41	1.48	6.1
	F	-	0.14	0.62	0.62	1.10	2.16	2.23	6.1
	T	-	0.14	0.48	0.65	1.03	1.79	1.92	6.1
Enteritis & other diarrhoeal diseases	M	1.31	0.48	0.69	0.52	0.48	0.17	0.27	5.6
	F	1.03	0.48	1.00	0.48	0.45	0.31	0.31	4.8
	T	1.10	0.48	0.72	0.48	0.48	0.24	0.31	5.2
Hypertensive diseases	M	-	-	0.07	0.55	1.03	2.34	1.82	5.4
	F	-	-	-	0.21	1.24	2.30	2.54	4.9
	T	-	-	0.03	0.38	1.13	2.30	2.27	5.1
Asthma, Bronchitis & Emphysema	M	1.92	0.38	0.14	0.27	0.17	0.62	1.03	5.4
	F	1.00	0.24	0.27	0.31	0.34	0.52	0.79	3.7
	T	1.41	0.31	0.17	3.06	0.24	0.55	0.93	4.5
Dermatological disorders	M	0.55	0.45	0.55	0.58	0.65	0.17	0.72	5.3
	F	1.03	1.31	0.96	0.62	0.48	0.38	0.31	6.1
	T	0.76	0.86	0.65	0.62	0.58	0.52	0.52	5.7
Gastritis	M	0.10	0.14	0.21	0.34	0.31	0.24	0.38	2.2
	F	0.17	0.34	0.65	0.38	0.27	0.55	0.62	3.2
	T	0.10	0.24	0.34	0.38	0.31	0.38	0.52	2.7
Diabetes mellitus	M	-	-	0.03	0.10	0.41	0.69	0.82	1.8
	F	-	-	-	0.10	0.79	1.20	1.48	2.3
	T	-	-	0.03	0.10	0.31	0.52	1.20	2.1
Conjunctivitis	M	0.03	0.00	0.21	0.21	0.21	0.17	0.17	1.3
	F	0.14	0.14	0.41	0.21	0.10	0.21	0.17	1.5
	T	0.07	0.14	0.24	0.21	0.17	0.93	0.10	1.4
Neurotic disorders	M	-	-	0.03	0.10	0.31	0.21	0.21	1.1
	F	-	-	0.14	0.14	0.24	0.38	0.27	1.1
	T	-	-	0.10	0.17	0.17	0.31	0.24	1.1
Disorder of menstruation	F	-	0.10	0.89	0.34	0.21	0.03	0.03	2.1
Ill-defined conditions	M	1.62	0.62	0.86	0.93	0.82	0.89	1.51	9.2
	F	1.13	0.79	1.96	1.24	1.07	1.58	1.72	10.4
	T	1.31	0.69	1.17	1.07	0.93	1.24	1.65	9.1
Others	M	16.77	6.95	8.64	10.81	9.88	12.00	14.06	27.4
	F	14.24	7.69	13.34	11.54	11.36	14.73	14.73	22.3
	T	14.77	7.31	10.92	11.15	10.61	13.35	14.42	23.4

APPENDIX B4 **PERCENTAGE DISTRIBUTION OF PATIENTS SEEN AT PRIMARY HEALTH CLINICS
BY ETHNIC GROUP FOR SELECTED MORBID CONDITIONS**

Condition	Ethnic Group				Total
	Chinese	Malay	Indian	Others	
Upper respiratory tract infections	32.8	29.4	26.6	54.2	33.6
Arthropathies/Rheumatism	4.8	8.3	8.0	3.2	6.1
Dermatological disorders	5.4	4.5	5.2	5.7	5.7
Enteritis and other diarrhoeal diseases	4.9	4.8	4.2	3.5	5.2
Hypertensive diseases	5.3	3.3	2.3	3.5	5.1
Asthma/Bronchitis	4.0	4.0	7.0	1.7	4.8
Gastritis	2.6	2.2	2.3	2.0	2.7
Diabetes mellitus	1.7	2.3	4.0	0.5	2.1
Conjunctivitis	1.0	2.8	0.8	0.7	1.4
Neurotic disorders	1.1	1.0	0.3	-	1.1
Ill-defined conditions	8.7	10.8	10.3	7.7	9.1
Others	27.7	26.6	29.0	17.3	23.1

APPENDIX B5 **PERCENTAGE DISTRIBUTION OF PATIENTS SEEN BY GENERAL PRACTITIONERS
BY SOCIO-ECONOMIC STATUS FOR SELECTED MORBID CONDITIONS**
(House-type is used as a proxy indicator for socio-economic status)

Condition	House-Type			Total
	HDB/HUDC	Private Apartment	Landed Property	
Upper respiratory tract infections	31.8	33.2	29.5	33.6
Arthropathies/Rheumatism	5.5	4.2	5.3	6.1
Dermatological disorders	5.3	5.2	5.6	5.7
Enteritis and other diarrhoeal diseases	5.2	2.4	3.4	5.2
Hypertensive diseases	4.6	4.6	5.1	5.1
Asthma/Bronchitis	3.9	5.2	4.6	4.8
Gastritis	2.6	2.4	1.9	2.7
Diabetes Mellitus	1.9	2.4	1.9	2.1
Conjunctivitis	1.3	0.6	0.8	1.4
Neurotic disorders	1.1	0.6	0.8	1.1
Ill-defined conditions	9.6	9.6	10.0	9.1
Others	27.2	29.6	31.1	23.1

No.	Disease Condition	Attendances	
		Percent	Number
I	INFECTIOUS AND PARASITIC DISEASES	7.89	924
01	Enteritis and other diarrhoeal diseases	4.17	489
02	Tuberculosis all forms, including late effects	0.03	3
03	Chickenpox	0.34	40
04	Rubella	0.05	6
05	Measles	0.02	2
06	Viral hepatitis	0.05	6
07	Herpes simplex/zoster	0.25	29
08	Mumps	0.12	14
09	Sexually transmitted diseases	0.25	29
10	Mycoses	0.61	71
11	All other infectious and parasitic diseases	2.01	235
II	NEOPLASMS	0.44	52
12	Malignant neoplasms including neoplasms of lymphatic and haematopoietic tissue	0.28	33
13	Benign and unspecified neoplasms	0.16	19
III	ENDOCRINE, NUTRITIONAL AND METABOLIC DISEASES AND IMMUNITY DISORDERS	3.54	415
14	Thyrototoxicosis with or without goitre	0.23	27
15	Diabetes mellitus	1.95	229
16	Gout	0.23	27
17	Obesity	0.39	46
18	Other nutritional deficiencies	0.02	2
19	Other endocrine, nutritional and metabolic diseases and immunity disorders	0.72	84
IV	DISEASES OF THE BLOOD AND BLOOD-FORMING ORGANS	0.25	30
20	Anaemias	0.20	24
21	Other diseases of blood and blood forming organs	0.05	6
V	MENTAL DISORDERS	1.93	227
22	Schizophrenic psychoses	0.21	25
23	Other psychoses	0.11	13
24	Neuroses, personality disorders	0.94	110
25	Other mental disorders	0.67	79
VI	DISEASES OF THE NERVOUS SYSTEM AND SENSE ORGANS	5.76	675
26	Epilepsy	0.07	8
27	Migraine	0.64	75
28	Other diseases of central nervous system	0.14	16
29	Disorders of peripheral nervous system	0.13	15
30	Cataract	0.51	60
31	Glaucoma	0.09	11
32	Refractive errors	0.28	33
33	Conjunctivitis	1.47	172
34	Inflammation of eyelids	0.62	73
35	Other disorders of the eye and adnexa	0.88	103
36	Otitis media and mastoiditis	0.28	33
37	Deafness	0.02	2
38	Other diseases of the ear and mastoid process	0.63	74

APPENDIX B6 (Cont'd) PRINCIPAL MORBID CONDITIONS SEEN AT PRIVATE CLINICS

No.	Disease Condition	Attendances	
		Percent	Number
VII	DISEASES OF THE CIRCULATORY SYSTEM	5.82	682
39	Acute rheumatic fever	-	-
40	Chronic rheumatic heart disease	0.04	5
41	Hypertensive disease	4.47	524
42	Acute myocardial infarction and other ischaemic heart diseases	0.40	47
43	Diseases of pulmonary circulation	-	-
44	Other forms of heart disease	0.20	23
45	Cerebrovascular disease	0.12	14
46	Diseases of arteries, arterioles and capillaries	0.11	13
47	Haemorrhoids	0.32	37
48	Other diseases of circulatory system	0.16	19
VIII	DISEASES OF THE RESPIRATORY SYSTEM	32.87	3851
49	Sinusitis	0.49	57
50	Tonsillitis	0.71	83
51	Pharyngitis	2.18	255
52	Other acute respiratory infections, including influenza	25.05	2935
53	Pneumonia	0.08	9
54	Bronchitis	1.00	117
55	Asthma	2.97	348
56	Other diseases of respiratory system	0.40	47
IX	DISEASES OF THE DIGESTIVE SYSTEM	4.94	579
57	Diseases of oral cavity, salivary glands and jaws	0.50	59
58	Peptic ulcer	0.20	23
59	Appendicitis	0.04	5
60	Gastritis and duodenitis	2.29	268
61	Hernia of abdominal cavity	0.04	5
62	Chronic liver disease and cirrhosis	0.11	13
63	Cholelithiasis and other disorders of gallbladder and biliary tract	0.10	12
64	Other diseases of digestive system	1.66	194
X	DISEASES OF THE GENITO-URINARY SYSTEM	3.73	438
65	Nephritis, nephrotic syndrome and nephrosis	0.11	13
66	Calculus of urinary system	0.02	2
67	Other diseases of urinary system	0.85	100
68	Hyperplasia of prostate	0.04	5
69	Other diseases of male genital organs	0.20	24
70	Diseases of breast, ovary, fallopian tube and peritoneum	0.19	22
71	Disorders of menstruation and other abnormal bleeding from female genital tract	0.93	109
72	Infertility, female	0.23	27
73	Other diseases of female genital tract	1.16	136
XI	CHILDBIRTH AND CONDITIONS RELATED TO COMPLICATIONS OF PREGNANCY AND THE PUERPERIUM	0.26	30
74	Haemorrhage of pregnancy and childbirth	0.06	7
75	Pregnancy with abortive outcome	0.03	4
76	Delivery in a completely normal case	0.02	2
77	Complications of the puerperium	0.02	2
78	Other complications mainly related to pregnancy and childbirth	0.13	15

No.	Disease Condition	Attendances	
		Percent	Number
XII	DISEASES OF THE SKIN AND SUBCUTANEOUS TISSUE	4.84	566
79	Infections of skin and subcutaneous tissue	0.83	97
80	Dermatitis and eczema	2.00	234
81	Sebaceous glands	0.54	63
82	Urticaria	0.70	82
83	Other diseases of skin and subcutaneous tissue	0.77	90
XIII	DISEASES OF THE MUSCULOSKELETAL SYSTEM AND CONNECTIVE TISSUE	5.41	633
84	Arthropathies and related disorders	1.56	169
85	Intervertebral disc disorders	0.15	17
86	Other dorsopathies	1.63	191
87	Rheumatism, excluding the back	1.94	227
88	Other diseases of the musculoskeletal system and connective tissue	0.14	29
XIV	CONGENITAL ANOMALIES	0.22	26
89	Congenital anomalies of heart	-	-
90	All other congenital anomalies	0.22	26
XV	CERTAIN CONDITIONS ORIGINATING IN THE PERINATAL PERIOD	0.07	8
91	Respiratory syndrome and other respiratory conditions of new born and distress fetus	-	-
92	Haemolytic disease due to isoimmunization (with or without jaundice) and other perinatal jaundice	0.05	6
93	Other conditions originating in the perinatal period	0.02	2
XVI	SYMPTOMS, SIGNS AND ILL-DEFINED CONDITIONS	8.82	1034
94	Symptoms involving hand and neck	0.96	113
95	Symptoms involving respiratory system and other chest symptoms	2.14	251
96	Symptoms involving abdomen and pelvis	1.19	139
97	Other symptoms, signs and ill-defined conditions	4.53	531
XVII	INJURY AND POISONING	3.48	406
98	Fractures and Dislocations	0.13	15
99	Sprains	0.53	62
100	Concussion	-	-
101	Intracranial injury of other and unspecified nature	0.14	16
102	Lacerations and open wounds	0.54	63
103	Burns	0.16	19
104	Poisoning by drugs, medicaments, biological and chemical substances	0.01	1
105	Complications of surgical and medical care	0.44	51
106	All other injuries and poisonings	1.53	179
XVIII	NON-MORBID CONDITIONS	9.73	1140
107	General examination and screening	1.85	217
108	Rehabilitation and aftercare/follow-up	0.64	75
109	Preventive measures	2.21	259
110	Prenatal care and observation	1.76	206
111	Postpartum care and examination	0.15	17
112	Family planning	0.43	50
113	Others	2.70	316
TOTAL (All conditions)		100.00	11716

APPENDIX C1 PERCENTAGE DISTRIBUTION OF OUTPATIENT ATTENDANCES AT GOVERNMENT CLINICS BY AGE, SEX AND ETHNIC GROUP

Age Group (in years)	Female					Male					All				
	Ethnic groups Total (N=100%)					Ethnic groups Total (N=100%)					Ethnic groups Total (N=100%)				
	C	M	I	O	All	C	M	I	O	All	C	M	I	O	All
Total number	5325	1180	706	173	7384	5091	1183	817	151	7242	10416	2363	1523	324	14626
Total percent	72.1	16.0	9.6	2.3		70.3	16.3	11.3	2.1		71.2	16.2	10.4	2.2	
Under 1	7.4	10.6	6.9	6.9	7.9	8.6	10.6	7.2	7.9	8.8	8.0	10.6	7.1	7.4	8.3
1 - 4	5.2	5.7	4.0	6.9	5.2	7.3	7.9	7.7	7.9	7.5	6.2	6.8	6.0	7.4	6.3
5 - 9	4.8	3.1	4.5	2.9	4.4	5.4	4.4	7.1	2.0	5.4	5.1	3.7	5.9	2.5	4.9
10 - 14	3.2	3.1	2.7	2.3	3.1	4.7	3.0	3.9	5.3	4.3	3.9	3.0	3.3	3.7	3.7
15 - 19	5.4	5.8	6.1	5.8	5.5	7.4	8.9	7.1	5.3	7.5	6.3	7.4	6.6	5.6	6.5
20 - 24	7.8	13.3	10.8	10.4	9.0	8.7	9.7	9.1	8.6	8.9	8.2	11.5	9.8	9.6	9.0
25 - 29	11.7	13.6	15.2	19.7	12.5	6.9	9.0	9.7	12.6	7.7	9.4	11.3	12.2	16.4	10.1
30 - 34	10.6	10.0	10.6	11.0	10.5	7.8	5.7	7.5	7.3	7.4	9.2	7.8	8.9	9.3	9.0
35 - 39	8.7	6.4	6.5	4.6	8.0	6.6	6.3	5.0	6.0	6.4	7.7	6.3	5.7	5.2	7.2
40 - 44	6.9	2.7	5.1	4.0	6.0	5.8	4.2	5.0	4.0	5.4	6.3	3.5	5.1	4.0	5.7
45 - 49	5.4	3.1	5.4	2.9	5.0	4.6	3.9	4.4	6.6	4.5	5.0	3.5	4.9	4.6	4.7
50 - 54	4.9	4.5	4.1	4.6	4.8	5.1	5.1	5.1	5.1	5.1	5.0	4.8	4.6	4.8	4.9
55 - 59	4.9	4.9	4.9	4.9	4.9	5.5	5.5	5.5	5.5	5.5	5.2	5.2	5.2	5.2	5.2
60 - 64	4.0	4.0	4.0	4.0	4.0	4.9	4.9	4.9	4.9	4.9	4.4	4.4	4.5	4.4	4.4
65 - 69	2.9	2.9	2.9	2.9	2.9	3.9	3.9	3.9	3.9	3.9	3.4	3.4	3.4	3.4	3.4
70 & above	6.3	6.3	6.3	6.3	6.3	7.0	7.0	7.0	7.0	7.0	6.7	6.7	6.7	6.6	6.7

C - Chinese M - Malay I - Indian O - Others

APPENDIX C2 AGE-SPECIFIC ATTENDANCES AT GOVERNMENT CLINICS PER 1000 PERSONS PER DAY, BY SEX AND AGE GROUP

Age (Years)	Female						Male						Total					
	Population@			Attendances			Age-specific			Attendances			Population			Attendances		
	Rate per 1000			Rate per 1000			Rate per 1000			Rate per 1000			Rate per 1000			Rate per 1000		
	1000s	Percent	Number	Percent	Rate per 1000	Attendances	Rate per 1000	Attendances	Rate per 1000	1000s	Percent	Number	Percent	Rate per 1000	Attendances	Rate per 1000	Attendances	Rate per 1000
Total	1299.7	49.9	7384	50.5	5.7	1347.4	50.1	7242	49.5	5.4	2647.1	100.0	14626	100.0	18.1			
0 - 4	100.9	7.8	966	13.1	9.6	108.5	8.1	1174	16.2	10.8	209.4	7.9	2140	14.6	10.2			
5 - 9	96.2	7.4	326	4.4	3.4	84.9	6.3	388	5.4	4.6	200.2	7.6	747	5.1	3.7			
10 - 19	206.6	15.9	637	8.6	3.1	220.7	16.4	859	11.9	3.9	427.3	16.1	1496	10.2	3.5			
20 - 29	269.3	20.7	1590	21.5	5.9	285.7	21.2	1201	16.6	4.2	555.0	20.0	2791	19.1	5.0			
30 - 39	253.5	19.5	1371	18.6	5.4	262.9	19.6	996	13.8	3.8	517.4	19.5	2367	16.2	4.6			
40 - 49	153.2	11.7	810	11.0	5.3	155.7	11.5	716	9.9	4.6	308.9	11.7	1526	10.4	4.9			
50 - 59	103.0	8.0	711	9.6	6.9	105.0	7.8	762	10.5	7.3	208.0	7.8	1473	10.1	7.1			
60 & above	117.4	9.0	973	13.2	8.3	103.5	7.7	1146	15.8	11.1	220.9	9.4	2119	14.5	9.6			

APPENDIX C3 AGE-SPECIFIC ATTENDANCES AT PRIMARY HEALTH CARE PER 1000 PERSONS PER DAY FOR SELECTED MORBID CONDITIONS, BY SEX AND AGE

Conditions	Sex	Age group					Percentage of cases	
		0-9	10-19	20-29	30-39	40-49	50-59	60 & above
Upper Respiratory Tract Infections (including Influenza)	M	1.56	1.11	0.56	0.44	0.51	0.50	0.41
	F	1.29	0.87	0.60	0.51	0.46	0.35	0.29
	T	1.36	0.99	0.59	0.47	0.49	0.42	0.34
Hypertensive diseases	M	-	-	0.04	0.11	0.47	1.19	1.76
	F	-	-	0.01	0.07	0.42	1.21	1.58
	T	-	-	0.03	0.09	0.45	1.20	1.68
Diabetes mellitus	M	-	-	0.01	0.01	0.03	0.22	0.66
	F	-	-	0.01	-	0.06	0.22	0.83
	T	-	-	0.01	0.01	0.04	0.22	0.74
Dermatological disorders	M	0.24	0.15	0.08	0.08	0.12	0.12	0.21
	F	0.19	0.13	0.10	0.09	0.10	0.17	0.14
	T	0.20	0.14	0.09	0.08	0.11	0.14	0.18
Enteritis & other diarrhoeal diseases	M	0.09	0.17	0.13	0.08	0.09	0.06	0.01
	F	0.08	0.09	0.16	0.07	0.05	0.02	0.01
	T	0.08	0.13	0.14	0.08	0.07	0.04	0.01
Arthropathies/Rheumatism	M	0.02	0.05	0.09	0.10	0.10	0.16	0.27
	F	0.01	0.01	0.03	0.10	0.04	0.15	0.20
	T	0.01	0.04	0.06	0.04	0.07	0.15	0.24
Asthma, Bronchitis & Emphysema	M	0.20	0.07	0.04	0.04	0.07	0.15	0.29
	F	0.12	0.02	-	0.04	0.07	0.08	0.13
	T	0.15	0.05	0.02	0.04	0.07	0.12	0.20
Tuberculosis	M	-	-	0.04	0.03	0.09	0.28	0.45
	F	-	-	0.04	0.02	0.04	0.09	0.08
	T	-	-	0.04	0.03	0.06	0.18	0.24
Conjunctivitis	M	0.05	0.10	0.06	0.06	0.04	0.03	0.01
	F	0.05	0.08	0.04	0.04	0.03	0.02	0.03
	T	0.05	0.09	0.05	0.05	0.03	0.02	0.02
Gastritis	M	0.01	0.02	0.02	0.04	0.01	0.03	0.10
	F	0.01	0.05	0.05	0.03	0.03	-	0.07
	T	0.01	0.04	0.03	0.03	0.02	0.01	0.08
Female genital tract disorders	F	0.01	0.04	0.07	0.10	0.11	0.02	0.02
Ill-defined conditions	M	0.28	0.19	0.13	0.09	0.15	0.10	0.22
	F	0.24	0.12	0.13	0.15	0.23	0.24	0.20
	T	0.25	0.16	0.13	0.12	0.19	0.16	0.21
Others	M	0.84	0.72	0.58	0.53	0.26	0.85	1.57
	F	0.56	0.67	0.66	0.32	0.39	0.30	21.0
	T	0.66	0.70	0.62	0.43	0.32	0.58	24.3

APPENDIX C4 PERCENTAGE DISTRIBUTION OF PATIENTS SEEN AT PRIMARY HEALTH CARE
CLINICS BY ETHNIC GROUP FOR SELECTED MORBID CONDITIONS

Condition	Ethnic Group				Total
	Chinese	Malay	Indian	Others	
Upper respiratory tract infections	26.5	32.6	29.6	28.9	27.9
Hypertensive diseases	14.3	7.7	8.2	10.4	12.4
Diabetes mellitus	8.0	6.0	10.5	7.4	8.0
Dermatological disorders	5.6	4.4	4.2	8.2	5.3
Enteritis and other diarrhoeal diseases	3.1	4.5	4.8	6.7	3.6
Arthropathies/Rheumatism	3.1	3.5	5.6	2.2	3.5
Asthma/Bronchitis	2.6	4.2	5.0	3.0	3.1
Tuberculosis	2.6	1.6	1.2	3.7	2.3
Conjunctivitis	1.7	3.1	1.6	3.7	1.8
Gastritis	1.3	0.8	1.1	1.5	1.2
Ill-defined conditions	6.5	7.6	6.3	6.7	6.5
Others	24.7	16.4	21.9	17.6	24.3

APPENDIX C5 **PERCENTAGE DISTRIBUTION OF PATIENTS SEEN AT PRIMARY HEALTH CARE CLINICS BY SOCIO-ECONOMIC STATUS FOR SELECTED MORBID CONDITIONS**
(House-type is used as a proxy indicator for socio-economic status)

Condition	House-Type				Total
	HDB/HUDC	Private Apartment	Landed Property	Others	
Upper respiratory tract infections	28.3	23.1	26.1	21.9	27.9
Hypertensive diseases	12.0	7.7	16.4	15.8	12.4
Diabetes mellitus	7.8	6.7	7.0	12.9	8.0
Dermatological disorders	5.1	9.6	6.7	6.5	5.3
Enteritis and other diarrhoeal diseases	3.7	2.9	3.2	3.2	3.6
Arthropathies/Rheumatism	3.5	6.7	2.9	3.9	3.5
Asthma/Bronchitis	3.2	6.7	3.2	2.9	3.1
Tuberculosis	2.1	2.9	2.9	5.5	2.3
Conjunctivitis	2.0	3.9	1.2	0.7	1.8
Gastritis	1.3	-	0.3	1.6	1.2
Ill-defined conditions	6.9	7.7	7.0	2.9	6.5
Others	24.1	22.1	23.1	22.2	24.3

APPENDIX C6

PRINCIPAL MORBID CONDITIONS SEEN AT GOVERNMENT CLINICS

No.	Disease Condition	Attendances	
		Percent	Number
I	INFECTIOUS AND PARASITIC DISEASES	6.17	902
01	Enteritis and other diarrhoeal diseases	2.09	306
02	Tuberculosis all forms, including late effects	1.63	239
03	Chickenpox	0.23	34
04	Rubella	0.01	2
05	Measles	0.01	1
06	Viral hepatitis	0.06	9
07	Herpes simplex/zoster	0.06	9
08	Mumps	0.08	12
09	Sexually transmitted diseases	0.16	24
10	Mycoses	0.24	35
11	All other infectious and parasitic diseases	1.59	233
II	NEOPLASMS	2.75	402
12	Malignant neoplasms including neoplasms of lymphatic and haematopoietic tissue	2.20	322
13	Benign and unspecified neoplasms	0.55	80
III	ENDOCRINE, NUTRITIONAL AND METABOLIC DISEASES AND IMMUNITY DISORDERS	5.44	796
14	Thyrotoxicosis with or without goitre	0.77	113
15	Diabetes mellitus	4.05	592
16	Gout	0.15	22
17	Obesity	0.32	47
18	Other nutritional deficiencies	0.01	1
19	Other endocrine, nutritional and metabolic diseases and immunity disorders	0.14	21
IV	DISEASES OF THE BLOOD AND BLOOD-FORMING ORGANS	0.38	56
20	Anaemias	0.19	28
21	Other diseases of blood and blood forming organs	0.19	28
V	MENTAL DISORDERS	3.75	549
22	Schizophrenic psychoses	2.34	342
23	Other psychoses	0.27	40
24	Neuroses, personality disorders	0.96	141
25	Other mental disorders	0.18	26
VI	DISEASES OF THE NERVOUS SYSTEM AND SENSE ORGANS	7.08	1035
26	Epilepsy	0.55	81
27	Migraine	0.29	43
28	Other diseases of central nervous system	0.43	63
29	Disorders of peripheral nervous system	0.17	25
30	Cataract	0.64	93
31	Glaucoma	0.25	37
32	Refractive errors	0.25	36
33	Conjunctivitis	1.18	172
34	Inflammation of eyelids	0.55	81
35	Other disorders of the eye and adnexa	1.33	194
36	Otitis media and mastoiditis	0.98	143
37	Deafness	0.12	18
38	Other diseases of the ear and mastoid process	0.34	49

APPENDIX C6 (Cont'd) PRINCIPAL MORBID CONDITIONS SEEN AT GOVERNMENT CLINICS

No.	Disease Condition	Attendances	
		Percent	Number
VII	DISEASES OF THE CIRCULATORY SYSTEM	8.64	1264
39	Acute rheumatic fever	-	2
40	Chronic rheumatic heart disease	0.16	24
41	Hypertensive disease	6.16	901
42	Acute myocardial infarction and other ischaemic heart diseases	0.97	142
43	Diseases of pulmonary circulation	-	2
44	Other forms of heart disease	0.37	54
45	Cerebrovascular disease	0.32	47
46	Diseases of arteries, arterioles and capillaries	0.04	6
47	Haemorrhoids	0.40	58
48	Other diseases of circulatory system	0.19	28
VIII	DISEASES OF THE RESPIRATORY SYSTEM	16.70	2442
49	Sinusitis	0.18	26
50	Tonsillitis	0.61	89
51	Pharyngitis	0.76	111
52	Other acute respiratory infections, including influenza	12.38	1811
53	Pneumonia	0.16	24
54	Bronchitis	0.20	29
55	Asthma	1.79	262
56	Other diseases of respiratory system	0.63	92
IX	DISEASES OF THE DIGESTIVE SYSTEM	3.44	503
57	Diseases of oral cavity, salivary glands and jaws	0.35	51
58	Peptic ulcer	0.25	36
59	Appendicitis	0.31	46
60	Gastritis and duodenitis	0.83	122
61	Hernia of abdominal cavity	0.18	26
62	Chronic liver disease and cirrhosis	0.08	11
63	Cholelithiasis and other disorders of gallbladder and biliary tract	0.16	24
64	Other diseases of digestive system	1.28	187
X	DISEASES OF THE GENITO-URINARY SYSTEM	4.32	632
65	Nephritis, nephrotic syndrome and nephrosis	0.34	50
66	Calculus of urinary system	0.37	54
67	Other diseases of urinary system	0.94	138
68	Hyperplasia of prostate	0.09	13
69	Other diseases of male genital organs	0.41	60
70	Diseases of breast, ovary, fallopian tube and peritoneum	0.43	63
71	Disorders of menstruation and other abnormal bleeding from female genital tract	0.48	70
72	Infertility, female	0.46	67
73	Other diseases of female genital tract	0.62	91
XI	CHILDBIRTH AND CONDITIONS RELATED TO COMPLICATIONS OF PREGNANCY AND THE PUERPERIUM	1.00	146
74	Haemorrhage of pregnancy and childbirth	0.14	21
75	Pregnancy with abortive outcome	0.62	91
76	Delivery in a completely normal case	0.01	2
77	Complications of the puerperium	0.05	7
78	Other complications mainly related to pregnancy and childbirth	0.17	25

APPENDIX C6 (Cont'd) PRINCIPAL MORBID CONDITIONS SEEN AT GOVERNMENT CLINICS

No.	Disease Condition	Attendances	
		Percent	Number
XII	DISEASES OF THE SKIN AND SUBCUTANEOUS TISSUE	4.23	618
79	Infections of skin and subcutaneous tissue	0.75	109
80	Dermatitis and eczema	1.12	164
81	Sebaceous glands	0.55	81
82	Urticaria	0.27	40
83	Other diseases of skin and subcutaneous tissue	1.53	224
XIII	DISEASES OF THE MUSCULOSKELETAL SYSTEM AND CONNECTIVE TISSUE	3.45	504
84	Arthropathies and related disorders	0.75	109
85	Intervertebral disc disorders	0.20	29
86	Other dorsopathies	1.01	147
87	Rheumatism, excluding the back	1.07	157
88	Other diseases of the musculoskeletal system and connective tissue	0.42	62
XIV	CONGENITAL ANOMALIES	0.74	108
89	Congenital anomalies of heart	-	25
90	All other congenital anomalies	0.57	83
XV	CERTAIN CONDITIONS ORIGINATING IN THE PERINATAL PERIOD	0.62	91
91	Respiratory syndrome and other respiratory conditions of new born and distress fetus	-	9
92	Haemolytic disease due to isoimmunization (with or without jaundice) and other perinatal jaundice	0.29	43
93	Other conditions originating in the perinatal period	0.27	39
XVI	SYMPTOMS, SIGNS AND ILL-DEFINED CONDITIONS	5.44	796
94	Symptoms involving hand and neck	0.51	74
95	Symptoms involving respiratory system and other chest symptoms	0.95	139
96	Symptoms involving abdomen and pelvis	0.86	126
97	Other symptoms, signs and ill-defined conditions	3.12	457
XVII	INJURY AND POISONING	6.23	911
98	Fractures and Dislocations	0.80	117
99	Sprains	0.72	105
100	Concussion	0.19	28
101	Intracranial injury of other and unspecified nature	0.38	55
102	Lacerations and open wounds	1.09	160
103	Burns	0.07	10
104	Poisoning by drugs, medicaments, biological and chemical substances	0.02	3
105	Complications of surgical and medical care	0.11	16
106	All other injuries and poisonings	2.85	417
XVIII	NON-MORBID CONDITIONS	19.63	2871
107	General examination and screening	1.62	237
108	Rehabilitation and aftercare/follow-up	2.32	339
109	Preventive measures	9.13	1336
110	Prenatal care and observation	1.51	221
111	Postpartum care and examination	1.39	204
112	Family planning	2.02	295
113	Others	1.63	239
TOTAL (All conditions)		100.00	14626

POORLY-CONTROLLED TYPE 2 DIABETES MELLITUS — ARE THERE ANY SOLUTIONS?

Dr K T Tan MBBS, M Med, MRCP (UK)

SUMMARY

Poorly-controlled Type 2 diabetes mellitus is a commonly encountered clinical problem. Many factors contribute to the difficulty in tackling this problem. These factors and some possible solutions are discussed.

INTRODUCTION

Diabetes mellitus is a common chronic disorder in Singapore. In the 1975 country-wide survey,¹ 2% of the population was found to have diabetes and in the 1985 survey, this had risen to 4.7%.² Of these, the vast majority suffered from Type 2 or non-insulin-dependent diabetes.

Although superficially Type 2 diabetes appears quite easy to treat and patients are often asymptomatic, it is difficult to achieve good control or near normal glycaemic control in many of these patients. Oral hypoglycaemic agents are the mainstay of treatment but there is a high rate of failure which increases with duration of diabetes (up to 10% per year).^{3,4} Furthermore conversion to insulin therapy does not always ensure that better control is achieved.⁵

Registrar
Department of Medicine I
Singapore General Hospital
Outram Road
Singapore 0316

Good glycaemic control remains the best 'insurance' against the development of long term complications of diabetes and should be the goal for all diabetics provided the risks of treatment especially hypoglycaemia are not unacceptably high. There are no easy solutions to the problem of poorly-controlled Type 2 diabetes. In this article some factors behind the difficulty and possible solutions that can be considered.

FACTORS CONTRIBUTING TO POOR CONTROL

1. Pathogenesis of Type 2 Diabetes

One of the main reasons for the difficulty in treating Type 2 diabetes is a lack of complete understanding of its pathogenesis. This is the basis for rational and effective treatment but in spite of extensive research it has proved difficult to completely unravel.

We know that genetic and environmental factors lead to the development of diabetes but the exact mechanism by which this is achieved is unclear. Twin studies show that genetics play a large role in Type 2 diabetes — with almost 100% concordance in identical twins.⁶ Overnutrition, stress, obesity and urbanisation are environmental factors that have been suggested.⁷

Genetic and environmental factors are believed to be mediated through the development of insulin resistance. However some degree of beta cell defect is also necessary for the development of overt diabetes.⁸ Many obese subjects have

insulin resistance without going on to develop diabetes. Therefore insulin resistance with beta cell defect together lead to hyperglycaemia. Hyperglycaemia itself aggravates the situation by causing further insulin resistance and beta cell dysfunction thus setting up a vicious cycle.⁹

The difficulty lies in understanding how insulin resistance and beta cell defect arises and how they can be reversed. At the moment we know that diet or weight loss, sulphonylureas and biguanides can reduce insulin resistance. Insulin therapy, by reducing hyperglycaemia, also leads to reduced insulin resistance and beta cell dysfunction. However not all patients respond to these forms of treatment and the reason remains unclear.

2. Motivation of patients with Type 2 diabetes

Patient factors also contribute to poorly-controlled diabetes. It is very difficult to motivate patients to treat diabetes seriously especially when they are often asymptomatic even in the face of hyperglycaemia. These patients are often reluctant to accept insulin treatment or to perform home blood glucose monitoring which may be necessary if euglycaemia is to be achieved without undue risk of hypoglycaemia.

Diet, weight loss and weight control are central to the treatment of Type 2 diabetes especially those who are overweight. This is one of the most difficult tasks as it often means a serious alteration to the patient's lifestyle. Long-term treatment of obesity is well known to have poor results. Obese patients require relatively little to maintain obesity but a large effort to reduce their weight in the short term and even larger effort to maintain that loss. Constant 'nagging' by their physician regarding their inability to adhere to their diet and implied ill discipline does not help these patients.

The best time to motivate a patient is when diabetes is first diagnosed and the opportunity to provide good education on diabetes is also best at this time. Physicians themselves must be convinced of the value of good glycaemic control. Besides

symptoms, which are often absent and long-term complications which may be many years away and difficult to appreciate, good control can lead to reduction of the incidence of infections and other short term complications and these are of value as well. Apart from certain patients with advanced disease, old age or patients who cannot understand the risk of hypoglycaemia, normal or near normal levels of blood sugar should be the aim from the outset. This is especially so for young patients or patients who have at least another 10 or more years of life expectancy whence they are liable to suffer consequences of long term complications of diabetes.

THE USUAL STRATEGY IN TREATING TYPE 2 DIABETES

Type 2 diabetes is commonly treated in the sequence of diet, oral hypoglycaemic agents and insulin. Patients are started on dietary control if the condition is mild. If this is not sufficient, either sulphonylurea or biguanide or both is added in increasing doses until the maximum recommended dose is reached beyond which insulin treatment is instituted.

With this strategy, a significant proportion of patients remain poorly controlled.⁵ We therefore need to look for other possible strategies.

OTHER STRATEGIES IN TREATING POORLY CONTROLLED DIABETES

1. Combination of insulin and oral hypoglycaemic agents

Interest in the use of combination of insulin and oral agents have fluctuated in the last 30 years. When sulphonylureas were first introduced there were reports of the efficacy of this combination.^{10,11,12} The interest then declined only to resurface in the last few years.

There are now many studies which have reported improved glycaemic control using the combination of insulin and sulphonylureas.¹³⁻¹⁹ Various permutations — insulin added to sulphonylurea or vice versa, placebo controlled and cross-over studies have been done. It is not clear however what is the best way to introduce this combination.

The most common regime is that of using glibenclamide 15mg daily with a single dose of intermediate or long-acting insulin. Holman et al added once daily long-acting insulin to patients with hyperglycaemia on maximal sulphonylurea and reported better glycaemic control without unwanted weight gain.¹⁶ Iavicoli also used a single dose of ultralente with similar results.¹⁸ Stenman et al added glibenclamide 15 mg daily to patients already treated with insulin (either once or twice daily) and found that glycaemic control improved and insulin dose could be reduced.¹⁷ In an attempt to define which patients would benefit from combination therapy, Lewitt et al found that patients with higher C-peptide level and shorter duration of insulin therapy responded to addition of glibenclamide.¹⁹

It appears that the strategy may be either:-

- a. to add a long-acting insulin daily to a patient with poor control on maximal doses of oral agent

or

- b. to add a sulphonylurea to patients who are poorly-controlled on the usual regime of insulin.

The prospects of using combination therapy is attractive but more experience in identifying the patients that can benefit from this regime is required. Long term results from this regime is also lacking at present.

2. Fenfluramine

The high incidence of secondary failure to sulphonylurea has led to the search for other agents that have hypoglycaemic effects. Much interest has been generated recently on fenfluramine which was developed as an anti-obesity agent.

Fenfluramine has been shown to have a hypoglycaemic effect which is mediated through enhanced insulin action rather than increased insulin secretion.²⁰ When given to patients with non-insulin-dependent diabetes, fenfluramine has a hypoglycaemic effect which is independent of its ability to reduce weight.^{21,22} In patients inadequately controlled on

maximum doses of oral hypoglycaemic agents, addition of fenfluramine (60 mg daily) resulted in a significant fall in fasting blood glucose.²² Increase in insulin action and enhanced metabolic clearance of insulin was demonstrated by euglycaemic hyperinsulinaemic clamp studies.²²

These studies suggest an interesting possibility of using fenfluramine in poorly-controlled Type 2 diabetes although its exact position in the treatment strategy remains to be defined.

3. Very low calorie diet

Another strategy that has been used is the very low calorie diet (less than 600 kcal per day) with supplementation of vitamins and minerals. This regime has been demonstrated to be safe and does not lead to cardiac arrhythmias (based on 24 hours Holter monitoring).²³ When applied to obese non-insulin-dependent diabetics, this low calorie diet resulted in weight loss and reduction in fasting blood glucose and glycosylated haemoglobin level.²⁴

Although this method appears effective and safe, the lack of familiarity and difficulty in managing a very low calorie diet limits its application on a wide scale. Nonetheless it may be a strategy for short-term control of obese Type 2 diabetics who are difficult to control and where both obesity and hyperglycaemia are of concern.

4. Intermittent 'intensive' insulin therapy

The possibility of a long-term 'remission' of the metabolic defects of Type 2 diabetes by normalisation of glucose levels using short-term insulin therapy has been studied. Insulin therapy improves insulin resistance and beta cell function.²⁵ Yki-Jarvinen et al studied the effects of 4 weeks of intensive insulin therapy on 19 Type 2 diabetics with secondary drug failure and demonstrated favourable responses after insulin therapy.²⁶ However, basal glucose production tended towards pre-insulin levels on follow-up suggesting the effects were short-lived. Weight gain was also an undesirable side effect. Intermittent insulin therapy does not appear to be a useful strategy at present for poorly-controlled Type 2 diabetics.

Conclusion

The number of alternative strategies suggests that the best answer to poorly-controlled Type 2 diabetes remains to be discovered. Nevertheless, based on present understanding, the following recommendations can be made to improve the treatment of Type 2 diabetes:

1. Early strict control of diabetes from the time of diagnosis is important. Chronic hyperglycaemia aggravates the metabolic defects that are already present and may make later attempts to improve control more difficult.
2. Proper dietary advice and a proper trial of diet is important before embarking on oral hypoglycaemic agents like sulphonylureas which can cause weight gain and compound the problem.
3. Oral hypoglycaemic agents should be used if dietary control alone is insufficient. If the patient is obese, metformin may be used as the first agent rather than a sulphonylurea.
4. Insulin therapy should be instituted if control is poor on oral hypoglycaemic agents. A single dose of intermediate or long acting insulin may be less liable to cause unwanted weight gain.
5. If the patient is still poorly-controlled on insulin therapy, other strategies like the addition of an oral hypoglycaemic agent may be considered.

Finally, more effort is still needed to find solutions to the problem of poorly-controlled Type 2 diabetes.

REFERENCES

1. Cheah J S, Lui K F, Yeo P P B, Tan B Y, Tan Y T, Ng Y K. Diabetes mellitus in Singapore; results of a country-wide population survey. In Cheah J S (ed) *Proceedings of the 6th Asia and Oceania Congress of Endocrinology* 1978 pp 227-38.
2. Thai A C, Yeo P P B, Lun K C et al. Changing prevalence of diabetes mellitus in Singapore over a ten-year period. *J Med Assoc Thailand* 1987; 70 Suppl 12: 63-7.
3. Shen S W, Bressler R. Clinical pharmacology of oral anti-diabetic agents. *N Engl J Med* 1977; 296: 787-93.
4. Marble A. Incidence and causes of secondary failure in treatment with tolbutamide: experience with 2500 patients up to 5 years. *JAMA* 1962; 181: 1-4.
5. Peacock I, Tattersall R B. The difficult choice of treatment for poorly-controlled maturity-onset diabetes: tablets or insulin? *Br Med J* 1984; 228: 1956-9.
6. Barnett A H, Eff C, Leslie R D G, Pyke D A. Diabetes in identical twins: A study of 200 pairs. *Diabetologia* 1981; 20: 87-93.
7. King H. Aetiology of non-insulin-dependent diabetes mellitus. *Bailliere's Clin Endocrinol Metab* 1988; 2: 291-305.
8. Tan K T, Cheah J S. Pathogenesis of Type 1 and Type 2 diabetes mellitus. *Ann Acad Med* (submitted).
9. Unger R H, Grundy S. Hyperglycaemia as an inducer as well as consequence of impaired islet cell function and insulin resistance: implications for management of diabetes. *Diabetologia* 1985; 28: 119-21.
10. Fabrykant M. Favourable effects of supplemental orinase in insulin treated labile diabetes. *Metabolism* 1957; 6: 509-17.
11. Fabrykant M, Ashe B I. Combined insulin-tolbutamide therapy in the management of insulin-dependent diabetes. *Ann NY Acad Sci* 1957; 82: 585-89.
12. Volk B W, Lazarus S S. Significance of the effectiveness of combined insulin-orinase treatment in maturity-onset diabetes. *Am J Med Sci* 1959; 237: 1-7.
13. Rizza R A. Combined sulphonylurea and insulin therapy in insulin-dependent diabetes: research or practice? *Diabetes Care* 1985; 8: 511-4.
14. Riddle M C. New tactics for type 2 diabetes: regime based on intermediate-acting insulin taken at bedtime. *Lancet* 1985; 1: 192-5.
15. Schade D S, Mitchell W E J, Grieve G. Addition of sulphonylurea to insulin treatment in poorly controlled Type 2 diabetes — A double blind randomised clinical trial. *JAMA* 1987; 257: 2441-5.
16. Holman R R, Steemson J, Turner R E. Sulphonylurea failure in Type 2 diabetes — treatment with a basal insulin supplement. *Diabetic Med* 1987; 4: 457-62.
17. Stenman S, Group P H, Saloranta C, Totterman K J, Fyhrqvist F, Groop L. Effects of combination of insulin and glibenclamide in Type 2 (non-insulin-dependent) diabetic patients with secondary failure to oral hypoglycaemic agents. *Diabetologia* 1988; 31: 206-13.
18. Iavicoli M, Cucinotta D, DeMattia G et al. Blood glucose control and insulin secretion improved with combined therapy in Type 2 diabetic patients with secondary failure to oral hypoglycaemic agents. *Diabetic Med* 1988; 5: 849-55.
19. Lewitt M S, Yu V K F, Rennie G C et al. Effects of combined insulin-sulphonylurea therapy in Type 2 patients. *Diabetes Care* 1989; 12: 379-83.

20. Turtle J R, Burgess J A. Hypoglycaemic action of fenfluramine in diabetes mellitus. *Diabetes* 1973; 22: 858-87.
21. Verdy M, Charboneau L, Verdy I, Belanger R, Bolte E, Chiasson J L. Fenfluramine in the treatment of non-insulin diabetes — hypoglycaemic versus anorectic effect, *Int J Obes* 1983; 7: 289-97.
22. Pestell R G, Crock P A, Ward G M, Alford F P, Best J D. Fenfluramine increases insulin action in patients with NIDDM. *Diabetes Care* 1989; 12: 252-8.
23. Armatruda J M, Biddle T L, Patton M L et al. Vigorous supplementation of a hypocaloric diet prevents cardiac arrhythmias and mineral depletion. *Am J Med* 1983; 74: 1016-32.
24. Armatruda J M, Richeson J F, Welle S L, Brodows R G, Cockwood D H. The safety and efficacy of a controlled low energy (very low calories) diet in the treatment of non-insulin-dependent diabetes. *Arch Intern Med* 1988; 148: 873-7.
25. Garvey W T, Olefsky J M, Griffin J, Hamman R F, Kolterman O G. The effect of insulin treatment in insulin secretion and insulin action in Type 2 diabetes mellitus. *Diabetes* 1985; 34: 222-34.
26. Yki-Jarvinen H, Esko N, Eero H, Marja-Riitta T. Clinical benefits and mechanism of a sustained response to intermittent insulin therapy in Type 2 diabetic patients with secondary drug failure. *Am J Med* 1988; 84: 185-92.

DUODENAL ULCER — DILEMMAS IN AETIOLOGY AND MANAGEMENT

Roland S E Chong *M Med (Int Med), MRCP (UK), MRCP (I)*

INTRODUCTION

Much money has been invested in the development of new drugs. New H_2 Receptor antagonists and even drugs that can completely inhibit acid secretion such as omeprazole are now available. Yet no drug has been found which can "cure" this disease or bring it into total remission. It continues to have a relapsing and remitting course which is only temporarily suppressed when drugs are taken. The inability to control this disease well is probably a result of the inadequate knowledge we have regarding

- a) the aetiology and pathogenesis of duodenal ulcer, and
- b) the course of the disease

Presently, ulcers are thought to result from an interplay between the "aggressive" and "protective" factors.¹ The "aggressive" factors being acid and the "protective" factors being substances like mucous, bicarbonate and prostaglandins. However, even after taking the "protective" factors into account, a great deal of overlap in the acid secretion of normal and afflicted patients exists and other factors not elicited yet must be present. The course of the disease is still not thoroughly known.² While some patients have a history of multiple relapses and remissions, others have short lived histories and suffer few attacks. With this background knowledge,

any claim of a "cure" for duodenal ulcer must be looked at carefully and perhaps skeptically.

CURRENT CONTROVERSIES IN AETIOLOGY AND MANAGEMENT

There are presently a few areas of controversy in the aetiology and management of peptic ulcer. The role that non-steroidal anti-inflammatory drugs (NSAIDs) have in the pathogenesis of duodenal ulcer is still not known. The organism, *Campylobacter pylori* which was first described by Marshall in 1983³ is still causing controversy as there is equal evidence for and against it as an important aetiological factor in duodenal ulcer. The management of refractory ulcers has taken a new turn with the advent of the sodium potassium ATPase inhibitor omeprazole, a new drug which has been around for only 3-4 years. While healing is excellent, the total achlorhydria resulting has its potential hazards and long term use of the drug should be looked at carefully. Lastly, with all these advances in medical treatment, the indications for elective surgery have become less obvious and has to be weighed against other factors like costs of long term maintenance drug therapy and its potential hazards.

NSAIDs AND ULCERS

Non-steroidal anti-inflammatory drugs are perhaps one of the commonest drugs used. The greatest fear when using these drugs has always been the effect it has on the gastric and duodenal mucosa. Unfortunately, controlled studies that have been reported have only been experiments performed on rats. Studies on humans have been to date only epidemiological or retrospective. From present evidence, it would

*Senior Registrar
Medical Unit III
Singapore General Hospital
Outram Road
Singapore 0316*

appear that while acute erosions and gastropathy are known complications,^{4,5,6,7} chronic ulceration is probably not caused by it. However, in the presence of chronic ulcers, a much higher incidence of perforations and haemorrhage has been documented.^{8,9} This, together with the high prevalence of drug usage by the elderly has led to an increasing morbidity and mortality in this age group. What is also more worrying is the masking effect these drugs have on pain, making ulcer recurrences silent but potentially dangerous.¹⁰ These drugs should therefore be used carefully and it would be advisable to use concomitant H₂ antagonists in the elderly and those with a history of ulcer disease.

CAMPYLOBACTER AND ULCERS

The organism *Campylobacter pyroli* was first described in 1983.³ It was initially thought to be an important factor in duodenal ulcer formation as there was a high incidence of *Campylobacter* associated gastritis (Type B gastritis) in such patients.^{11,12} Further studies even managed to show the presence of organisms in areas of gastric metaplasia in the duodenal bulb of patients with duodenal ulcer.^{13,14} Patients with this organism who were treated with bismuth salts had as good a remission from ulcer disease as those on H₂ receptor antagonists.^{15,16} Their long term relapse rate was also lower.^{17,18} While all these studies supported this organism being directly pathogenic, other studies showed a high incidence of *Campylobacter* associated gastritis without any evidence of a duodenal ulcer.^{19,20} Another study also demonstrated similar low relapse rates with Sucralfate, a drug which had no effect on this organism.²¹ At the present time, it is still difficult to conclude as to whether this organism is a passenger or pathogen. It might be an aggravating factor rather than a direct cause of this condition. Whatever the outcome, most gastroenterologists would treat this condition if it were seen in a patient with rather resistant ulcers. However, in the absence of any severe gastritis or ulcer, this finding is best left alone.

REFRACTORY ULCERS

Refractory ulcers constitute less than 5% of all cases in hospital. By definition, a refractory ulcer is one that fails to heal after 12 weeks of full H₂ receptor antagonist blockade.^{22,23} These patients are usually young and are heavy smokers with a strong family history of ulcer disease. The ulcers are large and often have associated *Campylobacter* infection. They are therapeutic dilemmas as neither increasing the dosage of H₂ receptor antagonists or surgery is thought to help much.²⁴ Various treatment regimes including adding a second drug or sending the patient for surgery then using H₂ receptor antagonists have been suggested. Among all these solutions, the new drug omeprazole shows the most promise.²⁵ This drug, a sodium potassium ATPase pump inhibitor, cuts off acid altogether and results in almost total healing in 2-4 weeks. The only unconfirmed fear presently is the possibility of a rebound phenomenon giving rise to recurrent ulceration. Further studies need to be done.

HAZARDS OF LONG TERM MEDICATION

While the inhibition of acid secretion brings healing to ulcers, the absence of acid can lead to potential effects²⁶ (see Table I). It would appear that these effects are negligible in the short term but they may be significant if drugs are used on a long term basis. One of the worst fears is the possibility of carcinoma of the stomach. The incidence has been thought to be similar to that after a gastrectomy which might take years to manifest. As it is, the earliest H₂ receptors have been around for the last 15 years and it is still a bit too early to dismiss this potential hazard. We should therefore be more cautious in our use of the new drug omeprazole on a long term basis. The indications for maintenance therapy are set out in Table II.

TABLE I: POTENTIAL HAZARDS OF PROLONGED ACHLORHYDRIA

- 1) Carcinoma of the stomach
- 2) Gastrointestinal infections
- 3) Enterochromaffin cell hyperplasia
- 4) Higher incidence of asymptomatic ulcer recurrences

TABLE II: INDICATIONS FOR MAINTENANCE THERAPY

- 1) Frequent recurrences
- 2) On Non steroidal anti-inflammatory drugs (with a history of ulcer)
- 3) Previous history of bleed/perforation
- 4) Age/other medical problems
e.g. renal/cardiac failure
(with a history of ulcer)
- 5) Relative indications
 - a) smoking
 - b) strong family history
 - c) takes more than 6 weeks to heal
 - d) documented hypersecretion

SURGICAL MANAGEMENT

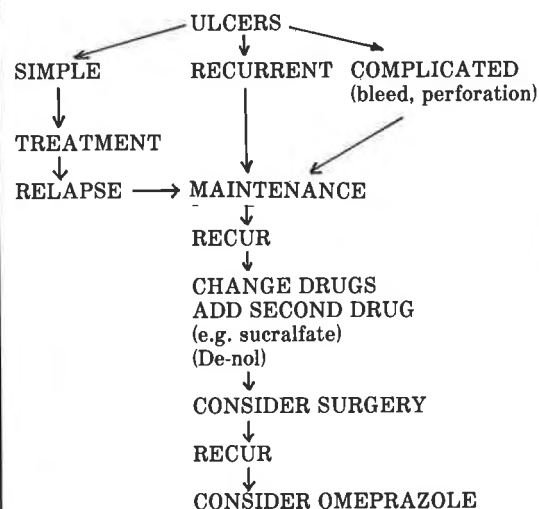
With all these advances, one questions the place for elective surgery in these patients. The guidelines have truly become rather blurred and opinions vary between surgeons and physicians. Most patients with recurrent ulceration can nowadays be controlled on maintenance H_2 receptors antagonists. With the advent of omeprazole, intractable ulceration might be a term of the past. Hence, other factors such as the costs and potential hazards of long term maintenance therapy must now be weighed against the potential morbidity of surgery. The few studies that have been done have shown that patients with recurrent ulceration who previously would have needed surgery now do reasonably well on maintenance therapy.^{27,28,29} However, none or few of them had drug free intervals and required drugs constantly as pain began once they were stopped. There are no fixed guidelines to surgery and each patient has to be individualised taking into account his or her age and socio-economic circumstances.

CONCLUSION

Many changes have occurred in the management of duodenal ulcer but the underlying aetiology, pathogenesis and course of disease is still not known. While new drugs like omeprazole show much promise, they should be used with caution and not on a long term basis. Surgery still has a place in the management of duodenal

ulcers but the patients should be individualised. A simple flow chart guiding the management of the duodenal ulcers is given in Fig. 1.

FIG. 1 ALGORITHM FOR THE MANAGEMENT OF DUODENAL ULCERS



REFERENCES

1. Schwenk M, Sewing K F. Pathophysiology of conditions related to hyperacidity in Research and Clinical Forums 1988; Vol 10 (2): 9-17.
2. Kaneko E et al. Natural history of duodenal ulcer detected by gastric mass surveys in men over 40 years of age. Scand J Gastroenterol 1989; 4: 165-170.
3. Marshall B. Unidentified curved bacillus on gastric epithelium in active chronic gastritis (letter). Lancet 1983; 1: 1273-5.
4. Fromm D. Drug induced gastric mucosal injury. World J Surg 1981; 5: 199-208.
5. O'laughlin J C, Hoftiezer J W, Ivey K J. Effect of aspirin on the human stomach in normals; an endoscopic comparison of damage produced 1 hour, 24 hours and 2 weeks after administration. Scand J Gastroenterol 1981; 16 (suppl 67): 211-4.
6. Graham D Y, Lacey Smith J. Aspirin and the stomach. Ann Int Med 1986; 104: 390-8.
7. Caruso I, Bianchi P G. Gastroscopic evaluation of anti-inflammatory agents. Br Med J 1980; 280: 75-8.
8. Somerville K, Faulkner G, Langman M. Non-steroidal anti-inflammatory drugs and bleeding peptic ulcer. Lancet 1986; 1: 462-4.
9. Walt R et al. Rising frequency of ulcer perforation in elderly people in the United Kingdom. Lancet 1986; 1: 489-92.

10. Mellem H et al. Symptoms in patients with peptic ulcer and haematemesis and/or melena related to the use of non-steroidal anti-inflammatory drugs. *Scand J Gastroenterol* 1985; 20: 1246-8.
11. Marshall B J, Warren J R. Unidentified curved bacilli in the stomach of patients with gastritis and peptic ulceration. *Lancet* 1984; 1: 1311-5.
12. Rollason T P, Stone J, Rhodes J M. Spiral organisms in endoscopic biopsies of the human stomach. *J Clin Pathol* 1984; 37: 23-6.
13. Steer H W. Surface morphology of the gastroduodenal mucosa in duodenal ulceration. *Gut* 1984; 25: 1203-10.
14. Johnston B J et al. Campylobacter like organisms in the duodenal mucosa and the effect of ulcer treatment on their presence (abstract). *Gut* 1985; 26: A579-80.
15. Vantrappen G et al. Randomised open controlled trial of colloidal bismuth subcitrate tablets and cimetidine in the treatment of duodenal ulcer. *Gut* 1980; 21: 329-33.
16. Salmon P R et al. Evaluation of colloidal bismuth (De-nol) in the treatment of duodenal ulcer employing endoscopic selection and follow-up. *Gut* 1974; 15: 189-93.
17. Martin D F et al. Difference in relapse rates of duodenal ulcer after healing with cimetidine or tripotassium dicitrate bismuthate. *Lancet* 1981; 1: 7-10.
18. Hamilton I et al. Healing and recurrence of duodenal ulcer after treatment with dicitrate bismuthate tablets or cimetidine. *Gut* 1986; 27: 106-110.
19. Langenberg et al. Campylobacter like organisms in the stomach of patients and healthy individuals (letter). *Lancet* 1984; 1: 1348-9.
20. Barthel J S et al. Pyrolic Campylobacter like organisms in asymptomatic volunteers (abstract). *Gastroenterology* 1986; 90: 1338.
21. Lam S K et al. Sucralfate overcomes adverse effect of smoking on duodenal ulcer healing and prolongs subsequent remission. *Gastroenterology* 1987; 92: 1193-201.
22. Bianchi P G, Parente F. Duodenal ulcer resistant to H_2 blockers: An emerging therapeutic problem. *Scand J Gastroenterol* 1988; 23 (suppl 153): 81-8.
23. Bardhan K D. Refractory duodenal ulcer. *Gut* 1984; 25: 711-7.
24. Hansen J H, Kruger U. Failure of proximal gastric vagotomy for duodenal ulcer resistant to cimetidine. *Lancet* 1984; 2: 84-5.
25. Brunner G et al. Therapy with omeprazole in patients with peptic ulceration resistant to extended high dose ranitidine treatment. *Digestion* 1988; 39: 80-90.
26. Wormsley K G. Risks of therapeutic achlorhydria. *Scand J Gastroenterol* 1988; 23 (suppl 153): 35-51.
27. Gray G R et al. Long term cimetidine in the management of severe duodenal ulcer dyspepsia. *Gastroenterology* 1978; 74: 397-400.
28. Gray G R et al. Five year study of cimetidine or surgery for severe duodenal ulcer dyspepsia. *Lancet* 1982; 1: 787-8.
29. Hansell D T et al. Maintenance cimetidine instead of surgery for duodenal ulcer: the first decade. *Gut* 1989; 30: 786-9.

FURTHER READING

1. Soll A H, Isenberg J I. Duodenal ulcer diseases. In: Sleisenger M H, Marvin H, Fordtran J S (eds). *Gastrointestinal Disease*. W B Saunders. Philadelphia. 1989 7th Edition (47): 814-79.
2. Maintenance treatment of hyperacid related conditions. In *Research and Clinical Forums*. Royal Wells Medical Press, England. Vol 10 (2) 1988.
3. Boyd E J S, Wormsley K G. Etiology and pathogenesis of peptic ulcer. In: Berk J E (ed). *Bockus Gastroenterology*. W B Saunders. Philadelphia. 4th ed 1985. Vol 2 (66): 1013-59.
4. Thomson A B R, Mahandren V. Medical management of uncomplicated peptic ulcer disease. In: Berk J E (ed). *Bockus Gastroenterology*. W B Saunders. Philadelphia. 4th ed 1985. Vol 2 (68): 1116-1154.

EPILEPSY AND ANTI-EPILEPTIC DRUGS

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

INTRODUCTION

Epilepsy comprises sudden, excessive depolarization of groups of cerebral neurones, which may remain localised (focal epilepsy) or which may spread to cause a generalised seizure. Epilepsy can be severely disruptive of life at home and at work. The medical management of epileptic seizures, whether single or recurrent, therefore involves much more than deciding to start anticonvulsant treatment. It includes:

- * determining the cause and eliminating it (if possible)
- * giving appropriate advice concerning the avoidance of fits, and the avoidance of injury or danger resulting from them
- * treatment with anticonvulsant drugs, if possible with laboratory control
- * genetic counselling
- * occasionally surgery in drug-resistant focal, especially psychomotor, fits
- * careful long-term follow-up
- * deciding whether, when and how to withdraw drug treatment

This article, however, will focus mainly on the drug treatment of epilepsy.

ANTICONVULSANT DRUGS

Anti-epileptic drugs that are currently available include phenytoin sodium, carbamazepine, sodium valproate, ethosuximide, phenobarbitone, primidone and clonazepam.

Mode of Action

The mechanism of action of the anti-epileptics is not fully understood. The final therapeutic effect could be a combination of the inhibition of the excitatory neuro-

transmitters like glutamate and aspartate and the enhancement of the inhibitory transmitter gamma-aminobutyric acid (GABA). The mechanism of action at the cellular level, probably involves interference with sodium, potassium and calcium permeability.

Pharmacokinetics

Anticonvulsants can be taken orally and are well absorbed and widely distributed in the body. The pharmacokinetic properties of anticonvulsant drugs are summarised in Table 1. In general they bind to plasma proteins and other tissues, are metabolised in the liver and excreted in the urine. Most anticonvulsant drugs are in part metabolised by hepatic microsomal enzymes, whose activity may be altered by some anticonvulsants and some other drugs.

With many anticonvulsant drugs used alone the rate of metabolism, and therefore the steady state plasma level, is approximately proportional to the dose (first-order kinetics), but an important exception is phenytoin sodium, which saturates the hepatic, microsomal system within the therapeutic range (kinetics then approaching zero-order). Thereafter, a small increase in dosage will result in increased half-life and hence a much larger than proportionate rise in plasma level.

Side Effects

These fall into four categories (Table 2):

- * acute (idiosyncratic) reactions
- * reversible dose-related reactions (intoxication)
- * chronic side effects, which are not clearly dose-related
- * teratogenicity

TABLE 1: PHARMACOKINETIC PROPERTIES OF ANTICONVULSANTS;

Drug	Starting daily dose (mg)	Mean daily dose (mg/kg)	Usual daily dose interval	Time to steady state (days)	Therapeutic range (mg/L)	Serum half life (hours)	Protein binding (%)
Sodium valproate	50-60 (c) 250 (a)	25 (c) 20 (a)	b.d. (occasionally t.d.s.)	2-4	50-100	4-14 (c) 7-17 (a)	90-95
Carbamazepine	100-200	15-20 (c) 10-20 (a)	b.d. (occasionally t.d.s.)	3-6	4-12	15-16 (c) 10-18 (a)	67-81
Phenytoin Sodium	200 (standard) 900 (loading)	5-15 (c) 5 (a)	once a day (occasionally b.d.)	5-10	10-20	4-11 (c) 22-40 (a)	65 (infant) 88-92 (a, c)
Ethosuximide	250	15-40 (c) 15-30 (a)	b.d. (occasionally t.d.s.)	6 (c) 12 (a)	40-100 (c) 50-60 (a)	30 (c) 30-60 (a)	0
Phenobarbitone	30-60	2-4	once a day	16-21	10-35	50 (c) 50-96 (a)	50
Primidone	50-25	10	b.d.	1-5	4-2	16	0-20
Clonazepam	0.25-0.5 (c) 1.0 (a)	0.1	Once at night			20-30 (c) 20-60 (a)	85

Key: (a) = adults, (c) = children

Therapy with phenytoin, carbamazepine and barbiturate may result in a beneficial side effect. Use of these anticonvulsants is associated with a rise in the blood of high-density lipoprotein (HDL), which has an inverse relationship with mortality from ischaemic heart disease. A 29% reduction in mortality from this cause in epileptics treated with these, but not with other antiepilepsy drugs has been suggested by a case control study (Muvronen A et al. Br Med J 1985, 291: 1481).

Drug Interactions

These are a common problem with anticonvulsants, they can interact with food, alcohol, other anticonvulsants and other drugs. One must be aware of the clinically more important interactions; several of these are listed in Table 3. Phenytoin sodium, probably because of its virtually saturated elimination capacity at therapeutic concentrations, is particularly vulnerable to interactions which inhibit its metabolism.

It must be emphasised, however, that not all of the interactions listed in Table 3 occur consistently, possibly because in

some cases they involve the sum of two processes:

- an induction of drug-metabolising capacity by one drug leading to diminished plasma concentrations of another; and
- competition for the induced metabolic pathway between the two drugs leading to raised plasma concentrations of both.

An interaction arising from the introduction or change in dosage of another drug and resulting in raised plasma concentrations (and hence pharmacological effect) of an antiepileptic drug is often first suspected when toxicity develops in a patient who has been taking a constant dosage for some time without previous untoward effect. Falling antiepileptic drug concentrations as a result of an interaction are less likely to be suspected until control of epilepsy deteriorates or plasma drug concentrations are measured.

STARTING TREATMENT

Although in some patients the diagnosis of epilepsy may be sufficiently clear-cut for the general practitioner to

TABLE 2: SIDE EFFECTS OF ANTICONVULSANT DRUGS

Drug	Idiosyncratic	Intoxication	Chronic	Pregnancy	Monitoring
Carbamazepine	rash, jaundice, oedema, aplastic anaemia	diplegia, unsteadiness, dizziness, drowsiness, nausea	hyponatraemia	Probably tetragenic	FBC (frequently initially), LFTs
Sodium valproate	rash, blood dyscrasia, acute hepatotoxicity, acute pancreatitis, acute thrombocytopenia	sedation, unsteadiness, behaviour change, nausea, vomiting	sedation, behaviour change, tremour, bleeding disorder, weight gain, hair loss, hyperaminoacidaemia, false positive urine test for glucose	Tetragenic	LFTs (early) FBC
Phenytoin sodium	rash, blood dyscrasia, immunological reactions	unsteadiness, ataxia, nystagmus, sedation, nausea, headache, behaviour change	sedation, intellectual blunting, mood change, behaviour change, cerebellar syndrome, metabolic bone disease, folate deficiency, hirsutism, gum hypertrophy, acne, connective tissue changes (e.g. skin thickening)	Tetragenic	FBC (regularly) Drug level, vitamin B12, folate if macrocytic
Ethosuximide	rash, blood dyscrasia, abnormal LFTs, precipitate grand mal	unsteadiness, ataxia, drowsiness, nausea, headache	headache, behaviour change	Not known	FBC (regularly) LFTs (occasionally)
Phenobarbitone	rash, blood dyscrasia	sedation, unsteadiness, headache, nausea, behaviour change	sedation, behaviour change, mood change, intellectual blunting, metabolic bone disease, connective tissue disorders	Not known folate if	CBC, Ca ⁺⁺ , Vitamin B12, macrocytic
Primidone	acute dizziness, unsteadiness, nausea, on initiation of therapy, rash, blood dyscrasia	as for phenobarbitone	as for phenobarbitone	Not known	as for phenobarbitone
Clonazepam	psychiatric problems (aggression), behaviour disturbance	sedation	dependence	Not known	

commence therapy, all patients should be referred for specialist assessment to allow appropriate investigation and counselling, particularly regarding prognosis. Generally single seizures should not be routinely treated; the proportion who, if left untreated, will have a second fit within 1-2 years of a first unprovoked fit is about

20-30%. Where two or more unprovoked episodes have occurred within a short interval, antiepileptic therapy is usually indicated. Even where seizures occur in close temporal relationship, the identification of specific precipitating factors may make it more important to counsel patients than to commence drug therapy. The commonest

TABLE 3: DRUG INTERACTIONS WITH ANTICONVULSANTS

Drug	Drugs interacting	Effect
Phenytoin sodium	carbamazepine, alcohol, folic acid, pyridoxine, tolbutamide, dizoxin	Lowered serum levels of phenytoin
	sulphonamide, cimetidine, isoniazid, oral anticoagulants, dextropropoxyphene, benzodiazepines, phenothiazines, tricyclic antidepressants, chloramphenicol, phenylbutazone, sulthiame	Raised serum levels of phenytoin
	phenobarbitone, sodium valproate	May increase or decrease serum levels of phenytoin
Carbamazepine	oral contraceptive pills, oral anticoagulants, steroids, folic acid, doxycycline, folic acid, theophylline, thyromine, pyridoxine	Decreased levels/ effectiveness of these drugs
	phenytoin, phenobarbitone, primidone	Lowered serum levels of carbamazepine
	erythromycin, cimetidine, isoniazid, dextropropoxyphene, verapamil, nicotamide	Raised serum levels of carbamazepine
Sodium valproate	oral contraceptive pill, oral anticoagulants, doxycycline	Decreased levels/ effectiveness of these drugs
	carbamazepine, phenytoin, phenobarbitone, primidone	Lowered serum levels of sodium valproate
	salicyclates	Raised serum levels of sodium valproate
Ethosuximide	carbamazepine	Lowered serum levels of ethosuximide
	phenytoin, sodium valproate	Raised serum levels of ethosuximide
Phenobarbitone	oral anticoagulants, chloramphenicol, thioridazine	Lowered serum levels of phenobarbitone
	phenytoin, sodium valproate, sulthiame	Raised serum levels of phenobarbitone
	cimetidine, oral anticoagulants, folic acid, griseofulvin, doxycycline, digoxin, chloramphenicol, oral contraceptive pills, noctripilone	Decreased levels/ effectiveness of these drugs

example are photosensitive epilepsy and alcohol-withdrawal fits. In treating the former, advice regarding viewing the television from a distance and using a remote control may be sufficient. A simple approach to decision making on starting therapy is summarised in Table 4.

Once a decision has been made to start antiepileptic treatment the chosen drug should be used in monotherapy — there is no place for starting a patient on two or more anticonvulsant drugs. The chosen antiepileptic should be introduced gradually in small doses, since rapid introduction may induce side effects (particularly in the case of carbamazepine). Start with about one-third of the expected maintenance

TABLE 4: STARTING ANTIEPILEPTIC TREATMENTS

Problems	Usual practice	Modifying factors
Single seizure (Clinically diagnosed)	No treatment	Progressive cerebral disorder Clearly epileptic EEG
Two or more fits (Clinically diagnosed)	Monotherapy	Seizures widely separated in time (e.g. more than 1 year) Identified precipitating factor(s) (e.g. drugs, alcohol) Probability of poor compliance (e.g. personality disorder, alcoholic)

dose and increase it weekly to reach the maintenance dose in 3 to 4 weeks, by which time any enzyme induction will have occurred and a steady state reached after the most recent dose increment. In the case of phenytoin sodium a substantial loading dose may be given, followed by the lower maintenance dose. If plasma concentration measurements are available, a blood sample, taken at the end of the longest interval between doses i.e. through or minimal concentration (usually, and inconveniently, early in the morning) provides useful background information for further dose adjustment.

If fits continue to occur the dose should be adjusted upwards until fits cease or adverse effects occur. The importance of compliance should be emphasized to the patient; poor compliance is the commonest cause of treatment failure.

CHOICE OF DRUG

This depends on the type of seizure to be managed. General recommendations are outlined in Table 5. In many patients the efficacy of the different anticonvulsant drugs may be approximately equivalent, and their relative toxicity may be determines which drug is used.

TABLE 5: ANTICONVULSANT DRUGS FOR DIFFERENT SEIZURE TYPES

Seizure type	Drugs of choice
Typical absences (petit mal)	Sodium valproate Ethosuximide
Waking tonic-clonic (grand mal)	Sodium valproate Carbamazepine Phenytoin sodium
Tonic-clonic with typical absences	Sodium valproate
Partial (temporal lobe, etc)	Carbamazepine Phenytoin sodium Sodium valproate
Myoclonic	Sodium valproate Clonazepam

Phenytoin sodium is a powerful and effective anticonvulsant for all seizure types except petit mal. But it is a difficult drug to use because its dose-serum level

relationship is non-linear and varies considerably between individuals, and because of its narrow therapeutic index. Furthermore phenytoin has the great disadvantage of causing gum hypertrophy, abnormal facial growth and excess body hair and it can interfere with vitamin D metabolism growth in children. It is now less often in paediatric practice and it also best avoided as a drug of first choice in young women not only because of its cosmetic side effects but also because of its teratogenicity.

Sodium valproate is a good general purpose anticonvulsant, and is particularly effective in controlling both petit mal absence and tonic-clonic grand mal seizures, so it may be the drug of choice in young people suffering from both forms of epilepsy, which is not uncommon. It is also effective in juvenile myoclonic epilepsy and in grand mal in adults. Attention and alertness is often improved in children transferred from ethosuximide to sodium valproate, so some authorities now prefer sodium valproate to ethosuximide in petit mal. Its use in children should be cautious because of the risk of hepatotoxicity. Sodium valproate should also be avoided in pregnancy because of risk of teratogenicity.

Carbamazepine is an increasingly used anticonvulsant as it does not have the unsatisfactory cosmetic side effects of phenytoin sodium. The efficacy of carbamazepine in grand mal and partial seizures is equivalent or superior to that of phenytoin and phenobarbitone, and its toxicity is significantly less than that of any other first line anticonvulsant drug. Common dose-related side effects, like giddiness and diplopia, are usually transitory, occurring 1 to 3 hours after taking the drug. If this happens, smaller individual doses taken 3 or more times a day, but with the same total daily dose, may eliminate the side effects. Carbamazepine (in contrast to barbiturates) causes much less impairment of mood and cognitive ability in children with chronic epilepsy, and reduction in fit frequency may result in actual improvement in behaviour and learning. Some authorities believe that carbamazepine has a positive psychotropic effect in adults and children.

Phenobarbitone is being rapidly superseded as an anticonvulsant because of its sedative and toxic effects. Its anticonvulsant efficacy is equivalent to that of phenytoin and carbamazepine and its use is now often restricted to patients who have not responded to other first line drugs. Phenobarbitone is relatively contraindicated in children because it often makes them irritable and hyperactive and sometimes unmanageable. It is, however, still widely used in the newborn because there is less experience of using other drugs at this stage of life.

Primidone is largely metabolised to phenobarbitone; whether primidone itself conveys any anticonvulsant effect additional to that of the derived phenobarbitone remains controversial. Primidone appears to cause less irritability than phenobarbitone.

Ethosuximide is used only in classical petit mal seizures. It can increase liability to grand mal seizures. If it fails to control petit mal absences or if grand mal seizures supervene, ethosuximide may be replaced with sodium valproate or prescribed in combination with phenytoin sodium.

Clonazepam, a long-active benzodiazepine, tends to be reserved for more obscure and difficult forms of seizures (e.g. photosensitive epilepsy, complex partial seizures, resistant tonic-clonic seizures, and status epilepticus-given intravenously), and so far has not been sub-Mect to large scale use as first-line drug. Routine use of clonazepam is limited severely by its tendency to cause drowsiness, psychiatric problems (aggression) and behaviour disturbances in children.

MAINTENANCE TREATMENT

About 70-80% of treated patients quickly enter a prolonged remission phase. Maintenance regimens vary from patient to patient. The aim is to achieve and maintain the lowest dose of an appropriate drug which permits complete seizure control without undue toxic effects. In many patients, seizures are controlled with levels below the normal therapeutic range; in these cases, it is incorrect to increase the dose of the drug simply to achieve

therapeutic levels.

If optimum level of a single first-line drug does not control seizures, or if unacceptable side effects occur, the initial drug should be substituted with another first-line anticonvulsant. If the second drug does not control seizures, monotherapy with a third anticonvulsant or combination therapy with two first-line drugs should be tried. The prognosis is significantly poorer for those requiring combination therapy.

If single drug treatment is ineffective, there are often unrecognised complicating factors:

- * compliance may be poor (the commonest reason)
- * prescription of a drug inappropriate for the type of seizure
- * failure to determine the plasma levels despite inadequate control
- * psychological problems may be present
- * seizures may be non-epileptic
- * a progressive neurological condition (e.g. tumour) may underlie the epilepsy

These factors should be explored first.

DRUG LEVEL MONITORING

Routine monitoring of plasma concentration is not necessary and wasted expensive resources. Measuring plasma anticonvulsant drug levels is useful:

- * when using drugs where absorption or metabolic capacity are variable or unpredictable
- * where there is a low therapeutic index
- * where toxicity can be hard to recognise clinically
- * where overdosage can lead to fits
- * as a check on compliance.

Monitoring plasma drug concentrations is not likely to be useful where half-life is short, where the therapeutic effect does not closely match the plasma concentration or where receptor tolerance develops. Monitoring is particularly useful and important in the case of phenytoin sodium because the dose-serum level relationship is non-linear and varies considerably between individuals, and because of its

narrow therapeutic index. Drug level monitoring is seldom really useful with most of the other anticonvulsants, except in detecting non-compliance and severe overdosage.

STOPPING TREATMENT

The question of when to withdraw anticonvulsant treatment in patients whose seizures have ceased is difficult. In no individual case can be safety of drug withdrawal be guaranteed, and this should always be made clear to the patient.

The risk of relapse on withdrawal of medication in patients who have been seizure free for 2 to 3 years is probably 20 to 50%, but a number of factors influence this risk. The longer the period of freedom from seizures the lower the risk of relapse on withdrawal of the anticonvulsant. If recurrent of seizures does occur, it is likely to be at the time the drug is being withdrawn, or shortly after withdrawal is completed; the longer the patient is free of attacks after the withdrawal period, the less likely is further recurrences. Some guidelines for the discontinuation of therapy are summarised in Table 6.

TABLE 6: STOPPING ANTI-EPILEPTIC TREATMENT

Absolute requirement	Factors in favour	Factors against
Two to three years free of all seizures	Primary generalised epilepsy Absence of cerebral disorder	Late onset epilepsy Partial epilepsy
Patient's informed agreement	Short duration of epilepsy Normal EEG Non-driver	Cerebral disorder Long duration of epilepsy Abnormal EEG Driver

Withdrawal should be carried out very slowly in staged decrements — monthly dosage decrements over a minimum period of six months may reduce relapse rates. If a fit occurs, full therapy must be resumed again for 2-3 years. Sudden withdrawal may result in status epilepticus and patients must be counselled about the need for compliance.

EMERGENCY DRUG TREATMENT OF SEIZURES

In epileptics already receiving treatment, emergency drug treatment is not usually necessary after a single epileptic attack. If a series of seizures occur, however, or if a single epileptic is usually followed by a series or cluster, the intravenous or rectal administration of diazepam will often prevent further seizures. Oral diazepam is absorbed too slowly to be of any use in the emergency situation, and intramuscular diazepam should never be used as absorption is very slow and incomplete.

A useful proprietary rectal solution of diazepam, ready made up with a soft rectal catheter, is easily administered by parents or relatives. Alternatively, the normal intravenous preparation may be successfully instilled rectally using an ordinary syringe and any soft plastic cannula. Wax diazepam suppositories are available, but are absorbed too slowly for emergency use.

Status epilepticus is commonly preceded by increasing serial seizures, and it is an important, but little realised, fact that the early use of rectal or intravenous diazepam in serial seizures will markedly cut down this risk.

If diazepam is not available, or is contraindicated, paraldehyde given by deep intramuscular injection into the buttocks may be highly effective. It is a very painful injection and may result in a sterile abscess. The injection site should be well away from the course of the sciatic nerve, as contact with the sciatic nerve will result in a severe causalgic syndrome. Paraldehyde may also be given rectally.

ANTICONVULSANTS AND PREGNANCY

Untreated maternal seizures are considered a greater risk to the foetus than well-controlled anticonvulsant medication, which is therefore not withdrawn when pregnancies are planned, unless the risk of further fits is thought to be very low. There is well-documented increase

(roughly a doubling) in the incidence of foetal malformation in babies exposed to phenytoin in utero (often distressing cranio-facial defects such as harelip, microcephaly and mental retardation). Carbamazepine has also recently been associated with retardation of foetal head growth, and sodium valproate with spina bifida. It is not certain that these effects are entirely due to the antifolate action of many anticonvulsants, but folic acid supplementation in women on anticonvulsants and who are pregnant or likely to become pregnant is considered necessary. This is unlikely to result in recurrent fits, and is preferable to withdrawing or changing anticonvulsants.

Anticonvulsant requirements increase during pregnancy and fall after delivery, so plasma levels need to be checked more frequently and dosages adjusted accordingly. The requirement during pregnancy is increased by greater maternal hepatic enzyme activity, and later, to a small extent, by addition of the foetal liver. Postpartum drug requirement usually falls rapidly to the non-pregnant state, so toxicity can easily occur at that time. Secretion of anticonvulsants into breast milk is negligible except in the case of ethosuximide, and even then it is seldom significant (i.e. it does not reach toxic levels) even though the neonatal liver metabolises drugs slowly.

Newborn babies of mothers taking antiepileptics sometimes have reduced clotting factors, prothrombin, etc, remediable by giving Vitamin K antenatally; it is attributed to the anticonvulsants, perhaps by enzyme induction.

ANTICONVULSANTS AND CONTRACEPTION

Anticonvulsants, with the exception of sodium valproate and clonazepam enhance the metabolic degradation of the low-dose oestrogen-containing combined oral contraceptive pills, with possible contraceptive failure. Thus it is prudent either to prescribe the higher dose oestrogen-containing oral contraceptive (at a level that avoids breakthrough bleeding e.g. 50 mg oestrogen) or, perhaps better, use a different mode of contraception.

ANTICONVULSANTS AND LIVER DISEASE

Hepatocellular disease will both show metabolism and be associated with hypoalbuminaemia. Hypoalbuminaemia may lead to toxicity at normal total plasma levels (remembering that it is the unbound fraction that is active). Salivary levels may be more appropriate in such patients, at least for those drugs where salivary levels are reliable — phenytoin, carbamazepine and ethosuximide. Portosystemic shunting in liver disease (either via varices or a surgical shunt) may increase drug bioavailability by reducing first-pass metabolism. For all these reasons the right dose will be smaller and given at longer intervals.

ANTICONVULSANTS AND RENAL DISEASE

Plasma protein binding may be reduced in renal failure, and albumin levels reduced, so that the unbound fraction is increased, and the upper therapeutic range therefore reduced. The half-life of drugs excreted by the kidney (e.g. phenobarbitone) is prolonged.

EPILEPSY IN CHILDREN

There are many discrete forms of childhood epileptic seizure disorders, which vary as the child matures. Epilepsy in children are treated as in adults, but children may respond differently and become irritable, e.g. with sodium valproate or phenobarbitone.

It remains uncertain whether antiepileptic drugs interfere with development and education and it is certainly unwise to assume they do not. The sensible course is to control the epilepsy with minimal doses of a single drug and attention to precipitating factors, with drug withdrawal when it is deemed safe to attempt it.

GENETIC COUNSELLING

Two questions often arise. The first and most common is asked by people with idiopathic or acquired epilepsy who are contemplating starting a family: what is the chance of a child of theirs developing epilepsy? The second comes from normal parents of an epileptic child, who want to know the probability of further children

being affected. Some relevant observations concerning major seizures are:

- * the prevalence of epilepsy in the general population is about 0.75%
- * if one parent has epilepsy, the incidence in children is about 2%-3%
- * if both parents have epilepsy, the incidence rises to 25%
- * if one parent has epilepsy and the other an "unstable" EEG, then the risk may be intermediate
- * risk is also increased in children of people with some types of acquired epilepsy (e.g. post-traumatic or following febrile convulsions)
- * a family history of fits (in first-degree relations) is often found in children with febrile convulsions (50%) or fits generally (30%).

A reasonable interpretation of these observations would be that a postulated tendency to seizures is inherited polygenically, at least some of the genes being recessive. Such a tendency may in some instances never be expressed as fits except under provocation (e.g. fever in infancy, head injury, alcohol or metabolic insult at any age). Some authorities believe this postulated tendency can be detected as EEG instability.

For petit mal the picture is more clear-cut, as about 40% of sibs of children with petit mal also have a spike-and-wave abnormality, suggesting an autosomal dominant inheritance. Certain of the neurological syndromes with associated epilepsy are also inherited as autosomal dominants (e.g. tuberosc sclerosis, neurofibromatosis,

Huntingtons disease), although formes frustes are common in the first two examples.

Other neurological syndromes with epilepsy are inherited as autosomal recessives (e.g. lipidosis, galactosaemia, mucopolysaccharidoses). Here, what is usually called for is advice after birth of an affected child, concerning the risk to further children, which is likely to be about 25%.

CONCLUSION

Close co-operation is necessary between the patient, family doctor and physician or neurologist. The aim of a treatment in epilepsy is to achieve and maintain a dosage of an appropriate drug adequate to prevent attacks without undue toxic effects. The choice of drug depends on the type of epilepsy.

REFERENCES

1. Chadwick D. Management of adult epilepsy — an overview. *Prescriber's Journal* Vol 28 No 4, August 1988, pg 130-138.
2. Hall JI. The falling sickness Update Vol 32 No 9, 1 May 1986, pg 782-794.
3. Laurence DR, Bennett PN (eds) (1987) *Clinical Pharmacology*, Churchill Livingstone, Edinburgh.
4. Parkes D, et al (eds) (1987) *Treatment in Clinical Medicine — Neurological Disorders*, Springer-Verlag, London.
5. Ross E. Epilepsy in childhood *Medicine International* Vol 2 No 46, Oct 1987, pg 1879-1883.
6. Sharon S. Management of epilepsy in adults *Medicine International* Vol 2 No 46, Oct 1987, pg 1874-1879.
7. Vadjia FJE. Drug Treatment of Epilepsy *Australian Family Physician* Vol 13 No 6, June 1984, pg 431-434.

MULTIPLE CHOICE QUESTIONS

1. In the treatment of epilepsy with drugs:
 - A a single daily dose of primidone can provide effective blood-levels throughout the 24 hours.
 - B phenytoin is the drug of first choice for petit mal.
 - C carbamazepine has a relatively short half-life and a single daily dose is not satisfactory.
 - D phenobarbitone in correct dosage has no important side-effects.
 - E if phenytoin is to be given to a woman who is taking an oral contraceptive the dose of the latter must be reduced.
2. In the long-term treatment of grand mal epilepsy:
 - A carbamazepine is effective.
 - B there is a direct and constant correlation between plasma phenytoin levels and the dose of the drug.
 - C clonazepam is effective.
 - D in children, sodium valproate should not be prescribed.
 - E phenobarbitone does not cause sedation.
3. Which of the following statements are true?
 - A phenobarbitone can induce its own metabolism.
 - B primidone is metabolised to phenobarbitone.
 - C sodium valproate is effective only for petit mal.
 - D phenytoin can cause gum hyperplasia and ache.
 - E chlorpromazine is useful as chronic therapy for grand mal epilepsy.
4. Sodium valproate
 - A is a drug of first choice for petit mal absences.
 - B should not be used for petit mal with tonic-clonic seizures.
 - C is likely to cause ache.
 - D may cause alopecia.
 - E should not be given to pregnant women.
5. Phenytoin
 - A is eliminated exclusively according to first-order kinetics.
 - B enhances its own metabolism during continued treatment.
 - C has the same plasma half-life at all doses and plasma concentrations.
 - D is highly protein-bound.
 - E is virtually free of acute toxic effects.
6. The following are recognised side effects of the anticonvulsants named:
 - A megaloblastic anaemia with phenytoin.
 - B acute thrombocytopenia with sodium valproate.
 - C hyperactivity with phenobarbitone.
 - D hyponatraemia with carbamazepine.
 - E cerebellar dysfunction with phenytoin.
7. The following statements are correct:
 - A phenytoin may enhance the metabolic degradation of warfarin.
 - B the metabolism of carbamazepine may be inhibited by erythramycin.
 - C sodium valproate may enhance the metabolic degradation of the low-dose oestrogen contraceptive pill.
 - D phenobarbitone in combination with sodium valproate can result in severe sedation.
 - E alcohol can render phenytoin less effective.
8. As regards anti-epileptic therapy used in pregnancy:
 - A there is a 2-3 fold increase in incidence of birth defects if used in early pregnancy.
 - B phenytoin is the drug of first choice.
 - C more than 90% of epileptic mothers bear normal children.

- D may precipitate serious haemorrhage in the first 24 hours of the neonate .
- E breastfeeding by mothers on anti-epileptic therapy is absolutely contraindicated.
9. The following may be used in the treatment of status epilepticus :
- A intramuscular diazepam .
 - B intramuscular paraldehyde .
 - C intravenous chlormethiazole .
 - D intramuscular chlorpromazine .
 - E intravenous clonazepam .

10. Carbamazepine

- a is ineffective in the treatment of temporal lobe epilepsy .
- B should not be given more than once daily .
- C serum levels can be elevated by cimetidine .
- D does not interact with warfarin .
- E is an ideal drug for the treatment tonic-clonic (grand mal) seizures in Women and adolescents.

ANSWERS

- | | |
|----------|--------------|
| 1. A C | 6. A B C D E |
| 2. A | 7. A B D E |
| 3. A B D | 8. A C D |
| 4. A D E | 9. B C E |
| 5. B D | 10. C E |

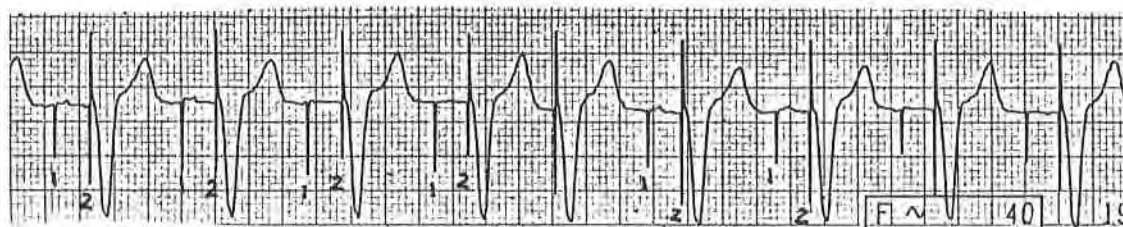
ECG QUIZ

Contributed by Dr Baldev Singh, MBBS (S'pore), M Med (Int Med), MRCP (UK)

In this era of high technology one of your patients may have a rhythm strip shown below. What is your diagnosis and comments?



ANSWER TO ECG QUIZ



The rhythm strip belongs to a patient in whom a dual chamber sequential pacemaker has been implanted. The vertical lines are pacing spikes. The pacing spikes occur in pairs labelled 1 and 2. The first spike is generated by the pacemaker lead implanted in the atrial wall and the second spike occurring at a first interval of 0.2 sec. later is generated by the ventricular lead.

Both leads can sense an intrinsic beat and are also inhibited by this and both leads can stimulate their respective chambers sequentially. Thus, if the atrium contracts spontaneously a normal P wave is generated and the atrial lead is inhibited. If this is conducted normally through the AV node and stimulate the ventricles to contract then the ventricular lead is also inhibited and a normal sinus beat occurs. If however the atrium fails to contract for a fixed period of time then the atrial lead paces the atrium producing an atrial spike on the ECG. If this is not followed by a normal ventricular contraction within 0.2 sec. via normal conduction through the AV node, then the ventricular lead stimulates the ventricle producing a ventricular spike and a QRS complex.

In this way the dual chamber pacemaker helps to preserve atrio-ventricular synchrony, i.e. atrial contraction precedes ventricular contraction in a physiological manner. This ensures better ventricular filling and hence better cardiac output. This would make a significant difference to the physical well being of the patient in certain situations. The pacemaker can be programmed in various ways using an external programmer. The more sophisticated pacemakers are rate responsive, i.e. they can automatically vary the rate of the pacemaker to suit the different activity levels of the patient.

P.S. The 5th complex has no atrial pacing spike. The atrial lead has probably sensed a P wave which is buried in the peak of the preceeding T wave and this has inhibited the atrial lead.



NEW BOOK ANNOUNCEMENT

Minor and Trace Elements in Breast Milk

Report of a Joint WHO/IAEA Collaborative Study

World Health Organization, 1989
xi + 159 pages (available in English; French and Spanish in preparation)
ISBN 92 4 156121 1
Sw.fr. 30.—/US \$24.00
Order no. 1150311

This book is the third and final volume in a series of reports stemming from a multinational study on breast-feeding*. Responding to inconsistencies in the published data on concentrations of trace elements in breast milk, the study analysed milk samples collected from women in Guatemala, Hungary, Nigeria, the Philippines, Sweden and Zaire in an effort to obtain reliable data on the quantities of minor and trace elements present in breast milk. The study was also designed to determine whether the concentrations of these elements varied significantly with the socio-economic group, geographical origin, or nutritional status of the mothers.

The 24 elements selected for study included all the known essential trace elements, with the exception of silicon, and some important toxic trace elements. Calcium, chlorine, magnesium, phosphorus, potassium and sodium were also included in the analysis.

The book opens with a brief explanation of the biochemical functions of trace elements and the association of deficiencies with various disorders of growth and development. The

second chapter explains the careful design of the study, which used standardized procedures for the collection of samples, a single reference analytical laboratory for each element, and appropriate analytical reference materials. Results are then presented for total dry matter and for antimony, arsenic, cadmium, calcium, chlorine, chromium, cobalt, copper, fluorine, iodine, iron, lead, magnesium, manganese, mercury, molybdenum, nickel, phosphorus, potassium, selenium, sodium, tin, vanadium, and zinc. For each element, information is given on any observed differences between study areas, variations in samples between urban and rural areas, and variations in concentrations according to time of year.

A discussion of findings compares results of the study with data from the literature, assesses sources of variation in the elemental composition of human milk, and compares the observed daily intake of minor and trace elements with recommended intake. The book concludes that when minor and trace elements are determined under similar conditions in the breast milk of groups of mothers living in different parts of the world, environmental conditions play a major role in determining concentrations. Findings from the study should also prove useful in the formulation of recommendations regarding levels of trace elements in the diet of infants.

*Previous titles:

Contemporary Patterns of Breast-feeding (WHO, 1981)

The Quantity and Quality of Breast Milk (WHO, 1985)

42ND INTERNATIONAL CONGRESS ON GENERAL PRACTICE

organised by

**the Societas Internationalis Medicinae Generalis (SIMG) —
International Society of General Practice
September 10-15, 1990, Klagenfurt (Austria)**

Monday: HEALTH PROMOTION
Tuesday: NEUROLOGICAL PROBLEMS IN GENERAL PRACTICE
Wednesday: ADOLESCENT'S CARE
Thursday: RESEARCH IN GENERAL PRACTICE
**Friday: EFFECTIVENESS IN PRIMARY HEALTH CARE, COST
AND CONSEQUENCES**
Saturday: GENERAL PRACTICE IN THE INNER CITIES

Congress languages: Monday, Tuesday, Wednesday: GERMAN
Thursday, Friday, Saturday: ENGLISH-GERMAN
simultaneously

Head of congress: OMR Dr Gottfried HELLER, FRCGP
A-9020 Klagenfurt, Bahnhofstrasse 22, Austria
Phone: (0463) 55449

**FIRST
ANNOUNCEMENT**



WONCA REGIONAL CONFERENCE ASIA PACIFIC REGION

June 24-28, 1990, Bali - Indonesia

theme

**FAMILY PRACTICE TOWARDS THE YEAR OF 2000:
PROSPECTS AND CHALLENGES**



Organized by:
The Indonesian College of Family Physicians

REGISTRATION FEE

	before 1 March 1990	after 1 March 1990
Participant	US\$275	US\$325
Accompanying person	US\$150	US\$175

ACCOMMODATION

- Pertamina Cottage (Congress venue)
- Surrounding Hotels

Please contact our College Secretariat Tel: 2230606 (Singapore) for further details.

ARCALION

2 tablets with breakfast

Treatment of all forms of asthenia

Arcalion 200 is the only true antiasthenic agent suitable for all tired patients, and this is because Arcalion 200 presents 3 major advantages:

- its versatile efficacy,
- its rapid, clearly demonstrated action,
- its simple and practical dosage.

The uniquely versatile action of Arcalion ensures its efficacy in all forms of asthenia

By selectively binding to neuronal structures specifically related to fatigue, such as the reticular formation, Arcalion 200 acts on all of the manifestations of asthenia, regardless of the cause.

Arcalion is the treatment for all types of functional asthenia whether physical, psychological or intellectual.

The rapidity of action of Arcalion has been clearly demonstrated

in an international clinical dossier of more than 1,300 cases, including 354 cases studied under double-blind conditions versus a reference product.

The value of Arcalion has been demonstrated in the following specific indications:

- post-infectious asthenia,
- physical asthenia (asthenia in athletes),
- sexual asthenia,
- asthenia during the post-partum period,
- asthenia associated with colonic disorders,
- asthenia associated with slimming,
- rehabilitation of coronary patients,
- withdrawal in alcoholics and drug addicts,
- asthenia in students.

An additional practical advantage of Arcalion is its simple and practical dosage

- 2 tablets every morning with breakfast in the majority of patients,
- in the adult with severe asthenia Arcalion's excellent acceptability allows to repeat the dosage 2 to 3 times a day.

Arcalion may also be used without hesitation in "fragile" patients, during pregnancy, in children (1 tablet daily from the age of five years, 2 tablets daily from the age of ten years) as well as in elderly patients.

ARCALION[®]200



Pharmacokinetics: Arcalion 200 is completely and rapidly absorbed. Peak plasma concentrations are attained after 45 minutes. The half life is 5 hours. Elimination starts after 2 hours; there is no accumulation. **Contra-**

indication: Hypersensitivity to sulbutiamine. **Overdos-**
age: In case of overdose, restlessness, euphoria and tremor may appear. These symptoms are transient and leave no sequelae. **General acceptability:** The therapeutic action of Arcalion 200 manifests progressively and there is no

stimulant effect. The action is sustained and increases with continuing treatment without development of dependence. The administration of Arcalion 200 is consistent with car driving and operating machinery. Laboratory results have remained normal in all clinical trials. Arcalion 200 can be combined with any other treatment. **Composition and presentation:** Arcalion has the following substance as its active principle: N,N'-[Dithiobis[2-(2-hydroxyethyl) 1-methylvinylene]] bis [N [(4-ami- no 2-methyl-5-pyrimidinyl)methyl]formamide]diisobutyrate(ester) or sulbutiamine. Arcalion 200: box of 100 tabs: each containing 200 mg of the active substance.

For further information, please write to:
Les Laboratoires Servier.
Gidy, 45400 Fleury-les-Aubrais
Correspondent:
Development International Servier.
24 rue du Pont, 92200 Neuilly-Sur-Seine France

represented by
ASIAMED PHARMACEUTICAL PRODUCTS (S) PTE LTD
421, Tagore Avenue, Singapore 2678. Tel: 4596011
F.E. ZUELLIG (MALAYSIA) SDN. BHD.
11th Floor, Wisma Damansara, Jalan Semantan, Damansara Heights,
50706 Kuala Lumpur, Malaysia
Tel: 2553842

Bactroban[®]

rapid success

- Fast and effective in bacterial skin infections.¹⁻⁵
- Highly active against the causative pathogens.
- Remarkably low incidence of side effects.⁶
- 96% success in a variety of skin infections.¹



PRESCRIBING INFORMATION

Presentation BACTROBAN ointment: mupirocin 2% w/w in polyethylene glycol base. 5g or 15g tubes.

Activity BACTROBAN is a topical anti-bacterial agent, active against those organisms responsible for the majority of skin infections, e.g. *Staphylococcus aureus*, including methicillin-resistant strains, other staphylococci and streptococci. It is also active at concentrations attainable on the skin against Gram-negative skin pathogens such as *Escherichia coli* and *Proteus* spp.

Indications Bacterial skin infections, e.g. impetigo, folliculitis and furunculosis.

Dosage and Administration Adults and children: BACTROBAN should be applied to the affected area up to three times daily, for up to 10 days.

Precautions Avoid contact of BACTROBAN ointment with the eyes. Use BACTROBAN with caution in patients with moderate or severe renal impairment.

Use in Pregnancy: There is inadequate evidence of safety to recommend the use of BACTROBAN during pregnancy.

Contra-indications Hypersensitivity to BACTROBAN or other ointments containing polyethylene glycols. This BACTROBAN formulation is not suitable for ophthalmic or intra-nasal use.

Side-effects Some minor localised effects such as burning, stinging and itching have been reported.

Storage BACTROBAN should be stored at room temperature (below 25°C). Not all presentations are available in every country. Further information is available from Beecham Pharmaceuticals, Brentford, England. BACTROBAN is a trademark.

Beecham
Pharmaceuticals
INTERNATIONAL DIVISION

References 1. Contemporary Dermatology, 1987, **1** (2): 32. 2. Proc. Int. Symp. on Bactroban, Excerpta Medica, 1984, 190. 3. Int. J. Derm., 1987, **26**(7):472.
4. Curr. Ther. Res. Exp., 1987, **41** (1): 114. 5. Proc. Int. Symp. On Bactroban, Excerpta Medica, 4, 141. 6. Roy. Soc. Med. Int. Cong. and Symp. 80, 173.

International Division 001/34/2/14/89



All the pieces fit for complete insomnia therapy.

Hypnotic therapy can often be like a jigsaw puzzle. Because, if the hypnotic you select does not have all the clinical benefits to meet your patients' nighttime and daytime needs, then complete insomnia therapy will never be achieved.

Somese Tablets (triazolam) have all the right pieces. Rapid absorption and an appropriate duration of effect mean patients fall asleep quickly and achieve a full, restful night's sleep. Prompt excretion means patients wake up refreshed and alert in the morning.

In addition, there is no drug accumulation so your patients' activities are usually not impaired.

It's no wonder Somese Tablets are the most widely prescribed hypnotic in the world for complete insomnia therapy.

Somese 0.25mg
(triazolam) TABLETS

**Good nights, good mornings,
good days.**

Availability: Somese Tablets are available as scored tablets of triazolam, 0.25 mg (power blue) in bottles of 100 and blister packs of 500.



Further information is available on request.

Distributed by:

F.E. Zuellig (M) Sdn. Bhd. P.O. Box 10251, Kuala Lumpur, 50708, Malaysia.

The Getz Corp. (S) Sdn. Bhd. P.O. Box 234, Singapore 9004.

8811 TRADEMARK: SOMESE FE 7018.1 M/S

Al
un
yd
dir
pe
lie
ita
dei
nyl
20
ver
se
pa
it-r
er
con
ec
g/c
IF
an
PH
re
are
OS

NEW
FROM ICI

ZESTRIL

lisinopril

An advance in ACE inhibition

Once daily
■
Full 24-hour
control of
hypertension
■
Well tolerated

DESCRIPTION NOTES

Round tablets, containing 5, 10 and 20 mg of lisinopril hydrate coloured pink, pink and red respectively.

Indications

Hypertension and congestive heart failure (CHF). In patients with CHF 'Zestril' is used as an adjunct to digitalis and diuretics.

Dosage

In hypertension the usual effective maintenance dose is 20 mg once daily. 10 mg once daily can be used where a starting dose is appropriate. A lower starting dose (2.5 or 5 mg) is required in patients with renal impairment, renovascular hypertension, volume or salt-depleted patients and some elderly patients. Hereafter dosage should be adjusted according to blood pressure response. The maximum dose used in long-term controlled clinical trials was 80 mg/day. CHF 5 to 20 mg once daily, starting dose 10 mg if 2.5 mg.

Contraindications

Hypersensitivity to any component of this product.

Precautions

Caution is required in volume or salt-depleted patients, those previously treated with diuretics and those

with renovascular hypertension as symptomatic hypotension may occur. In hypertensive patients in whom the diuretic cannot be discontinued initial dose of 'Zestril' should be 5 mg. Hypotension may occur during anaesthesia. Correct by plasma volume expansion. Renal impairment may occur in some CHF patients who experience hypotension on starting 'Zestril'. This is usually reversible. Dosage in renal impairment (and elderly) should be based on creatinine clearance.

Pregnancy

There are no studies in pregnant women. Should only be used if potential benefit outweighs the risk to the fetus. Caution if given to nursing mother. No paediatric experience.

Diuretics potentiate the antihypertensive effect of 'Zestril'. Symptomatic hypotension can be minimised by discontinuing diuretic prior to 'Zestril'. Avoid use of potassium sparing diuretics and potassium supplements with 'Zestril' especially in patients with renal impairment. If used concurrently, frequent monitoring of serum potassium is required. Indomethacin may diminish the antihypertensive efficacy of concomitantly administered 'Zestril'. 'Zestril' ameliorates diuretic-induced hypokalaemia.

Side effects

Mostly mild and transient: dizziness, headache, diarrhoea, fatigue, cough, nausea, rash, hypotension, orthostatic effects, palpitation, chest pain, asthenia. Angioneurotic oedema has been rarely reported. If it occurs, discontinue 'Zestril' promptly. Treatment with antihistamines or adrenaline may be appropriate. Increases in blood urea and serum creatinine, usually reversible, have been seen. Small decreases in haemoglobin and haematocrit have occurred. Hyperkalaemia may occur.

Overdosage

No data. Correct hypotension with plasma volume expansion. 'Zestril' is dialysable.

'Zestril' is a trademark
Further information is available on request



ICI (Singapore) Private Limited
Finlayson House 4 Raffles Quay
Singapore 0104
Telephone 2243811

THE POWER TO STRIKE AT PAIN



Rx1.O.P. **ONCE DAILY**
Oruvail 200
 controlled release ketoprofen

STRIKING PERFORMANCE IN ARTHRITIS

Prescribing Information

Dosage Orally with food, 200mg daily. **Indications** Rheumatological disorders, including osteoarthritis, rheumatoid arthritis, ankylosing spondylitis and acute musculoskeletal conditions; dysmenorrhoea. **Contra-Indications** Recurring history of/ or peptic ulceration; chronic dyspepsia; use in children; in patients sensitive to aspirin or other non-steroidal anti-inflammatory drugs known to inhibit prostaglandin synthetase or with bronchial asthma or allergic disease, severe renal dysfunction. **Precautions** Pregnancy; lactation. Concomitant administration of protein-bound drugs. **Side-effects** Occasional gastro-intestinal intolerance. Very rare gastro-intestinal haemorrhage/skin rashes. **Presentation** 200mg capsules PL0012/0158; 100mg capsules PL0012/0143. Basic NHS cost (Jan. 1987) 28 x 200mg capsules in calendar blister pack 100 x 100mg capsules ORUVAIL is a trade mark. Further information is available from:



RHÔNE-POULENC SINGAPORE PTE LTD 14 CHINBEE ROAD JURONG SINGAPORE 2261

MA/2888