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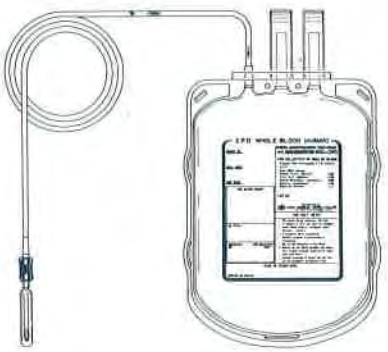

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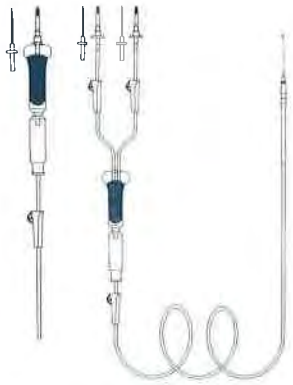

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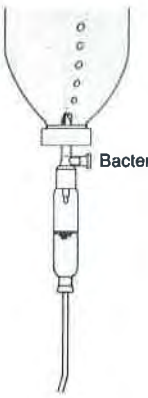

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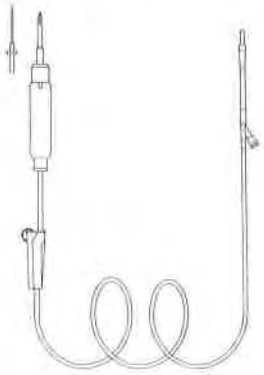

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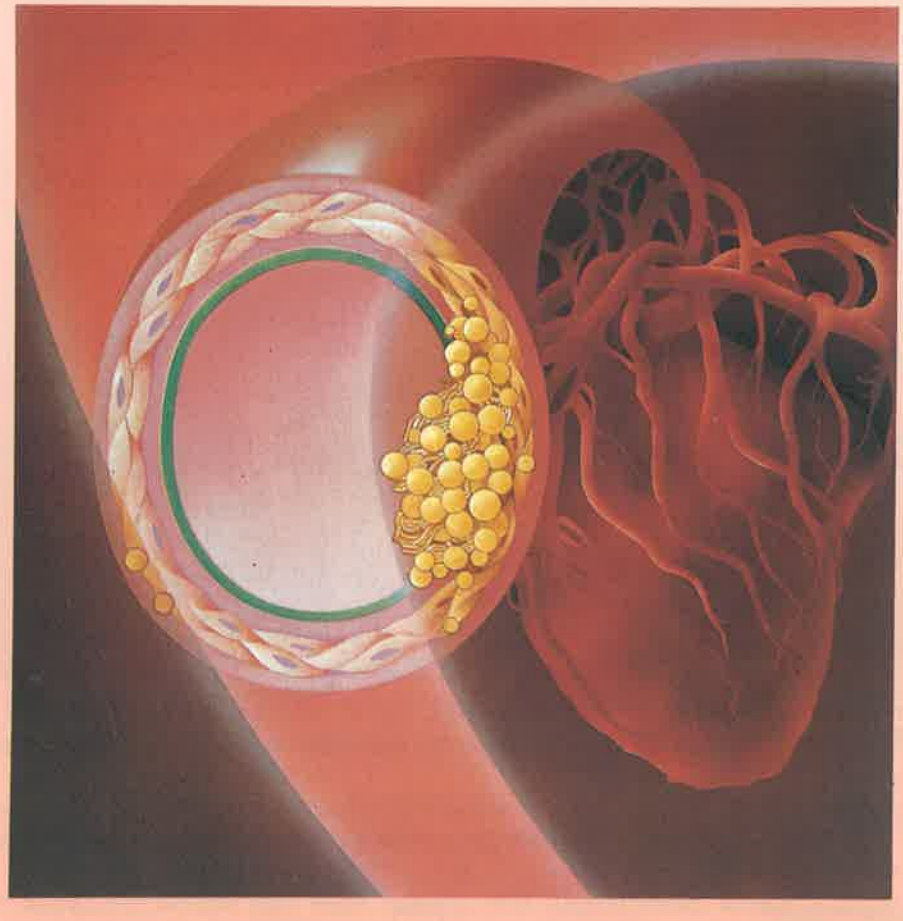

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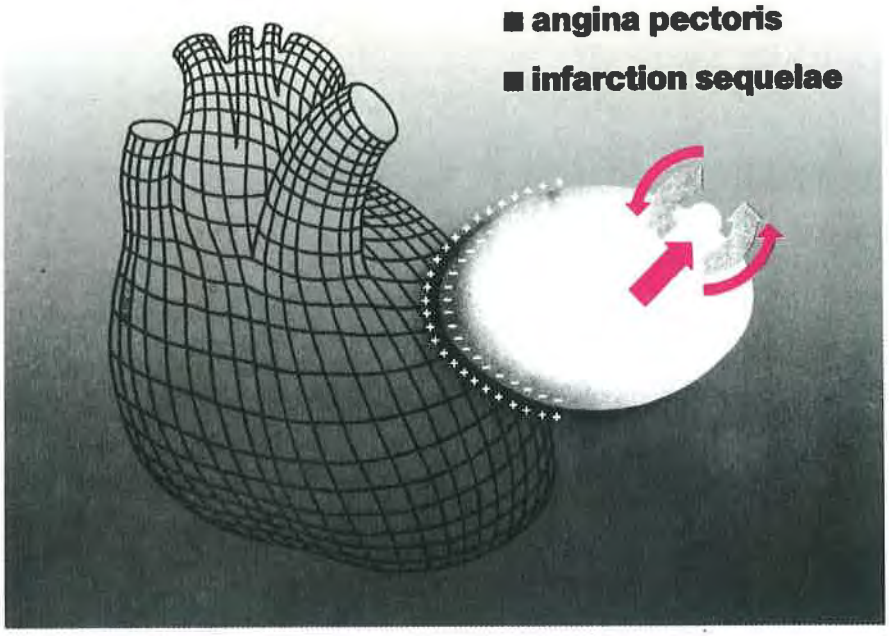

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EDITORIAL

URINARY INCONTINENCE IN THE ELDERLY — A NEGLECTED DISORDER

Urinary incontinence is a prevalent and distressing disorder. However it is commonly neglected by medical practitioners although it is treatable. A clear understanding of the burden and needs of the incontinent subject and his family, developing a logical approach to management, and appreciating the supportive role the family doctor can play will help to redress this neglect.

PREVALENCE

Urinary incontinence ranks amongst the famous four "I"s in geriatric text books, (the "I"s are Intellectual disability, Instability, Immobility and Incontinence). The general practitioner will encounter patients in his clinic, on his home visits, as well as in community homes for the aged. Epidemiological studies show prevalence rates ranging from 3.2%[1] to 42%[2] in the community, and even higher rates in institutions. Initial returns from a local community based study[3] currently underway suggest a prevalence of about 5% amongst the elderly in our community.

THE BURDEN OF INCONTINENCE: IMPACT ON THE PATIENT AND HIS FAMILY

The burden of incontinence is substantial and can be measured in medical, pychosocial and financial terms.

Medically, incontinent individuals are predisposed to perineal rashes, pressure sores, lower limb eczema and cellulitis, urinary tract infections, accidental falls and fractures.

Financially, the treatment of incontinence and its associated morbidity (as above), the use of incontinence aids, and increased laundry all incur expenses — sometimes a family member has to resign from his/her job to care for the incontinent elderly, and this affects family income.

The psychological and social impact on the patient and his carers is the most important of all. The dependant incontinent elderly who is unable to attend to his own private and personal care would suffer an embrassing and humiliating loss of dignity. For the carer, it can be painful and depressing to see a loved one deteriorating and wasting away. Incontinence can disrupt and limit activity, causing isolation and loneliness. The elderly incontinent may not dare to travel, or leave the house to visit friends, for fear of leakage. The carer being so involved in the care of a dependant elderly may not have time for herself. She may even quit her job and curtail her social activities. The family may not welcome visitors to the home because of unhygienic smell. Hence the incontinent elderly and his family or carer are at risk of being cut off from the outside world.

Relationship problems may arise within the family because of incontinence. While some carers may come to terms with the task of caring, others may feel so frustrated with the whole task that they want to give up — they find it emotionally "too draining", physically difficult and unpleasant, especially when there is the lack of support from other family members and lack of advice on the task of caring and information on community resources of help.

The elderly incontinent then is at high risk of being institutionalised, whereby so often, the incontinent is treated with neglect, and left, "to decay". In institutions it is still not uncommon to see dependant elderly suffer the indignity of lying in wet bedding or sitting over an improvised commode through the day.

The needs of the incontinent patient and his family would therefore be counsel, help and relief in the medical, financial, pyschological and social areas.

ATTITUDES, BELIEFS AND MYTHS

Even though incontinence is a distressing problem, it is not uncommon for the affected person and his family to suffer without seeking help. There are two common reasons for this failure to seek help: a pessimistic belief that nothing can be done, and a feeling of shame.

Many elderly people are fatalistic, pessimistic and resigned about their problems, and incontinence is often accepted as inevitable and untreatable. Some elderly people believe that incontinence is a normal concomitant of ageing, and because they know that there is no treatment for ageing, they assume that their incontinence is untreatable. The Cantonese Chinese have a reference to incontinence as the "last decay". Even doctors are known to tacitly endorse the myth that incontinence is part of ageing and untreatable.

This myth should be dispelled. While normal ageing predisposes to urinary incontinence due to changes like uninhibited contractions, decreased ability to postpone voiding, and decreased urethral closing pressure in the female, none of these changes cause incontinence since they occur in normal continent subjects. However, these predisposing physiological changes, coupled with the increased likelihood of pathologic or pharmacologic insults underlie the higher incidence of incontinence in the elderly.

Understanding the attitude of the elderly, identifying the physiologic changes as predisposing factors, and identifying the pathologic or pharmacologic insults precipitant causes would be relevant in developing preventive and management strategies.

The other reason for failure to seek help is the feeling of shame. Urination and bowel movements are natural human functions. Unfortunately, these are pictured as "dirty" waste discharging activities in many minds. Incontinence brings with it a sense of shame at being unable to attend to these "dirty" functions, and may induce some affected old people as well as their families to try to conceal the problem from friends, neighbours and professional helpers.

A more open, accepting and unembarrassed attitude by doctors and professional helpers will help towards encouraging the affected patients and their families to raise these problems. The GP attending to the elderly should make specific enquiry about difficulty with micturition, and emphasize to the affected elderly that his problems are not due to "old age" but to a disease which can be treated.

TERMINOLOGY: CAUSES AND TYPES OF URINARY INCONTINENCE

Classification of the causes of incontinence into "transient" versus "established" incontinence emphasizes the readily reversible nature of the first category. Causes of "transient incontinence" include drugs, infections, stool impaction, atrophic urethritis, restricted mobility, confusion states and various acute debilitating disorders. Causes of "established incontinence include outlet obstruction (e.g. prostatic enlargement), outlet incompetence (e.g. post-prostatectomy), detrusor overactivity (e.g. senile dementia) and detrusor underactivity (e.g. neurologic overflow incontinence). Even with "established incontinence", treatment and ameliorating strategies are available.

Clinical typing into urge, stress and reflex or overflow incontinence helps point to the diagnosis of the underlying cause, and suggest treatment strategies even without the use of sophisticated urodynamic studies.

"Environmental incontinence" is a useful diagnostic category in the elderly who suffer from urgency. A distant or poorly accessible toilet, plus slow painful or restricted mobility, plus urgency give rise to incontinence. Besides treating the urgency, it is important to make the toilet more accessible and easier to use for these elderly.

A COMPREHENSIVE APPROACH IN MANAGEMENT

A careful clinical history and examination can identify the clinical types and likely cause of incontinence as well as the attitudes and concerns of the patient and relatives. The family doctor who makes a home visit may discover indicators of previously undetected incontinence like unusual laundry and smell. The home visit can also provide invaluable input for managing environmental, psychological and social problems.

Whole person management involves medical, psychological and social dimensions — in each of these, the family doctor can assert positive contributions, clearly showing that urinary incontinence is treatable.

MEDICAL AND NURSING MANAGEMENT

Easily reversible causes of transient incontinence like urinary tract infection, stool impaction and inappropriate use of sedatives or diuretics can be corrected. Drug treatment for incontinence includes use of anti-cholinergics for detrusor overactivity and oestrogens for atrophic urethritis and bladder changes. Surgically treatable causes like obstructive prostatomegaly can be referred for specialist management.

In addition, various nursing strategies can alleviate incontinence. Bladder training, pelvic floor exercises, advice on use of incontinent aids ranging from diapers to marsupial pants, body worn urinals, sheaths and catheters may all be considered. Making the toilet more accessible, and the use of commodes will also be helpful.

CONCLUSION

Addressing a neglected problem

Urinary incontinence is a treatable disorder. Much can be done by the health care services to reduce the morbidity from this condition. Public health education to dispel the myth that urinary incontinence is an inevitable and untreatable accompaniment of ageing will encourage patients and families to seek medical attention. The family doctor is in an ideal position to identify, assess and provide initial management of the problem.

The application of environmental manipulation and nursing strategies can be supervised by the doctor with the support of home visiting nurses. Incontinence clinics can be established in primary health care centres. These can be operated by a medical and paramedical team oriented towards a comprehensive management of the incontinent patient.

Psychological and social management

Coupled with the medical and nursing measures, the doctor's attention and concern for the incontinent problem would be a psychological booster for the patient as well as the carer or his family. The carer can also benefit from simple advice on coping with incontinent laundry and unhygienic smells like rinsing wet clothes and bedding as soon as possible. The family doctor can also help organise family resources to cope with the problem, including arranging respite periods for carers, and helping to direct the family to community sources of help like the Home Nursing Foundation.

CCJ

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2. Milne JS, Williamson J, Maule MM, and Wallace ET. Urinary symptoms in older people. Mod Geriatrics 1972; 2: 198.

ERRATUM

The Singapore Family Physician
Vol XV, No 2, April/June, 1989

What's New in the Eye in Diabetes Mellitus
Dr ASM Lim & Dr C Chiang
Pg 85

Fig. 4 Proliferative diabetic retinopathy with vitreous haemorrhage.

Fig. 5 Advanced stage of diabetic retinopathy with traction retinal detachment.

Fig. 6 Diabetic retinopathy with pan retinal photocoagulation treatment.

PAPER PRESENTED AT THE PLENARY SESSION OF 12TH WONCA WORLD CONFERENCE 29 MAY 1989

THE OUTCOME FOR THE PATIENT; HOW MUCH DO WE HELP?

Dr Alfred W T Loh, *MBBS (S'pore), MCGP (S'pore), FCGP (S'pore)**
Dr L G Goh, *MBBS (S'pore), FCGP (S'pore), M Med (Int Med) MRCGP***

PREAMBLE

Several writers in the past have questioned the contribution of the medical doctor to the health outcome of the patient. Skepticism about the value of medical care provided by doctors became prominent in the mid-1970s mainly as a reaction to i) rising complexities of medical technology (diagnostic and therapeutic) and ii) increasing cost incurred in their use.

In its most extreme, which was articulated by Ivan Illich, a former priest, who in his book "Medical Nemesis — The Expropriation of Health"[1] published in 1975, the stand was that the physician usually did more harm than good. The famous first sentence of Ivan Illich's brilliant polemic 'Medical Nemesis' reads: "The medical establishment has become a major threat to health". Illich argued that the respect given to medicine is totally unjustified and that the apparent successes of medicine in the past could mostly be attributed to other factors. He added that modern medicine did almost nothing for human beings; and if the whole health care structure were dismantled, life expectancy would remain unchanged. Rick Carlson, a lawyer, similarly questioned the need of the doctor in his paper also in 1975, entitled "The End of Medicine".[2]

There were others. A careful search through the medical literature available would reveal that the doubt expressed by these two writers are not at all new. The whole value of clinical medicine provided by doctors, in contrast to preventive health measures like good living conditions have long been strongly questioned by public health scholars. Even as early as 1790, Johann Peter Franck[3] mentioned of poverty as the "mother of diseases". In 1936, Rene Sand,[4] the great Belgian scholar of social medicine, wrote of how the achievements of public health measures dwarfed those of individual physicians. In 1944, CEA Winslow[5] showed that the greatest drop in American death rate between 1900 — 1940 was due mainly to public health programmes rather than clinical medicine.

More recently in 1976, Thomas McKeown[6] showed that in England, the infant mortality rate and mortality from tuberculosis began their decline in the early 19th century long before the discovery of the tubercle bacilli.

It can be argued that the demographic statistics shown by McKeown reflected only the public health dimension but not the impact of the doctor-patient encounter dimension.[7] Many have since challenged Illich's view on medicine. One of the devastating critiques is that by Horrobin in his book, "Medical hubris".[8]

EVIDENCE OF THE BENEFIT OF MEDICAL CARE

The task we have this morning is to argue that whilst there have been health benefits from all the past public health measures instituted in the various countries around the world, we the Family Physicians have our unique role to play and that we do contribute positively to the health outcome of our patients. Arising from the clearly demonstrable achievements of preventive health measures, combined with the ever

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increasing cost of medical therapy, there arises an artificial separation or dichotomy between prevention (which is supposed to give a high value returns for low cost) and medical therapeutics (which give questionable value results at high cost). This dichotomy has ignored the fact that the medical health care process is itself a major channel for the delivery of preventive health service.[9]

A study of the records will show that Neonatal Death Rates in all the developed countries and even in the developing countries have over the years been steadily decreasing. Thus, the rate in USA declined from about 45 per 1000 live births in 1915 to 9.5 per 1000 in 1978. Neonatal mortality in Singapore also showed a decreasing trend. In 1936 it was 49.2 per thousand live births compared to 9.7 per 1000 in 1978 and 6.5 per 1000 in 1986. Is this due entirely to public health services? Probably not. It is more likely to be a combination of several contributions: improved antenatal care and better handling of the childbirth process including delivery of newborns in hospitals or maternity homes with modern facilities.

At the other extreme of life scale — the elderly population, similar improved figures are also noted. In the US, before the 1960s, lifestyle strategy for disease prevention and health promotion hardly existed on a large scale, yet US life expectancy in 1960 at age 65 was 11.9 additional years. By 1960, it was increased to 14.3 additional years, an increase of 20.2%. How much of this increase was due to lifestyle and how much was it due to the benefit of direct medical care in the elderly such as the medical treatment of heart disease, diabetes and pneumonia? In the absence of a drastic change of lifestyles, the increase must surely be the direct benefit of medical care and therapeutics. Skeptics may argue that adding years of life after 65 tells us nothing of the quality of life in those extra years. A crucial benefit of medical care is precisely its impact on the quality of life that is, the ability of the patient to be free from symptoms, to function socially and be happy.

McDermott has addressed this issue in a paper, "Medicine: the public good and one's own",[7] in the World Health Forum in 1979. He said that health care is partially done by oneself, but in a large measure done "by others". It comes in two forms: the public health system, which embraces the activities of a wide range of health personnel serving with various degress of autonomy and only loosely and indirectly related to the physician; and the personal-encounter physician system, in which a considerably narrower range of health professionals work in a much more direct relationship to the patient. The personal-encounter physician is the doctor familiar to us all — the one who deals with one patient at a time on a direct doctor-to-person basis. These two systems — the encounter system and the public health system — exert a biomedical influence on us all, and we are also influenced by a third force — the way of living, the lifestyle — permitted by our socio-economic or technologic status. It is not generally appreciated that we have no established indicators by which to measure the impact of the personal-encounter physician system. Indicators as we do have, such as the various death rates, are designed to measure the impact of the public health system. Nevertheless, the functional effectiveness can be clearly demonstrated as has been done by McDermott.[10] In studying the trend of US age-adjusted death rates for all causes, he noted the steady decline of death rates from 1900 to 1937, then the abruptly steeper drop for the next 20 years to 1957. During those last 20 years (1937—1957), there were no major changes in lifestyle and no major new preventive strategy, but sulfa drugs (1937) and penicillin (1945) became new tools in the hands of the physician. Administration of these antibiotics by physicians led to saving of thousands of patients from pneumonia, septicaemia, bacterial endocarditis, osteomyelitis and other bacterial infections that were not preventable by the usual public health preventive measures.

Other positive benefits of the personal encounter system are not reflected in available demographic statistics. For example, there are thousands of patients with serious heart disease who are not only kept alive but are enabled to work and function socially because of skilled medical care.

There are also those with degenerate hip joints who are enabled to walk again and diabetics who cannot be cured but are kept alive, well and fully active for years. Personal care provided by physicians is the mainstay of treatment for hypertension, one of the major risk factors in cardiovascular and cerebrovascular accidents. Medication does not cure hypertension but controls it. The patient stays well and possible complications are reduced. Glaucoma, a major cause of blindness, is similarly treated with topical medication to reduce intra-ocular pressure thereby preventing blindness and deterioration of vision. Psycho-pharmaceutical treatment has also greatly reduced disability from psychoses and depressive disorders, the latter being the commonest of all psychoneurotic disorders. Patients of rheumatoid arthritis may not be able to look forward to a cure, but direct medical intervention with the appropriate medication would enable the patient to live comfortably at home and prevent permanent deformities associated with the arthritic process itself. Such is the evidence that we do contribute positively to the patient even though the eventual outcome is still death or disability. What we have done is to give the patient a better quality of life and may have indeed extended some years to the patients' lives as for example, through timely treatment of congestive heart failure.

Rather striking evidence of the influence of access to medical care on the mortality rates of the elderly is shown by the age-specific data of the United States and Canada.[11] In 1966-67, the age-adjusted death rates of all American women was 5.7/1000, compared with 5.1/1000 in Canada; the Canadian record was better than that of the United States for all age groups. In 1966, the US Medicare and Medicaid programmes came into effect, making medical service financially accessible to virtually all Americans 65 years of age and older. Financially accessible care was available to all Canadians in 1966. A decade later, in 1976-77, the age-specific mortality rates of both populations had declined; those of US women were however, still higher than those of Canadian women at all specific age levels except for the age group 65 and over. In this age group, which in the US had been entitled to socially financed medical care for a decade (1966-1977), the relationship reversed and the US mortality rate became lower than that of Canada (36.0/1000 versus 37.5/1000).

Besides all these physical benefits, we know that there are the psycho-emotional benefits that accompany the patient — doctor consultation, the value of which family physicians here in this audience would testify to in his or her practice. How many a time have we not heard the patient saying to us, "Doctor, just having this consultation and chat with you has made me see things differently and I feel much better already". We have given the patient some peace of mind. Yet it is difficult to obtain statistics to quantitiate this contribution to the patient.

Jack Hadley in his book entitled, "More medical care, Better health?"[12] in 1982 provided convincing arguments on the benefit of medical care provided by doctors. In his analysis of about 400 US Counties, and using demographic data from the 1970 US Census with medical care utilisation data as well, he was able to conclude: "The principal finding of this study is that medical care has a negative (that is, reducing) impact on mortality rates. This implies that health is better when medical care use is higher. After controlling for other factors such as income, education, marital status, work experience, cigarette consumption and disability, cohorts with greater estimated use of medical care appear to have lower mortality rates (for all causes of death)."

TREATMENT AND PREVENTION — THE INSEPARABLE TWINS

Hugh Leavell and Gurney Clark in their Textbook of Preventive Medicine[13] emphasised the close linkage between prevention and treatment in medical care. They wrote of the 4 levels of prevention:

i) Health promotion — as for example, good nutrition, clean water and environment.

ii) Protection against specific illnesses — as for example, immunization against measles, rubella, tuberculosis, diptheria and pertussis.

iii) Early disease detection with early treatment — as for example, health screening programmes.

iv) Limitations of disability — as for example, rehabilitation.

Whilst the first level, health promotion, may be more the arena of the public health authorities, the next three levels are definitely within the realm of the family physician. The competent and conscientious family doctor almost inevitably give preventive service when he or she renders medical care. A word of caution from the doctor about smoking will be more effective than printed warnings on a thousand packs of cigarettes. This can be repeatedly reinforced as the patient comes periodically for his blood pressure check, diabetes or cardiac review or for the frequent bouts of cough and influenza.

In our daily role as family doctors, however, the opportunities for practising preventive medicine are usually not fully exploited especially when the day's patient load is heavy. The average doctor is mainly orientated to the treatment or therapeutic aspect of the presenting disease and to the relief of the patient's symptoms. The doctor associates prevention with the government health authorities and sees his role as a personal and curative one. Yet a well organised and rationally planned health care system should provide integrated preventive and therapeutic medical care that strengthens the effectiveness of both. We, family physicians should all appreciate the importance of prevention in our relationship with every one of our patients. In this way, we would enhance our roles in ensuring a better outcome for our patient.

CONCLUSION

An attempt has been made to refute the notion that we doctors have not helped in the outcome for the patient. There is sufficient historical, demographic and other evidence to show that we do. It is important for our psychological well-being that we constantly remind ourselves that we are contributing positively to the outcome for our patient. We must also communicate our worth to the patient. It is the feeling of self-fulfilment that we do contribute to the good outcome of our patients that provide us the energy and motivation to continue to do a good job. We must not let this motivation be eroded away by familiarity of what we are doing to the extent that it breeds contempt.

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MANAGEMENT OF DIABETES MELLITUS IN GENERAL PRACTICE: A STUDY OF 499 CASES IN SINGAPORE

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ABSTRACT

The Research Committee of the College of General Practitioners Singapore initiated this study on the management of diabetes mellitus in 1988. 499 diabetic patients who had been managed for at least 3 months by the general practitioners (GP) were accrued by 4 participating GPs during a 3 month study period.

88% were managed on oral hypoglycaemic agents (OHA) either singly or in combinations, with glibenclamide emerging as the most popular OHA followed by chlorpropamide. Metformin was used mainly in combination with another OHA. 7.4% were managed on diet alone, 2.4% on insulin, and 2.0% on insulin plus OHA.

Glycosylated haemoglobin (HbA1) was used to evaluate the degree of glycaemic control. The HbA1 cutoff values used for various degree of control were: good as ⩽8%, fair as 8 to 10%. Using these criteria, the categories of glycaemic control were: 29% good, 37.8% fair, and 32.3% poor. As assessed by their GPs, 66.0% and 10.4% were deemed to have good compliance to medication and dietary modification respectively. 47.0% were found to be obese with a Body Mass Index of over 25.

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INTRODUCTION

Diabetes mellitus is a major cause of mortality and morbidity in Singapore. It was the sixth commonest cause of death in 1987 and the death rate per 100,000 population attributed to diabetes mellitus has increased from 6.5% in 1970 to 18.8% in 1987.[1,2] A population-based survey of 17,679 persons aged 15 and above in Singapore estimated a prevalence rate of 1.99%.[3] This indicates that the disease affects over 53,000 persons when projected to the current Singapore population of 2.6 million. Diabetes mellitus was the third commonest disease seen in the government outpatient clinics in 1986, constituting 10.0% of all consultations.[4] Complications of diabetes mellitus which involve the eyes, kidneys, heart and limbs are important causes of disability. Diabetic gangrene was the cause for amputation in 83.2% of 209 elderly persons referred to the Artificial Limb Centre from 1982 to 1985.[5]

Various aspects of diabetes mellitus have been studied in Singapore, such as epidemiology,[3,6] home glucose monitoring,[7,8] use of glycosylated haemoglobin in assessing glycaemic control,[9,11] and complications resulting from diabetes.[12-14] Management of diabetic patients is an important part of the work of the general practitioner. To date, there has been only one local study on diabetes in the context of general practice — a family physician's approach to its diagnosis.[15] Thus, the Research Committee of the College of General Practitioners Singapore (CGPS) initiated a project on the management of diabetes in general practice with the following objectives:

a. to obtain a profile of diabetic patients managed in general practice;
b. to describe the modes of management and treatment regimen used;
c. to assess the degree of glycaemic control by using glycosylated haemoglobin (HbA1) measurements.

MATERIALS AND METHODS

Subjects

A simple protocol was designed in which history, clinical examination requirements, and blood tests were reduced to the minimum to encourage participation in the project.

Invitations to participate in the project were sent to 850 general practitioners (GP) from both members and non-members of the CGPs. Altogether 43 GPs agreed to participate and they accrued 499 subjects during the study period 1st December 1987 to 28th February 1988. Each GP was personally instructed on the purpose and conduct of the study. Subjects to be included in the study had to fulfill the following criteria:

(a) they must have been treated regularly by the GP for a period of at least 3 months;
(b) their consent for blood to be taken by venepuncture for random blood glucose and HbA1 measurements must be given; and
(c) data for each subject is to be entered once only.

GPs were instructed not to forewarn their patients that blood tests would be taken so as to prevent them from taking any measure to "improve" their results.

Tests for Blood Glucose and HbA1

Measurements for random blood glucose was done at the GP's clinic by either Boehringer Mannheim's or Ames' glucometer. The time interval between blood taken and the last meal was noted. Prior arrangements were made with Boehringer Mannheim and Ames companies to supply free glucose strips to all participating GPs. Glucometers were provided to GPs who did not have them and training provided to ensure their proficiency in measuring blood glucose in their clinics

Blood for HbA1 was allocated into an EDTA-container and sent by courier service to a designated private clinic laboratory on the same day. Prior arrangements were made with this laboratory to do the test at a reduced price and the method used was the "Glyco HB Quik Column Procedure" (normal range 5.5% to 7.7%).

Data Processing

The data in the record forms were coded and entered into a personal computer using dbase III. It was then uploaded to the National University of Singapore's mainframe computer and analysed using the SPSS package.

RESULTS

Profile of Patients

Table 1 shows the contribution of patients from the participating clinics in the project. The top nine GPs contributed 239 patients or about half of all subjects.

TABLE 1: CONTRIBUTION OF PATIENTS FROM GENERAL PRACTITIONERS

No of Patients accrued per General Practitioners	No of General Practitioners	No of Subjects accrued
30 or more	3	105
20-29	6	133
10-19	12	178
1-9	23	83
TOTAL	44	499

Table 2 shows the socio-demographic profile of the diabetic patients. There were about equal number of male and female patients. Majority (82.1%) of them were between the ages of 40 and 69 years. There was none below 20 years and only 0.8% were between the ages 20 to 29. The proportion of Indians (15.6%) in the study was more than twice that of the proportion of Indians in the general population. 47.5% of the subjects were working, 34.1% were housewives, and 15.4% were retired. A

TABLE 2: PROFILE OF DIABETIC PATIENTS

Characteristics	Number	(Percent)
SEX		
Male	257	(51.5)
Female	242	(48.5)
AGE		
20–29	4	(0.8)
30–39	40	(8.0)
40–49	116	(23.3)
50–59	149	(29.9)
60–69	144	(28.9)
70 & above	46	(9.2)
RACE		
Chinese	351	(70.3)
Malay	66	(13.2)
Indian	78	(15.6)
Others	4	(0.8)
EMPLOYMENT STATUS		
Working	237	(47.5)
Housewife	170	(34.1)
Retired	77	(15.4)
Unknown	15	(3.0)
FAMILY HISTORY		
Yes	169	(33.9)
No	320	(64.1)
Unknown	10	(2.0)
DURATION OF DIABETES		
2 years	110	(22.0)
2– 5 years	141	(28.3)
5– 10 years	130	(26.1)
10 or more years	118	(23.6)
DURATION OF TREATMENT BY GP		
2 years	173	(34.7)
2– 5 years	167	(33.5)
5– 10 years	101	(20.2)
10 or more years	58	(11.6)

family history of diabetes mellitus was present in 33.9% of patients, and 49.7% of them had been suffering from the disease for five years or more. 31.8% of patients had stayed on with their GPs for diabetes treatment of over 5 years.

MODES OF MANAGEMENT

Table 3 shows the various modes of treatment the patients were given. 440 patients (88.2%) were managed with oral hypoglycaemic agents (OHA), either singly or in combinations, together with dietary advice. 36 patients (8.2%) were managed with diet alone. 12 patients (2.4%) were on insulin treatment and another 10 patients (2.0%) were on insulin plus OHA regime.

TABLE 3: MODES OF TREATMENT

Modes of Treatment	Number	(Percent)
Diet	37	(7.4)
Insulin	12	(2.4)
Insulin & Oral Hypoglycaemic agents	10	(2.0)
Oral Hypoglycaemic agents +	440	(88.2)
One drug therapy	329	
2-drug therapy	109	
3-drug therapy	2	

TABLE 4: DRUGS USED FOR THE 440 DIABETICS ON ORAL HYPOLYCAEMIC MODE OF TREATMENT

ON ONE DRUG THERAPY			ON MULTIPLE DRUG THERAPY		
Drug Name	Number	(%)	Drug combinations	Number	(%)
Glibenclamide	130	(39.5)	Glibenclamide + Metformin	44	(39.6)
Chlorpropamide	108	(32.8)	Chlorpropamide + Metformin	35	(31.5)
Tolbutamide	46	(14.0)	Tolbutamide + Metformin	14	(12.6)
Gliclazide	22	(6.7)	Gliclazide + Metformin	9	(8.1)
Metformin	16	(4.9)	Tolbutamide + Chlorpropamide	3	(2.7)
Glipizide	7	(2.1)	Glibenclamide + Chlorpropamide + Metformin	2	(1.8)
			Others*	4	(3.6)
All drugs	329	(100.0)	All combinations	111	(100.0)

* One subject each for: tolbutamide + glibenclamide, chlorpropamide + glibenclamide, chlorpropamide + gliclazide and glibornuride + metformin.

For the 440 patients on oral hypoglycaemic therapy, 329 (74.8%) were on single drug and 109 (24.7%) on 2-drug therapy, and 2 (0.5%) on 3-drug therapy. The most popular single drug therapy was glibenclamide, followed by chlorpropamide, tolbutamide, gliclazide, metformin, and glipizide (see Table 4). For those on multiple drug therapy, the most popular combination was glibenclamide and metformin, followed by chorpropamide and metformin, and tolbutamide and metformin.

Glycaemic Control

The degree of glycaemic control was evaluated against the standards set by the American Diabetic Association (ADA) which was accepted by the US Department of Public Health Service in 1986.[16] The criteria for various categories of glycaemic control are as given in Table 5. For our study, the criteria were modified by combining the two categories good and acceptable into one category "good". Results of the HbA1 measurements from 495 subjects

TABLE 5: RECOMMENDATED STANDARDS OF GLUCOSE CONTROL FOR PATIENTS WITH DIABETES MELLITUS*

Index	Good	Acceptable	Fair	Poor
Fasting glucose — serum (capillary) +	115(100)	140(120)	200(170)	200(170)
2-Hr postprandial glucose — serum (capillary) +	140(120)	175(150)	235(200)	235(200)
Glycoslyated haemoglobin	60%	8%	10%	10%

* Standards set by the American Diabetes Association and the US Department of Public Health Service, 1986.
+ Each value expressed as mg/dl.

TABLE 6: GLYCAEMIC CONTROL AS MEASURED BY GLYCOSLYATED HAEMOGLOBIN, FASTING BLOOD SUGAR AND 2 HOUR POSTRADIAL BLOOD SUGAR ACCORDING TO RECOMMENDED STANDARDS BY AMERICAN DIABETES ASSOCIATION*

CHARACTERISTICS	Number**	Mean (S.D.)	Percent with Glycaemic control		
			Good	Fair	Poor
AS MEASURED BY % HbA1					
ALL MODES	495	9.4 (2.2)	29.9	37.8	32.3
TREATMENT MODE					
Diet	37	7.9 (1.6)	67.6	21.6	10.8
Oral Hypoglycaemic Agents (OHA)	436	9.4 (2.2)	28.0	39.0	33.0
Insulin	12	10.3 (2.5)	8.3	41.7	50.0
Insulin + OHA	10	11.1 (2.3)	—	40.0	60.0
AS MEASURED BY FASTING GLUCOSE (FG) +	72	162.7 (63.3)	30.5	29.2	40.3
AS MEASURED BY 2HPPG +	88	208.6 (81.3)	38.4	30.7	40.9
COMBINING FG & 2HPPG MEASUREMENTS	160	—	29.4	30.0	40.6

* Refer to table 5 for the recommended standards set for HbA1, fasting blood glucose (serum), and 2HPPBG (serum) measurements. The standards have been modified by combining the categories good and acceptable into one "good" category for our study.

** 4 subjects did not have HbA1 measurement, 339 subjects had their last meals at various times and cannot be used in the evaluation by FG and 2HPPG measurements.

+ FG = Fasting glucose (capillary)
2HPPG = 2 hour postprandial glucose (capillary)

were evaluated based on the above criteria. The categories of glycaemic control were: 29.9% good, 37.8% fair, and 32.3% poor (see Table 6). The mean percent HbA1 was different for the various treatment modes. The highest was insulin plus OHA mode with 11.1%, next insulin mode with 10.3%, followed by OHA mode with 9.4%, and the lowest was dietary mode with 7.9%.

Fasting capillary glucose (viz. those whose last meal was more than 8 hours before blood test) and 2-hour postprandial capillary glucose measurements were obtained in 72 and 88 subjects respectively. (The other 339 patients had their last meals at various time intervals and thus cannot be used in the evaluation by glucose measurements). Combining the fasting blood glucose and 2HPPBG results and applying the modified ADA standards, the categories of glycaemic control were: 29.4% good, 30.0% fair, and 40.6% poor.

Compliance to Treatment

Table 7 shows the compliance level of subjects to medication and dietary modification as assessed by their GPs. 66.0% of them had good compliance to medication, 31.2% fair compliance, and 2.8% poor compliance. (In 37 subjects, this information was not recorded). The GPs did not gave their opinion on the compliance level to dietary modication in 211 subjects. Of the remaining 288 subjects, 10.4% was assessed as good, 63.9% as fair, and 25.7% as poor compliance to dietary modification.

Body Mass Index (BMI), derived from the equation BMI = (body weight in kilograms) divided by (height in metres),^{2} was used to assess weight control. 47.0% of the subjects were found to be obese with BMI of over 25 (see Table 8). 47.6% of subjects had weights within the normal range (BMI between 20 to 25) whilst 5.4% were underweight (BMI 20).

TABLE 7: ASSESSMENT BY GENERAL PRACTITIONER OF PATIENTS' COMPLIANCE TO MEDICATION AND DIETARY MODIFICATION

Degree of Compliance	Compliance to Medication		Compliance to diet	
	Number*	(%)	Number*	(%)
Good	305	(66.0)	30	(10.4)
Fair	144	(31.2)	184	(63.9)
Poor	13	(2.8)	74	(25.7)
Total	462	(100.0)	288	(100.0)

* Not recorded in 37 subjects.
** Not recorded in 211 subjects

TABLE 8: PERCENT DISTRIBUTION OF BODY MASS INDEX BY SEX

Body Mass Index†	Male	Female	Combined Sexes	
	%	%	Number*	%
Underweight (<20)	12.3	4.8	26	5.4
Normal weight (20-25)	44.4	50.6	230	47.6
Obese (25)	49.6	44.6	227	47.0
Total	100.0	100.0	483	100.0
Mean	25.2	24.9	25.1	
(S.D.)	(4.1)	(3.4)	(3.7)	

† BMI = (body weight in kizograms) divided by (height in meters)^2
* Weight & height measurements not recorded in 16 subjects.

DISCUSSION

This study is a first attempt at getting data on the profile of diabetic patients seen in general practice locally and their management. From the outset we have deliberately kept the study design simple so that general practitioners in their busy clinics would not find it too daunting to participate. We are encouraged by the responses — 499 subjects accrued by forty-three GPs. Subjects in this study were not selected based on a randomised probability sampling of all eligible diabetic patients attending general practice clinics. It would be ideal if this was possible, but the realities of general practice in Singapore made this extremely difficult if not impossible. Thus, the findings of this study pertain specifically to the experience of the 43 GPs who responded. However, the results reflect in general terms the pattern of diabetic patients and their care in general practice in Singapore.

As expected, the majority of the subjects were in the older age groups: 29.9% between 50-59 years, 28.9% between 60-69 years and 9.2% 70 years and over. This pattern fits in with the known fact that the risk of contracting diabetes increases with age. With the demographic trend towards an ageing population in Singapore, we can expect more diabetic patients in the future and that a greater proportion will be in the elderly age groups. From the employment profile, it can be seen that the GPs catered to the whole spectrum of people: those working, the housewives, and the retired. Indians constituted 15.6% of all patients in our study which was 2.4 times more than that of their percentage in the general population in Singapore. This finding is consistent with the higher prevalence rate of diabetes in Indians (6.07%) compared to that of the general population (1.99%) found in the population-based survey.[3]

The general practitioner is well placed to provide continuing primary care for the diabetic patients. It has been estimated that more than two-thirds of all outpatient consultations are seen in general practice.[17] From our study, two thirds (65.3%) of the patients had stayed on with their general practitioners for more than two years, and a third (31.8%) for more than five years. This may be an overestimate of the general pattern since the respondent GPs are likely to be more established in their practice.

About three-quarter of the patients on oral hypoglycaemic mode of therapy were on single drug regimen. It is certainly more convenient for the patient if satisfactory control can be maintained with one drug alone. Glibenclamide has emerged the most popular OHA. 130 subjects were on glibenclamide alone while another 44 subjects had glibenclamide plus metformin combination. This is not surprising in view of the fact that this newer generation OHA has the advantage of a convenient dosage (o.m. or b.d.) without substantial increased risk of prolonged hypoglycaemia. Chorpropamide was the second most popular OHA — used in 108 subjects alone and another 35 subjects in combination with metformin. Perhaps the popularity of chorporpamide has declined because of its proneness to prolonged hypoglycaemia especially in the elderly. Metformin was used mainly in combination with another OHA. It was prescribed alone in only 16 subjects while in another 105 subjects it was used in combination with other OHAs. There were 10 patients on OHA and insulin. The Diabetes Annual 4 (1988)[18] has this to say: "the combination using sulphyluea together with insulin was not generally espoused" and goes on to say that "this combination is probably worth a trial if all else fails... Personal experience with such combinations suggest that such treatment must be continued at least for a month or two to determine possible effectiveness before being discontinued".

Determination of the concentration of glycosylated haemoglobin (HbA1) is the best tool in the assessment of long-term integrated glycaemia. In one value it provides an accurate index of the mean blood glucose concentration during the preceding two months.[19] Its use in clinical care of diabetic patients has also been advocated in recent years.[20,21] For instance, in a consensus statement issued by diabetologists from 14 European countries meeting in 1986, it was recommended that HbA1 be measured every 2-4 months.[22] In this study HbA1 levels were measured in

all except four of our subjects and thus it was possible to evaluate the degree of glycaemic control based on this measurement. There are different criteria for categorising the degree of glycaemic control. The ADA standards, established in 1986, were chosen because it was accepted by the US Public Health Service. We have combined the good and acceptable categories into one "good" category because it would be difficult to achieve near normal glycaemia without running the risk of hypoglycaemia. The modified ADA values for the various categories were similar to that suggested by Fluckiger and Berger.[23] Based on the ADA cutoff value of HbA1 10% as poor control, almost a third (32.3%) of our subjects were found to be poorly controlled. A higher proportion (40.6%) were classified as poorly controlled if the combined fasting capillary glucose and 2-hour post-prandial capillary glucose measurements were used. This finding suggests that a very substantial proportion of diabetics are poorly controlled in general practice in Singapore.

It would be interesting to compare our results with diabetic patients managed in different settings based on the same criteria and using the same method of measuring HbA1. There is one study published locally on the degree of glycaemic control among 116 diabetic patients managed in the specialist outpatient clinic of Alexandra Hospital.[9] It reported that 20.6% of patients had HbA1 levels of 9% or less using the Helena Glycosylated Haemoglobin Quik Column Method. However, it was not possible to compare our results with theirs because in their study patients were pre-selected. Patients were selected into the study only "if they professed to be regular with, and had no change made to, their therapy for at least two months". Thus, their more favourable result could be partly or wholly due to a selection bias of excluding the poorer controlled diabetics.

The causes for failure to achieve satisfactory glycaemic control are multiple. In this study we looked into compliance to medication and dietary modification. Inadequate patient compliance with prescribed treatment is a common problem with all chronic diseases, including diabetes mellitus.[24,25] In the local context, there is a dislike to taking "western" medicine on a longterm basis in the erroneous cultural belief that it is bad for health. It is not uncommon to meet patients who reduce the frequency and/or dosage of medication on their own initiative. The 66.0% with good compliance to medication was an estimate based on the GP's assessment; it may well be lower if a more rigorous method of assessment was used.

The compliance to dietary modification was even worse — only 10.4% were assessed by their GPs to have good compliance. Another indication of poor dietary compliance was the fact that 47.0% of them were obese. Diet is the cornerstone of treatment for all diabetics. However, the GP is faced with many barriers in educating and motivating the patient to adopt the necessary dietary changes for good control. Firstly, many diabetic patients are elderly and not well educated and they have difficulty in understanding dietary instructions. Secondly, there is the problem of cultural food tradition whereby a lot of rice has to be taken to constitute a proper meal. In this regard, it is timely that the Singapore Dietitian Association has recently come out with locally adapted dietary recommendations for individuals with diabetes mellitus.[26] It is encouraging to note that the GPs are increasingly making better use of the health education materials produced by the Training and Health Education Department of the Ministry of Health. Moreover, practical aspects of diabetic management in the context of local general practice has been reviewed based on discussions at a CGPS teaching seminar on "Continuing Care — Diabetes Mellitus".[27]

In conclusion, this study has been a significant milestone in general practice research in Singapore. It has delineated the profile of 499 diabetic patients accrued by 443 GPs, their modes of management and regimen used, and the degree of glycaemic control. It is hoped that GPs will take it as a challenge to continuously upgrade the quality of care which is the primary goal of our research project.

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THE USE OF RAST IN ALLERGY

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SUMMARY

Total IgE measurement helps in general to distinguish those who have Type 1 disease (ie Bronchial Asthma, Allergic Rhinitis, Urticaria) in correlation with clinical history and skin tests. Specific IgE test (RAST) identifies the antigen and should be used as a basis for antigen avoidance if possible, or desensitization in some cases.

INTRODUCTION

The appropriate use of Radio Allergo Sorbent Test (RAST) in allergic diseases requires the physician to be familiar with the immunological mechanisms of disease. There are at least 4 common types of immunological mechanisms causing disease. It is the IgE mediated disease which occurs in Type I immediate hypersensitivity that concerns us here. It is useful to note that there are psudeo allergic or non IgE mediated mechanisms which mimick IgE mediated diseases. These when suspected should not be investigated by RAST, which is only useful for IgE mediated disease. As shown in Figure 1 the concept of allergic break through should also be borne in mind,[1] for example nasal infection may unmask or aggravate allergic rhinitis.

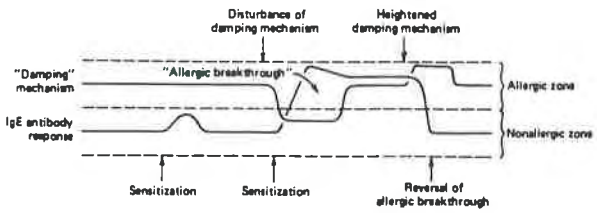
The approach to an allergic problem be it allergic rhinitis or extensive bronchial asthma can be summarized in Table 1.

TABLE 1
APPROACH TO THE ALLERGIC (TYPE I) PROBLEM

(1) History
(2) Skin Test
(3) IgE Measurement:
Total
Specific
(4) Provocation Test
(5) Test of Cure-Blocking Antibody Measurement

History is of paramount importance.[2] When the history is suggestive, appropriate skin tests are indicated. The advantages and disadvantages of prick and intradermal skin testing are summarized in Table 2. A study correlating measurement of IgE antibody by RAST showed a 93% concor-

FIGURE 1



POSSIBLE PATHOGENESIS OF ALLERGIC BREAKTHROUGH

TABLE 2
ADVANTAGES AND DISADVANTAGES OF SKIN TESTING IN IGE MEDIATED DISEASE

Advantages

(1) Provides immediate results.
(2) Cheap and easy office procedure.
(3) Good concordance with clinical history.

Disadvantages

(1) Extensive dermatitis or urticaria prevents its use.
(2) Can be dangerous in very sensitive individuals.
(3) Difficult to perform in children.
(4) Very susceptible to anti-histamines.

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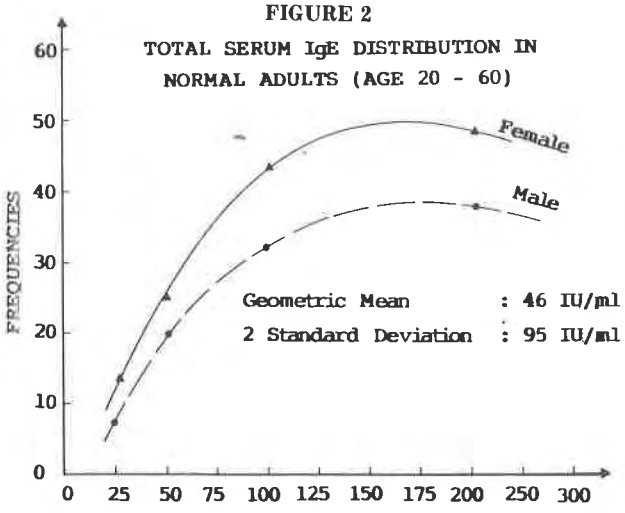
dance rate in fish allergy but 59% with house dust mite. False negative RAST determinations occur in 16% whereas false positive determination occur in 15%. This data only confirms that a good clinical history is of vital importance and this is to be followed up by skin testing and subsequently confirmed by RAST testing.

TOTAL IgE

In vitro IgE measurement was carried out first by Wide by a sandwich radio immunosorbent test and has now been modified to be used via an Elisa technique. There are two types of IgE measurement i.e. total serum IgE and antigen specific serum IgE. The time taken to perform this has been considerably shortened from 2 days overnight incubation to around 6 to 8 hours.[8] To the physician, total serum IgE level may be useful in confirming clinical diagnosis and providing supporting evidence of allergic diseases. The clinician must refrain from discounting allergy when the serum IgE is low or automatically inferring allergy when the level is high.[4] This is best illustrated in Figure 2 which

FIGURE 2

TOTAL SERUM IgE DISTRIBUTION IN NORMAL ADULTS (AGE 20 - 60)



shows the distribution of the IgE in a group of local healthy adult blood donors. Note the wide range, with a geometric mean of 40 IU/ml and 2 standard deviation of 100 IU/ml. These figures differ slightly when compared to Caucasian figures. There is also considerable variation in age-specific total IgE in the paediatric population and therefore its interpretation is to be related to the patient's age. The advantages and disadvantages of RAST measurement are shown in Table 3.

TABLE 3
ADVANTAGES AND DISADVANTAGES OF VITRO IGE MEASUREMENT (RAST)

Advantages

(1) Suitable for children and those with extensive dermatoses.

(2) Can be used to standardize potency of allergen extracts.

(3) Useful when there is doubt over safety of skin tests.

(4) Monitor the efficacy of Immunotherapy. Useful in food allergy.

Disadvantages

(1) Costly and time consuming test to perform.

(2) Requires careful interpretation.

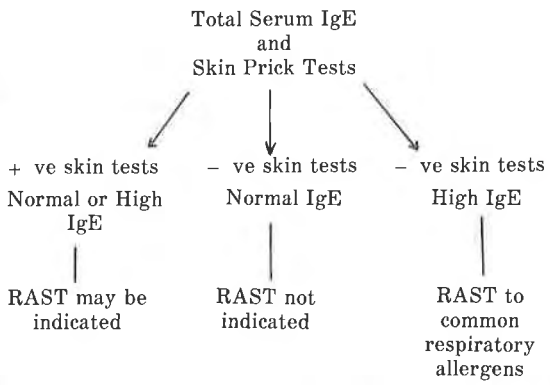
(3) Only confirms history and skin tests.

SPECIFIC IgE (RAST)

Specific IgE tests should only be done after total IgE is elevated although there may be some cases where this is not altogether true. Specific IgE measurement is usually given as class score of 0 to 4 and only Class 3 or 4 should be considered of sufficient significance. Many panels are available commercially. For example panels of common food allergens, air borne allergens or mould allergens can be done.

TABLE 4
INVESTIGATION OF PATIENTS WITH ALLERGY

Adults with Rhinitis and Asthma



The proper use of RAST (Specific IgE)[5] is indicated in Table 4. Used appropriately it yields valuable information.

CONCLUSION

Total IgE measurement distinguishes Type I allergic problems from non-allergic diseases. The specific IgE (RAST) identifies the potential allergen. Both tests should be interpreted in the proper clinical context. Treatment is mainly by suppression of symptoms, avoidance of allergens and desensitization[6] in selected cases.

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ANTIPARKINSONIAN DRUGS

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INTRODUCTION

The medical treatment of Parkinson's disease is one of the most important pharmacological advances in neurology of the last decade. Notwithstanding this, it is important to say that Parkinson's disease does not reduce the patient's insight into his disability and its progressive nature. The management of the patient, therefore, must always include detailed consideration of the psychosocial aspects; these patients require support and supervision at all stages of their disease if optimal management is to be achieved. This article being a drug review will be limited to drug therapy only.

PATHOPHYSIOLOGY AND PHARMACOLOGY

Two balanced systems are important in the extrapyramidal control of motor activity at the level of the corpus striatum and substantia nigra; in one the neurotransmitter is acetylcholine, in the other it is dopamine. In Parkinson's disease, the nigrostriatal pathway is defective and the striatal concentrations of dopamine are very reduced so that the cholinergic system is dominant. The essential dysfunction appears to be in the dopamine neurone and dopamine receptor. There is now evidence for the existence of two types of dopamine receptor. D1 receptors are linked to a dopamine-sensitive adenyl cyclase and their activation results in an increase in cyclic AMP concentrations; they are found in the corpus striatum. D2 receptors are independent of adenyl cyclase and exist in the terminals of the nigrostriatal and corti-costriatal neurones as well as in the corpus striatum itself. While the full functional significance of the individual subtypes is not yet understood, evidence suggests that Parkinson's disease probably arises from dysfunction of D2 receptors.

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There are two approaches to restoring the dopaminergic/cholinergic balance:

1. Reduce cholinergic activity by anticholinergic drugs, and
2. Enhance dopaminergic activity by dopaminergic drugs which may:
   a. replete neuronal dopamine through giving levodopa (L-dihydroxyphenylalanine), which is a natural precursor. Administration of dopamine itself is ineffective as it does not pass into the brain from the blood.
   b. release dopamine from stores and inhibit its re-uptake (amantadine).
   c. prolong the action of dopamine through selective inhibition of its metabolism (selegiline).
   d. act as dopamine agonists (bromocriptine, lisuride, pergolide).

Both approaches are effective in therapy and may be usefully combined. It comes therefore as no surprise that drugs which prolong the action of acetylcholine (anticholinesterases) or drugs which deplete dopamine stores (reserpine) or block dopamine receptors (neuroleptics, e.g. chlorpromazine or haloperidol) will all exacerbate the symptoms of parkinsonism or induce a Parkinson-like state. Other parts of the brain in which dopaminergic systems are involved include the medulla (induction of vomiting), the hypothalamus (suppression of prolactin secretion) and certain paths to the cerebral cortex. Different effects of dopaminergic drugs can be explained by activation of these systems, namely emesis, suppression of lactation (mainly bromocriptine) and occasionally psychotic illness. Drugs used to manage psychotic behaviour, e.g. phenothiazines, may act by blockade of dopaminergic paths and as is to be expected, they are also anti-nauseant, may sometimes cause galactorrhoea and may induce parkinsonism. Neuroleptic-induced

parkinsonism is alleviated by antichlolinergics, but not by levodopa or amantidine, probably because the neuroleptics block dopamine receptors on which these drugs act. But many neuroleptics also have some anticholinergic activity. Those with greatest potency in this respect e.g. thioridazine, are the least likely to cause parkinsonism. Figure 1 shows how certain drugs affect dopaminergic cells and receptors.

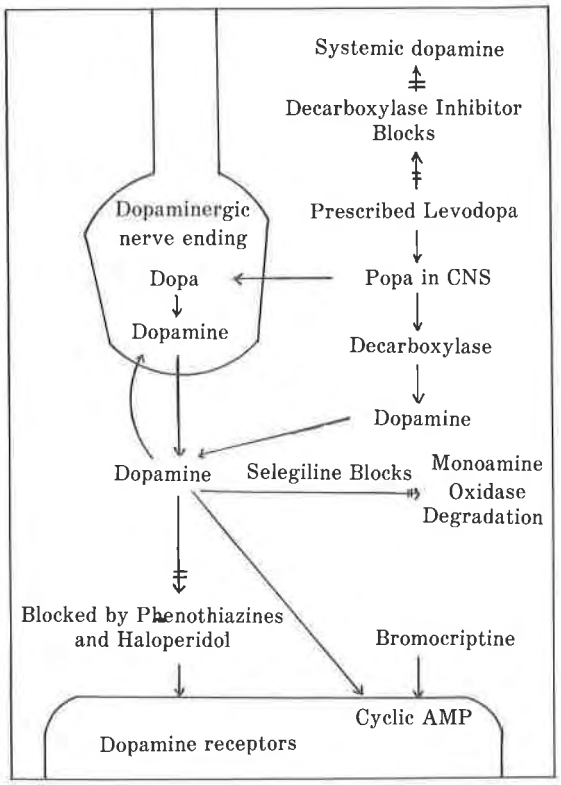

FIGURE 1
DIAGRAM SHOWING HOW CERTAIN DRUGS AFFECT DOPAMINERGIC CELLS AND RECEPTORS.

DRUG THERAPY

The drug management has evolved partly through serendipity (anticholinergics) and partly rationally from an understanding of the pathophysiology of the disease (dopaminergics). Drug therapy may improve function, decrease morbidity and allow the patient to lead an active life for a longer period than before. Medical treatment should be started only when symptoms interfere with activities that are important to the patient. Delaying treatment does not worsen the long-term prognosis. With the possible exception of selegiline, none of the presently available antiparkinsonian drugs affects the progression of the underlying pathology.

ANTICHOLINERGIC DRUGS

They act by reducing the increased cholinergic activity that develops in the brain as a response to the depletion of dopamine. Their use originated when hyoscine was given to parkinsonian patients in an attempt to reduce sialorrhoea, and it then became apparent that had other beneficial effects of the Parkinson's patient. Anticholinergic drugs produce improvements in tremor, rigidity, sialorrhoea, muscular stiffness and leg cramps, but not akinesia.

Synthetic derivatives are now used. These include benzhexol, benztropine, orphenadrine and procyclidine. Patients often report that one anticholinergic drug suits them better than another, but there are no general principles to determine the choice of drug. No advantage has been established for any one anticholinergic over another in the treatment of any one particular clinical feature.

Side effects include dryness of the mouth, constipation, hesitancy of micturition, blurring of near vision and slight impairment of memory often with thought — block. More serious peripheral side effects include precipitation of glaucoma or retention of urine. The most serious side effect is the development of a toxic confusional state (often with hallucinations) and is particularly common in the elderly, arteriosclerotic or demented.

Mild early cases may be adequately controlled with anticholinergics for some time. They are the first line of treatment for patients with drug-induced parkinsonism in whom it is not practicable to withdraw the offending drug. Such a situation arises in schizophrenic patients on phenothiazines or butyrophenones. Levodopa is contraindicated in these patients as it may worsen the psychosis.

DOPAMINERGIC DRUGS

Levodopa is a natural amino acid precursor of dopamine. The latter cannot be used because, being poorly lipid soluble it is not well absorbed from the gut and it does not usefully penetrate the CNS. Levodopa is absorbed from the small bowel by active transport and has a plasma half-life of about one hour.

The drug can traverse the blood-brain barrier and within the brain it is decarboxylated to the neurotransmitter dopamine. But a major disadvantage is that levodopa is also extensively decarboxylated to dopamine in peripheral tissues and only about 5% of an oral dose of levodopa reaches the brain. (Dopamine formed in peripheral tissues does not enter the brain because it is not lipid soluble). Thus large quantities of levodopa have to be given and the drug and its metabolities cause significant adverse effects, especially nausea and vomiting.

This problem has been solved partly by the development of decarboxylase inhibitors (benserazide and carbidopa). They do not cross the blood-brain barrier, so that they prevent only the extracerebral metabolism of levodopa; thus levodopa which enters the CNS can still be covered to dopamine at the site where it is needed — the striatum. No unwanted side effects have been attributed to carbidopa or benserazide. Each drug is usually administered in a combined formulation containing levodopa. (Levodopa with carbidopa is available as 'Sinimet' and levodopa with benserazide is available as 'Madopar').

The administration of carbidopa or benserazide in combination with levodopa has the following advantages:

- The dose of levodopa can be reduced by 80% without plasma concentration falling.
- Certain adverse reactions, especially nausea, emesis and cardiac arrhythmias, are reduced.
- Alleviation of emesis allows the dose of levodopa to be built up to a maximum tolerated level over days rather than months resulting in early clinical benefit, in 1-2 weeks rather than 1-2 months.
- The concomitant use of pyridoxine (e.g. in self-medication with a multivitamin preparation) does not increase peripheral levodopa metabolism; dopadecarboxylase is a pyridoxine-dependent enzyme.

Levodopa

Levodopa has several side effects. The commonest dose-limiting adverse reaction is the appearance of involuntary movements, usually choreo-athetoid in character, which may affect the oro-linguo-buccal musculature or the muscles of the limbs. Levodopa-induced dyskinesia is readily lessened by lowering the dose but usually at the cost of a worsening in the parkinsonian symptoms of slowness and tremor.

Vomiting, with or without nausea, occurs 20-90 minutes after a dose of levodopa in about 80% of patients. Tolerance to this emetic effect occurs in most, but not all subjects after several months of treatment. Nausea can be minimised by introducing the drug slowly and by advising the patient to take it with food. In some patients it may be necessary to give cyclizine or domperidone, particularly with the first dose of the day. Phenothiazine antiemetics, such as prochlorperazine, should be avoided as they block dopamine receptors in the basal ganglia and cause a worsening of parkinsonian symptoms. Combining levodopa with a decarboxylase inhibitor reduces the incidence of nausea to less than 15% of patients.

Other common side effects include postural hypotension and mental changes. Agitation, anxiety elation, insomnia, drowsiness, depression, aggression, paranoid ideas, hallucinations, delusions or unmasking of dementia may occur within a few days to several years after starting levodopa, and usually occur at the time of levodopa dose increase pyrexia, or the addition of another antiparkinsonian drug. Levodopa induced neuropsychiatric disorders can be treated if necessary with a psychotropic drug or non-phenothiazine derivative such as chlormethiazole. Tricyclic anti-depres-

sants are safe and have an additional and beneficial anticholinergic action. Monoamine exidase inhibitors should never be used with levodopa as they may provoke a dangerous hypertensive crisis.

Other adverse reactions to levodopa are infrequent and include cardiac arrhythmias, hot flushes, gout and gastrointestinal bleeding in patients with a history of peptic ulceration. The incidence of cardiac arrhythmias can be reduced still further by the addition of a decarboxylase inhibitor or domperidone. One of these combinations should be used in parkinsonian patients with heart disease. The benefits of levodopa in practice usually outweigh any risk of cardiac arrhythmia.

Levodopa improves all the clinical features of parkinsonism, but the amelioration of hypokinesia is most important because this is the most disabling functional disturbance, and has proved relatively refractory of most alternative forms of treatment. Approximately 30% of patients obtain substantial benefit and a further 30% derive worthwhile but less spectacular help. Of the remainder, most cannot take levodopa because of adverse effects, and a few fail to respond even though they are receiving a high dose. If a patient reports no improvement in spite of tolerating a large intake of levodopa, the diagnosis of idiopathic parkinsonism should be reviewed. After six years on average 25% of patients will still derive substantial or moderate benefit from levodopa and experience an almost normal life expectancy. About 50%, however, fail to sustain the effect or find they cannot tolerate its adverse effect.

One of the main problems with long-term treatment is fluctuation in response to levodopa. This is often a gradual process beginning with:

- early morning akinesia progressing to
- peak dose dyskinesia
- end-of-dose deterioration and
- the "on-off" phenomenon.

This latter term describes random fluctuations from mobility of dyskinesia or to parkinsonian immobility. Severe dystonic muscle cramps of the hand or foot may accompany the dyskinesia. In some patients, fluctuations are related to the timing of drug administration, when peak plasma concentrations coincide with the dyskinetic phase and low plasma concentrations with immobility, but other patients swing between states of mobility and akinetic states without relation to the timing of doses. After receiving levodopa for 10 years, over 50% of patients experience such swings.

Management of problems with long-term levodopa is difficult; it may involve the following:

- Rearranging the schedule of levodopa treatment; smaller doses are required at shorter intervals — two hourly or less.
- Timing of dose in relation to meals is also important for these interfere with absorption of the drug, especially when the protein content is high.
- Gradual and partial substitution of levodopa with selegiline, which delays dopamine breakdown; this is effective for end-of-dose akinesia in about 40% of patients but it is not successful at alleviating severe 'on-off' fluctuations.
- Substitution of levodopa with bromocriptine has also been advocated because the latter has a longer duration of action, but this drug is more likely to produce adverse effects than is selegiline.

Amantadine

Amantadine is an antiviral drug which given for influenza to a parkinsonian patient, was noticed to be beneficial. The two effects are probably unrelated. Its mode of action remains unclear. Various mechanisms have been suggested, however, including anticholinergic actions, blockade of dopamine reuptake, and enhancing the sensitivity of postsynaptic dopamine receptors.

Amantadine has been shown to produce a modest improvement in all the clinical features of parkinsonism. It may be effec-

tive in the early stages of the disease, either alone or in combination with an anticholinergic drug. When given alone, amantadine often loses its effectiveness after a few weeks or months. Slow withdrawal followed by reintroduction may restore therapeutic response for a limited period. Amantadine is much less efficacious than levodopa, whose action it will slightly enchance; it is more useful than anticholergic drugs, with which it has an additive effect.

Amantadine has the advantage of being relatively free from side effects in the doses normally employed. These include postural hypotension, ankle oedema, livedo reticularis, cardiac arrhythmia, restlessness, nausea, headache, urinary hesitancy or retention, and dry mouth. And like all antiparkinsonian drugs, amantadine may cause hallucinations and confusion in susceptible individuals and therefore should not be given to patients with dementia. Overdoses can precipitate seizures. Side effects with amantadine are dose-related, and are usually reversible 2 to 6 weeks after withdrawal of the drug. Amantadine does not cause dyskinesias.

Bromocriptine

Bromocriptine is an ergot derivative that has dopamine agonist activity; it directly stimulates both pre- and post-synaptic dopamine receptors. The pharmacokinetic and pharmaco dynamic properties of bromocriptine differ somewhat from levodopa. It is rapidly absorbed and its plasma half-life and its therapeutic action are more prolonged (despite its lower potency), so that its action is smoother than that of levodopa. This can be an advantage in patients who develop end-of-dose deterioration on levodopa. In fact, until recently, bromocriptine was mainly used as an adjunct to levodopa in the management of fluctuations — the major long-term complication of levodopa therapy.

However, several recent studies have suggested that bromocriptine, in low dosage (less than 30 mg/day), may be useful as sole therapy in mildly affected patients at the onset of their disease. While probably not producing the same degree of improvement as levodopa, bromocriptine appears to be less prone to cause dyskinesia and fluctuations. Vomiting is infrequent, so bromocriptine may be a satisfactory alternative when levodopa causes persistent nausea, despite the addition of decarboxylase inhibitors and antiemetics.

However, the main problem with bromocriptine is that psychiatric adverse effects (confusion, delusions or hallucinations), are more common and persist longer after stopping treatment, though they do resolve ultimately. Occasionally, patients develop marked postural hypotension on very low doses of bromocriptine. Rare complications with bromocriptine include Raynaud's phenomenon, erythromelalgia, livido reticularis and pulmonary fibrosis with or without pleural effusions. All of these reactions resolve when bromocriptine is withdrawn.

SELEGILINE

Selegiline is an inhibitor of MAO-B and it has been suggested that by decreasing the oxidation of dopamine this drug will conserve transmitter and reduce the likelihood of nigral damage by toxic free radicals. MAO type B is chiefly found in the substantial nigra and corpus stratum, where its physiological role is the metabolism of dopamine. Selegiline, because it inhibits MAO-B, delays the breakdown specifically of nigrostriatal dopamine prolonging its effect. Thus its principal therapeutic benefit is to extend the action of levodopa in those patients who experience end-of-dose deterioration. It appears to be ineffective against the on-off swings in motor activity that occur with levodopa.

Selegiline can cause insomnia and it increases the risk of levodopa induced psychosis. The dose of levodopa usually has to be reduced by some 20% when treating patients with selegiline. Because it is a MAO-B inhibitor, it does not have the 'cheese effect' and thus, unlike MAO-A inhibitors, selegiline does not induce a hypertensive crisis with levodopa.

Lisuride & Pergolide

Lisuride and pergolide (the latter not marketed locally yet) are ergot derivatives. Lisuride is soluble and may be given intra-

venously. It has a very rapid duration of action, 1 to 2 hours, and peak effects occur 15 to 30 minutes following administration of lisuride 0.15 mg intravenously. Clinical actions and side effects are similar to levodopa, with the exception that lisuride often causes sedation, and may sometimes be effective in the treatment of dystonia.

The dopamine receptor stimulant effect of pergolide, unlike bromocriptine, is independent of dopamine stores. Pergolide is the longest-acting ergot derivative so far investigated in parkinsonism, although motor effects (6 to 8 hours) are shorter than hormonal effects (prolactin suppression for 24 to 36 hours).

CONCLUSION

Drugs do not cure but can confer considerable benefit. The various antiparkinsonian drugs discussed (summarised in Figure 2), all have a role in treating the illness but selection of the optimal drug for a given patient depends on the stage and severity of the illness and the presence of co-existing factors such as dementia or postural hypotension.

FIGURE 2.
ANTIPARKINSONIAN DRUGS SUMMARISED.

Drug	Mode of action	Usual dosage regimen	Side effects and precautions
Anticholinergics			
Benzhexol Benztropine Orphenadrine procyclidine	Block the action of acetycholine at muscurinic receptors and blocks the reuptake of dopamine mild anti-parkinsonian effect.	2 mg 3 times daily 1mg 3 times daily 50 mg 3 times daily 2.5 mg 3 times daily	Blurred vision, constipation, urinary retention, dry mouth, postural hypotension, Contraindicated in dementia, prostatism, closed angle glaucoma. Withdraw drug slowly to avoid cholinergic crisis.
Dopaminergic Agents			
Levodopa	Repletes dopamine stores in brain	500-1000 mg daily increased to 4-6 g daily if necessary	Abnormal involuntary movements, dyskinesia, anorexia, nausea, hypotension, psychiatric disturbances. Avoid phenothiazines and pyridoxine. Administer with food if possible check lying and standing blood pressure regularly.
Levodopa/ benserazide 200/50 100/25 50/12.5	Decarboxylase inhibitors inhibit break down of levodopa in periphery	Initially 100 mg levodopa increased over several months until satisfactory response or side effects occur.	Nausea is less when levodopa is given in combination with decarboxylase inhibitor but other effects still occur.
Levodopa/ carbidopa 250/25		Usually 400-800 mg/day levodopa is adequate but doses up to 1500 mg/day may be tolepated by some patients.	

Amantadine	Remains unclear but may be due to anticholinergic actions blockade of dopamine reuptake and/or enhancing sensitivity of postsynaptic dopamine receptors. Mild anti-parkinsonian effects.	Initially 100 mg daily for a week increased to 100 mg twice daily	Ankle -- oedema livedo reticylaris, urinary retention or hesitancy, dry mouth, hallucinations, confusion. contraindicated in patients with dementia
Bromocriptine	Dopamine receptor agonist, directly stimulates both pre- and postsynaptic dopamine receptors. Useful in low dosage as sole therapy in mild disease. Also as adjunctive therapy with levodopa to manage fluctuations	2.5 mg daily increased to 10 mg 3 times daily over 2-3 months.	Nausea, vomiting, dizziness, fatigue, hypotension. contraindicated in patients with dementia or postural hypotension; check blood pressure initially
Selegiline	MAO-B inhibitor; delays breakdown specifically of nigrostriatal dopamine, prolonging its effect. Of most value in the treatment of late parkinsonism and the control of dose -- related response swings to levodopa.	2.5 mg – 5 mg after breakfast and lunch.	Intake after 1600 hours likely to cause insomnia It increases the risk of levodopa-induced psychosis pose of levodopa has to be reduced by some 20% when treating patients with selegiline.

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Multiple Choice Questions

1. Parkinsonism may be aggravated by
   - A. promethazine
   - B. haloperidol
   - C. anticholinesterase drugs
   - D. diazepam
   - E. reserpine.

2. The following statements about anti-parkinsonian drugs are true.
   - A. benzhexol stimulates cholinergic receptors
   - B. levodopa is converted to dopamine in the CNS
   - C. bromocriptine has dopamine agonist activity
   - D. selegiline is an MAO-A inhibitor
   - E. amantadine improves all clinic features of parkinsonism.

3. In the treatment of Parkinson's disease
   - A. anticholinergic agents are most rewarding in patients with prominent hypokinesia
   - B. salbutamol is particularly useful for controlling tremors
   - C. chlorpromazine is relatively contraindicated
   - D. benserazide is useful because it has a selective action on the CNS
   - E. amantadine should not be combined with an anticholinergic drug.

4. Side effects of antiparkinsonian drugs include
   A. choreoathetosis with levodopa
   B. pupillary constriction with benzhexol
   C. ankle oedema with amantadine
   D. postural hypotension with bromocriptine
   E. insomnia with selegiline.

5. The following statements are true
   A. carbidopa reduces the incidence of emesis associated with levodopa
   B. bromocriptine does not cause neuropsycliatric adverse effects
   C. when given alone, amantadine often loses its effectiveness after a few weeks or months
   D. a combination of benserazide with levodopa is best avoided in parkinsonian patients with cardiac disease
   E. bromocriptine induces less dyskinesia than levodopa.

6. In the drug management of parkinsonism it is correct to say that
   A. levodopa is best absorbed after a meal rich in protein
   B. amantadine does not cause dyskinesia
   C. a tricyclic antidepressant should not be given to a patient taking levodopa
   D. drug-induced parkinsonism should be treated with levodopa
   E. bromocriptine will antagonise the effects of levodopa.

7. Levodopa should not be used with the following drugs
   A. benztropine
   B. selegiline
   C. an MAOI antidepressant
   D. benserazide
   E. propranolol.

Answers

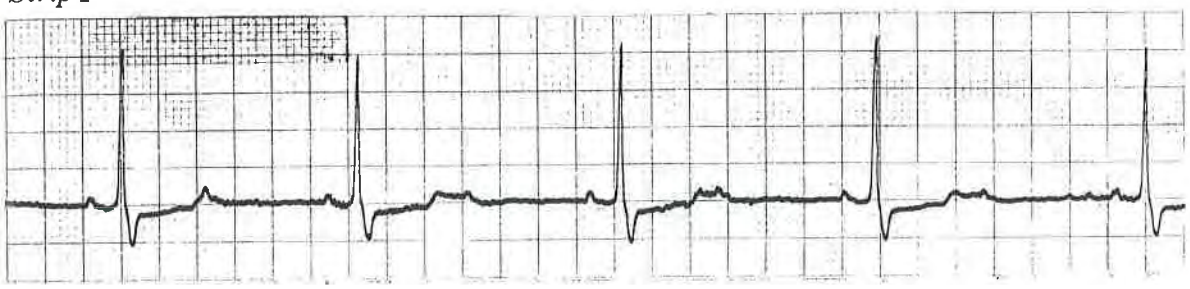
1. A B C E
2. A C E
3. C
4. A C D E
5. A C E
6. B
7. C

ECG QUIZ

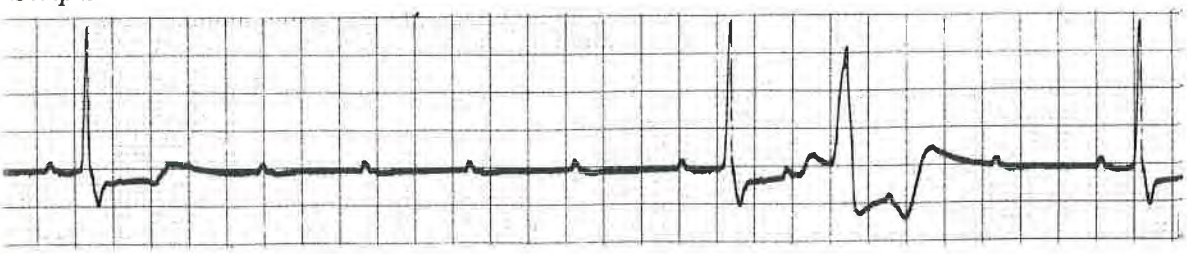
Submitted by **Dr Baldev Singh,** *MBBS (S'pore), M Med (Int Med), MRCP (UK)*

The ECG strip recordings shown below belong to a 58-year-old Malay female who was admitted for dizziness and near-fainting spells. She had no chest pain. The 12-lead ECG showed a Right Bundle Branch Block but no infarct pattern. Serial cardiac enzymes were normal.

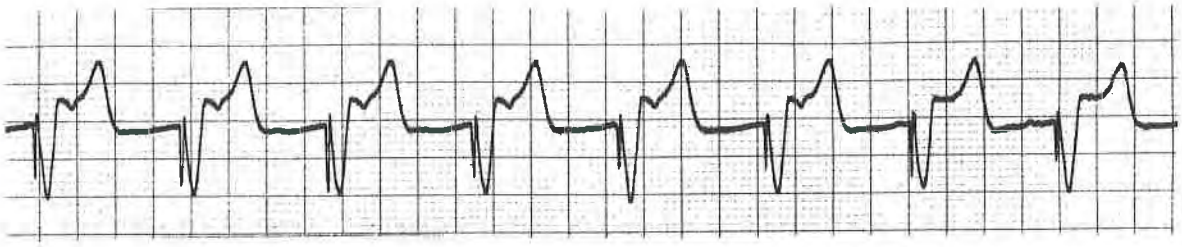
Strip 1



Strip 2



Strip 3



1. What rhythm abnormality is seen in the first 2 strips?
2. What interventional therapeutic modality is demonstrated in Strip 3?
3. What long term treatment mode can be recommended?

ANSWERS TO ECG QUIZ

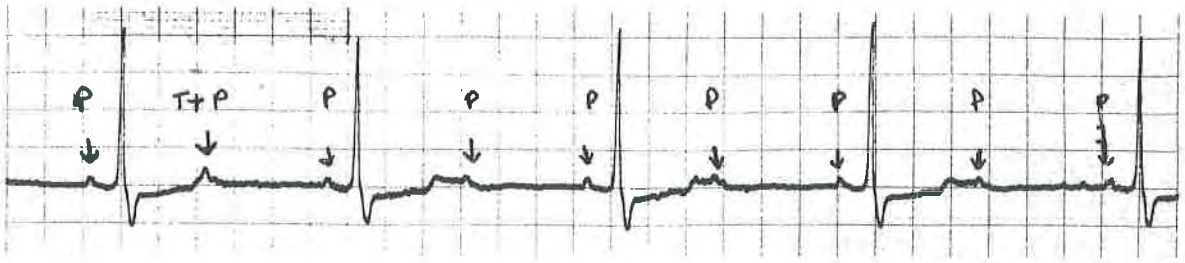
The first step shows a 2: 1 A-V Block, every alternate P wave is seen but not followed by a QRS complex.

The 2nd strip shows a high grade A-V Block with a run of P waves but no QRS complexes. A premature ventricular contraction is also seen.

Strip 3 was taken after a temporary pacemaker was inserted. The pacing spike can be seen as a vertical line before each QRS complex.

A-V Block associated with an inferior myocardial infarction has a high chance of recovery back to sinus rhythm but in a situation like this when the block occurs de novo as a primary event, it is likely to be recurrent or persistent.

The long term solution would be permanent pacemaker implementation. In this situation a dual chamber A-V sequential pacemaker would be ideal. A dual chamber pacemaker will have one lead in the atrium and another in the ventricle. Both leads are able to sense and stimulate. When a P wave occurs the atrial lead will sense it and after a fixed delay the ventricular lead will pace the ventricle if the P wave does not conduct downwards to the ventricles due to A-V Block. If no P waves occur the atrial lead will pace the atrium and this will be followed by ventricular pacing. In this way dual chamber pacing preserves atrio-ventricular synchrony and leads to better ventricular filling and hence a better cardiac output.



12TH WONCA WORLD CONFERENCE — A REPORT

The venue was Jerusalem — one of the 82 names that has been given to this city of ancient civilisation of 4000 years. The occasion was the Twelfth WONCA World Conference held from 27 May to 1 June 1989 and the theme of the Conference was "Universal Issues in Family Medicine". Before the main body of delegates met there was the WONCA Executive Meeting (24 – 26 May).

Dr Alfred Loh represented our College in the WONCA Executive Meeting and I went along as a delegate to the Conference and as observer at this Meeting. In Jerusalem we met another Singaporean at the Conference, Dr Chng Pui Siang.

WONCA EXECUTIVE MEETING

WONCA Council under the able leadership of Dr Rajakumar from Malaysia and his effective Executive Council had achieved much in the last three years. Three items stood out in the proceedings:

(a) The groundwork and help provided to China by Dr Peter Lee, Chairman of Council and Dr Rajakumar in introducing Family Medicine to this huge country: Beijing has already incorporated Family Medicine in one university and will be hosting its first International Conference on Family Medicine from 1st to 7th November this year.

(b) The report by the working party headed by Prof Bent Bentsen on the definition of Family Medicine: It is now some 20 years that Family Medicine has been initiated as a distinct discipline. Developing a paradigm of the knowledge, attitudes and practice skills required to be effective General Practitioner/Family Physician has been an ongoing endeavour. The document by Bent Bentsen and his party attempts to decribe a paradigm of Family Medicine applicable worldwide. The document has been accepted in principle but it is still open for 180 days for amendments to polish up the document.

(c) WONCA Fellowships were awarded to Dr David Game, who has been involved with WONCA for the last 17 years; Dr Max Polliack, of Family Medicine in Israel; and Prof Pertti Kekki, who was the Liaison person of WONCA and WHO.

HIGHLIGHTS OF THE 12TH WONCA WORLD CONFERENCE

The WONCA Conference was a success. Congratulations must go to Israel, the Host Organising Committee for producing a well planned Scientific and Social Programme.

Plenary Sessions

Over the four days of the Conference, each plenary session served to define an area of family medicine. This included the areas of outcome, clinical and competence was then attempted the family, the practice & community. The approach of the Conference was to get plenary speakers to present opposite viewpoints. A synthesis of viewpoints were attempted at the meeting. This proved to be an innovative way of exploring different angles of each topic.

In the first plenary session, Dr Alfred Loh agreed that we as family doctors do contribute to our patients' physical and emotional outcome. Dr Walt Rosser spoke of the potential harm we may be inflicting on our patients unless we are discriminative in our advice on prevention. The cholesterol prevention programme was cited as an example. The Chairman of the first session, Dr Henk Lamberts in his synthesis spoke of the activities of WONCA in developing a better measure of that elusive notion — "The Outcome to the Patient". Quantifiers under consideration include patient perspectives of physical outcomes, as well as emotional, social and activities of daily living outcome.

In the second plenary session the subject of clinical competence was approached from both ends. Dr Eric Fisher spoke of an objective approach to diagnosis — the so called "Doctor-centred approach". Dr

Joseph Levenstein spoke of the "Patient-centred approach". Unlike the doctor-centred approach which is objective based, the patient-centred approach tends to be probability based. The latter approach has the appeal of being more suited to dealing with undifferentiated problems. The synthesis of this plenary session was that Family Medicine requires the ability to practice both approaches depending on the way the patient presents his problems. A cut finger (where an objective approach is appropriate) or a nervour stomach (which is better dealt with in a subjective approach) are examples in point.

The third plenary session focused on the individual in relation to the family. Prof David H H Metcalfe spoke on the importance of the individual in defining what is to be done to him versus the family's mandate in matters that impinges on the individual's relationship with the family. The importance of the family was dealt by Dr G Smilkstan. The conclusion that both the individual and family perspectives are important and a balance of how best their respective interests can be served remains a challenge in clinical practice.

The final plenary session dealt with the perspective of practice and community. The speakers were Dr G Fischer and Dr M Weingarten. Dr Ramos summed up the session by saying that family medicine is a mixture of both the individual and his family on the one hand and the practice and health care team on the other. Family Medicine looks at the patient with twin perspectives. On one aspect, there is the teleperspective of the man which is represented by disciplines like science, anthropology and behavioural sciences. The microperspective on the other aspect is represented by the well-known biomedical perspective of system, organ, cell and organelles. The synthesis of these perspectives yield the holistic approach to family medicine.

Free Papers

Free papers gave opportunities for show-and-tell and collegiate sharing. At it turned out, the scope was quite wide and it was not possible for one to participate in all of them simultaneously.

Social Programme

All work and no play will make Jack a dull boy. The Organising Committee must have believed in that. Prof Yodfat, himself an archaeologist brought us on a tour to the Holy City and the museum in Jerusalem. We were also treated to a sight and sound show on the history of Jerusalem. The cultural shows at the opening and closing ceremonies, particularly the Doctor (and patients) orchestra and the Israeli dances were treats that will remain memorable.

FUTURE WONCA EVENTS

Next year, the Regional WONCA Conference will be hosted by Indonesia in Bali. In 1992, the 13th WONCA Conference will be hosted by Canada in Vancouver; Prof Brian Hennen has made a special appeal to all family practitioners to attend this Conference. Family Practitioners please mark these dates on your calendar.

GLG

2ND ANNUAL SCIENTIFIC CONFERENCE OF THE COLLEGE OF GENERAL PRACTITIONERS SINGAPORE

Date: Saturday, 11 November 1989 & Sunday, 12 November 1989
Venue: College of Medicine Building

SECOND ANNUAL SCIENTIFIC CONFERENCE

Theme:

TEAM WORK IN HEALTH CARE

Saturday 11 November 1989

2.00 pm	Registration
2.30 pm	Welcome Address **Dr Koh Eng Kheng** *President,* *College of General Practitioners, Singapore (CGPS)*
2.40 pm	Opening Address by Guest of Honour, **Dr Seet Ai Mee** *Minister of State for Community Development & Education, Singapore*
3.00 pm	Citation for Sreenivasan Orator **Dr Alfred W T Loh** *Vice President, CGPS*
3.05 pm	THE SREENIVASAN ORATION **TEAM CARE FOR FAMILIES** **Dr Dixie Tan** *Member of Parliament for Ulu Pandan*
3.40 pm	Conferment of Honorary Fellowship on **Dr Syed Mahmood bin Syed Hussain**
3.45 pm	Opening of Meditech Exhibition by **Dr Seet Ai Mee**
3.55 pm	Tea Break

SEMINAR 1
Public Forum on Teamwork in Health Care

4.15 pm Role of the National Kidney Foundation (NKF)
Mr T T Durai
Honorary Secretary, NKF, Singapore

4.35 pm Role of the Home Nursing Foundation (HNF)
Dr Rilly Ray
Executive Director, HNF, Singapore

4.55 pm Role of the Diabetic Society of Singapore (DSS)
Dr Tey Beng Hea
Secretary, Professional & Scientific Subcommitte DSS),
& Consultant Physician, Alexandra Hospital, Singapore

5.15 pm PANEL DISCUSSION
Chairman: **Dr Koh Eng Kheng**
President, CGPS

SEMINAR 2
Consensus Statement on STD & AIDS

8.00 pm What our Community can do
Dr Ong Yong Wan
Chairman, AIDS Task Force & Medical Director,
Blood Transfusion Service, Singapore

8.15 pm What our Profession can do
Dr T Thirumoorthy
Consultant Dermatologist,
National Skin Centre, Singapore

8.30 pm What the Individual can do
Dr Roy Chan
Secretary, Action for Aids Singapore & Registrar,
National Skin Centre, Singapore

8.45 pm Workshop on Consensus Statement*
Diagnosis (2A)
Treatment & Followup (2b)
Public education (2C)

9.30 pm Coffee Break

9.45 pm Presentation of Workshop Consensus

10.15 pm Panel Discussion

10.35 pm Summing up
Chairman: **Dr Goh Lee Gan**
Senior Lecturer,
Department of Community, Occupational &
Family Medicine (COFM)
National University of Singapore

Sunday 12 November 1989

SEMINAR 3
The General Practitioner in Practice

2.30 pm Effective Use of Community Resources
Dr Patrick Kee
General Practitioner

2.45 pm Ensuring Continuity
Dr Goh Lee Gan
Senior Lecturer, COFM

3.00 pm Getting the Message Across
Dr Ong Choo Khim
Chairperson, Community Health Education Committee,
Singapore Medical Association

3.15 pm Judge or Physician?
Dr Wong Wee Nam
General Practitioner

Chairman: **Dr Cheong Pak Yean**
Chairman,
2nd Annual Scientific Conference

LAUNCH OF GP PAPER NO 2
The General Practitioner in Practice

SEMINAR 4
Total Care in Practice

4.00 pm The Stroke Patient
Dr Helen Tjia
Head & Consultant Neurologist
Tan Tock Seng Hospital, Singapore

4.20 pm The Terminal Patient
Dr Anne Merriman
Senior Teaching Fellow, COFM & Vice Chairman,
Hospice Care Group, Singapore

4.40 pm The Psychiatric Patient
A/Prof Teo Seng Hock
Medical Director,
Woodbridge Hospital, Singapore

Chairman: **Dr Lee Suan Yew**
Immediate Past President, CGPS

ONE PIECE COMPLETES THE PICTURE

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

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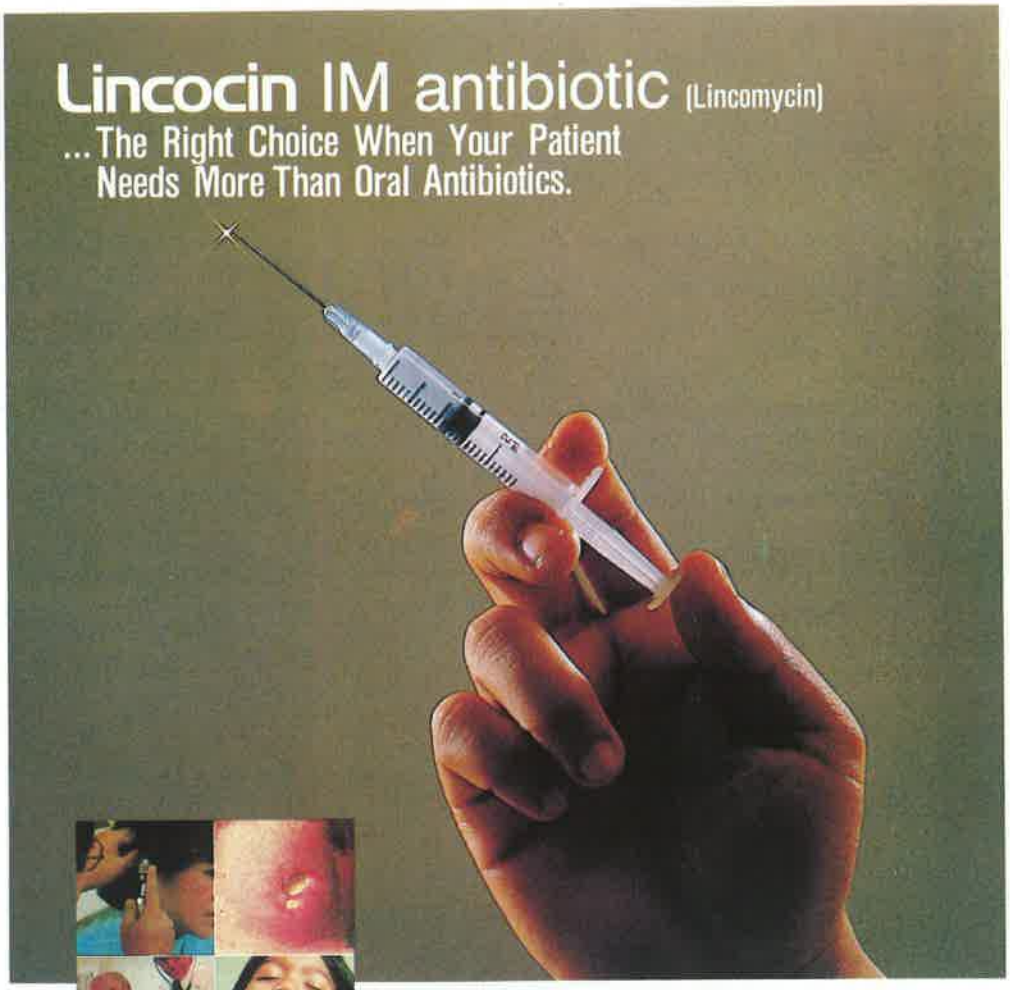
Lincocin IM antibiotic (Lincomycin)

...The Right Choice When Your Patient Needs More Than Oral Antibiotics.



The safe and reliable injectable that induces relief rapidly.

- Proven clinical efficacy – Effective against major gram-positive pathogens
- Rapidly achieves high serum levels – within 30 minutes of a single 600mg (2ml) IM
- Rapid penetration to the site of infection with high antibacterial levels in soft tissue, skin and bone
- Not inactivated by penicillinase, pus or tissue exudates
- Low toxicity
- Rare occurance of hypersensitivity reactions – no cross antigenicity with penicillins or cephalosporins
- Slow development of resistance
- Relatively painless injections
- Enhancement of host defences

Indications: Serious infections caused by gram-positive organisms which are susceptible to the action of Lincocin, particularly staphylococci (including penicillinase-producing staphylococci), streptococci and pneumococci. Not active against *Streptococcus faecalis*, yeasts, or gram-negative organisms including *N. gonorrhoeae*, *N. meningitides* and *H. influenzae*. Some cross resistance with erythromycin has been reported.

Lincocin Sterile Solution has been demonstrated to be effective in anaerobic wound, soft tissue, pulmonary, and bacteraemic infections. The anaerobic spectrum of activity includes *Clostridium tetani*, *Clostridium perfringens*, *Corynebacterium diphtheriae*, *Corynebacterium acnes*, *Bacteroides*, *Fusobacterium*, *Peptococcus*, *Peptostreptococcus* and *Actinomyces*.

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- Outstanding clinical success[1,2,4]
- Excellent activity against aerobes and anaerobes[3]
- A replacement for multiple drug therapy[2,4]
- Reliable prophylaxis against post surgical infection[7,8,9]

References 1. Excerpta Medica, **CCP4**, (1983), 207. 2. J. Antimicrob. Chemother., (1982) **10**, (1), 67. 3. Proc. 4th Mediterr. Congr. Chemother. Rhodes, (1985), **4**, (2 suppl.): 199. 4. Excerpta Medica, **ICS 544**, (1980), 277. 5. Ibid (1980), 173. 6. Med. Int. (1983), (Suppl. Readings in Infections), 2. 7. Proc. 4th Mediterr. Congr. Chemother. Rhodes, (1985), **4**, (2 Suppl.), 776. 8. Br. J. Surg., (1985), **72**, (7), 571. 9. Proc. 26th ICAAC New Orleans, (1986), Abstr. 472.

Prescribing Information

INDICATIONS: Bacterial infections of the upper and lower respiratory tracts, skin and soft tissue, genito-urinary tract; other infections such as osteomyelitis, septicaemia, peritonitis and post-operative infections. AUGMENTIN IV is also indicated for prophylaxis of major surgical procedures.
DOSAGE: Adults and Children over 12 years. Oral:- mild/moderate infections: One 375mg AUGMENTIN tablet tds. Severe infections: One 625mg AUGMENTIN tablet tds or two 375mg AUGMENTIN tablets tds. The 625mg tablet is not available in all countries. **Intravenous:-** 1.2g 6-8 hourly. **Children. Oral:- 7-12 yrs:** 10ml AUGMENTIN 156mg syrup tds or 5ml AUGMENTIN 312mg syrup tds. **2-7 yrs:** 5ml AUGMENTIN 156mg syrup tds. **9 months - 2 yrs:** 2.5ml AUGMENTIN 156mg syrup tds. In severe infections these dosages may be doubled. **Under 9 months:** no suitable oral presentation available. **Intravenous:- 3 months - 12 yrs:** 30mg/kg 6-8 hourly. **Under 3 months:** see Pack Insert Leaflet. Each 30mg AUGMENTIN IV contains 25mg amoxycillin and 5mg clavulanic acid. Treatment with AUGMENTIN should not be extended beyond 14 days without review.
CONTRA-INDICATION: Penicillin hypersensitivity.
PREGNANCY (and lactation): Use in pregnancy is not recommended unless considered essential by the physician. During lactation, trace quantities of penicillins can be detected in breast milk.
PRECAUTIONS: Changes in liver function tests have been observed in some patients receiving AUGMENTIN. Intravenous AUGMENTIN should be used with care in patients with severe hepatic dysfunction. In patients with moderate or severe renal impairment AUGMENTIN dosage should be adjusted as recommended in the Pack Insert Leaflet.

SIDE-EFFECTS: Uncommon, mainly mild and transitory. Diarrhoea, pseudomembranous colitis, indigestion, nausea, vomiting, candidiasis have been reported. If gastro-intestinal side-effects occur with oral therapy they may be reduced by taking AUGMENTIN at the start of meals. Phlebitis at the site of injection has also been reported. Hepatitis, cholestatic jaundice, rare cases of erythema multiforme, Stevens-Johnson syndrome and exfoliative dermatitis have been reported. In the event of urticarial or erythematous rash, discontinue treatment. Angioedema and anaphylaxis have been reported as with other ß-lactams.
AVAILABILITY: AUGMENTIN Tablets: 375mg (250mg amoxycillin/125mg clavulanic acid); 625mg (500mg amoxycillin/125mg clavulanic acid).
AUGMENTIN Syrup: 156mg (125mg amoxycillin/31.25mg clavulanic acid per 5ml). 312mg (250mg amoxycillin/62.5mg clavulanic acid per 5ml). In oral presentations amoxycillin is present as the trihydrate and clavulanic acid as the potassium salt.
AUGMENTIN Intravenous: 600mg vials (500mg amoxycillin/100mg clavulanic acid). 1.2g vials (1g amoxycillin/200mg clavulanic acid). In IV presentations amoxycillin is present as the sodium salt and clavulanic acid as the potassium salt. Each 1.2g vial contains approximately 1.0mmol of potassium and 2.8mmol of sodium. Not all presentations are available in every country.
Further information is available from Beecham Pharmaceuticals, International Division, Brentford, Middlesex TW8 9BD, England.
AUGMENTIN is a trademark.
International Division 001/03/2/57/89

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'NIF-TEN'
Prescribing Notes

USE: Management of hypertension.

DOSAGE (adults only):
One capsule daily; recommended where monotherapy is inadequate. If necessary, one capsule twice daily.

CONTRAINDICATIONS:
Heart block, cardiogenic shock, overt heart failure, pregnancy and lactation, co-administration of cardio-depressant drugs (eg. verapamil), marked renal impairment.

PRECAUTIONS:
Poor cardiac reserve, conduction defects, anaesthesia. Caution in patients with chronic obstructive airways disease or asthma. Withdrawal of beta-blocking drugs should be gradual in patients with ischaemic heart disease. Withdrawal of clonidine. Co-administration with Class I antidysrhythmic agents.

Interaction with cimetidine or quinidine.

Diabetes: Rarely, a transient increase in blood glucose has been observed with nifedipine in acute studies. Modification of the tachycardia of hypoglycaemia may occur.

SIDE EFFECTS:
Dizziness and bradycardia may occur. Headache, flushing, fatigue and oedema have been reported. Skin rashes and dry eyes have been reported with beta-blockers – consider discontinuance if they occur.

Rare reports of jaundice and gingival hyperplasia with nifedipine.

PRESENTATION:
'Nif-Ten' capsules each containing atenolol 50 mg and slow-release formulation of nifedipine 20 mg.

'Nif-Ten' is a trademark.
Further information is available on request.

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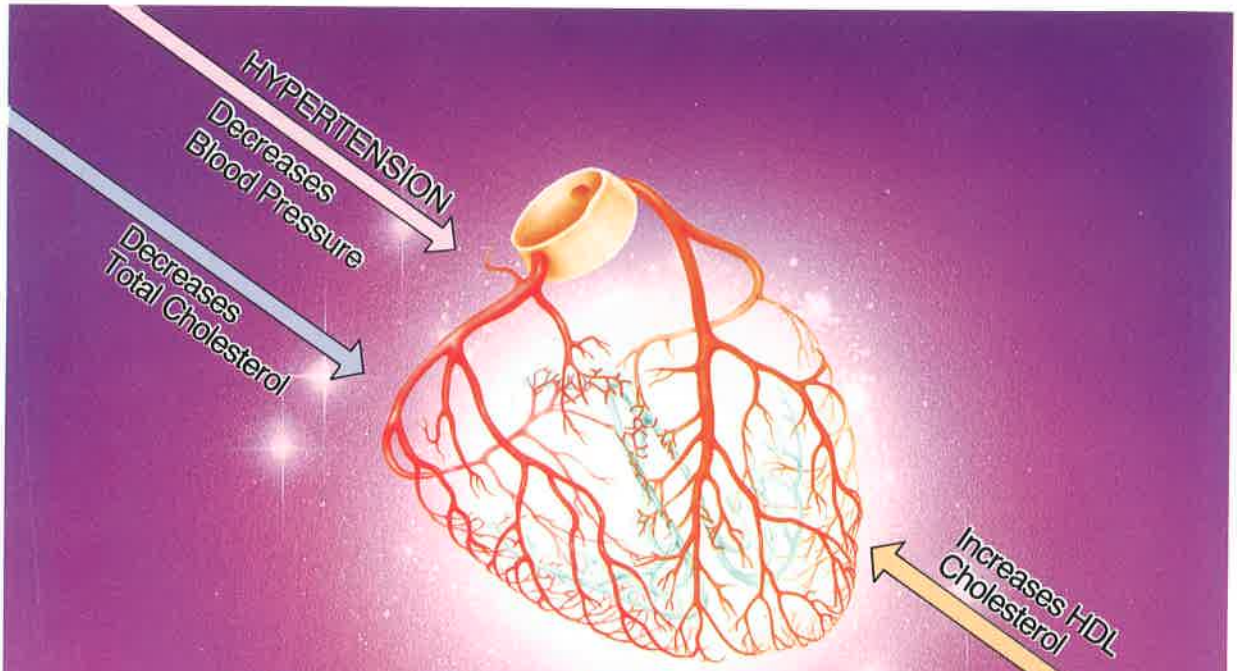

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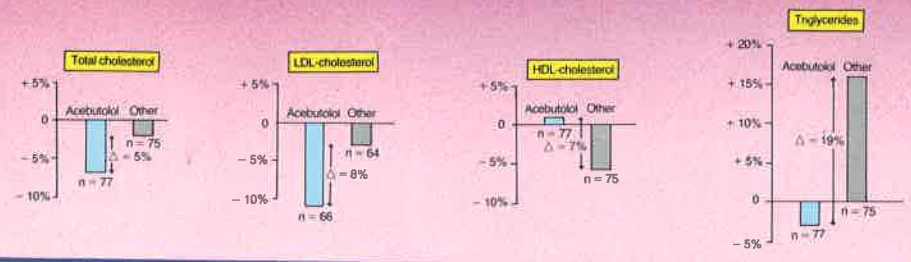
A. Clucas and N. Miller, Drugs 36 (Suppl. 2): 41-50 (1988)

Effects of acebutolol and other β-blockers studied in comparative trials on 4 major lipid parameters.



Prescribing Information:
Dosage: (Hypertension) 400mg orally once daily at breakfast. If response inadequate after two weeks increase up to 800mg once daily at breakfast; up to 1,200mg in divided doses may be required. (Angina) Most patients respond to an oral dose of 200mg twice daily. In severe forms up to 300mg t.i.d. may be required. (Cardiac arrhythmias) **Intravenous:** In severe arrhythmias, dosage depends on the degree of urgency and clinical state of the patient. Twenty-five mg may be administered fairly rapidly intravenously over 3-5 minutes. Initial dose may be followed by a further 25mg slow infusion over an hour or more, again depending on urgency. **Oral:** May take about three hours to exert its full effect. Thereafter dosage may be maintained at 100-200mg two or three times a day. **Contra-indications:** Cardiogenic shock, heart block. Sectral (acebutolol) should not be used with verapamil or within several days of verapamil therapy (or vice versa). **Precautions:** In asthmatics; in pregnancy and those with blood pressures of the order of 100/60 or below. In the presence of bradycardia; with catecholamine-depleting drugs such as reserpine; signs of heart failure; with insulin dependent diabetes and metabolic acidosis dosage adjustment may be required. If preferred, discontinue 24-48 hours before anaesthesia. If a beta-blocker and clonidine are given concurrently, the clonidine should not be discontinued until several days after the withdrawal of the beta-blocker. (see Prescribing Information on clonidine). **Side effects:** Bradycardia, gastro-intestinal effects, depression have occured infrequently. There have been reports of skin rashes/dry eyes associated with the use of all beta-adrenoceptor blocking drugs. Symptoms have cleared when treatment was withdrawn. Discontinuation should be considered if such reaction is inexplicable, cessation of therapy with beta-blockers should be gradual. **Presentation:** 100mg, 200mg capsules; 400mg tablets; Injection sol. 2ml ampoules.

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