The Singapore Family Physician

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Practitioners Singapore
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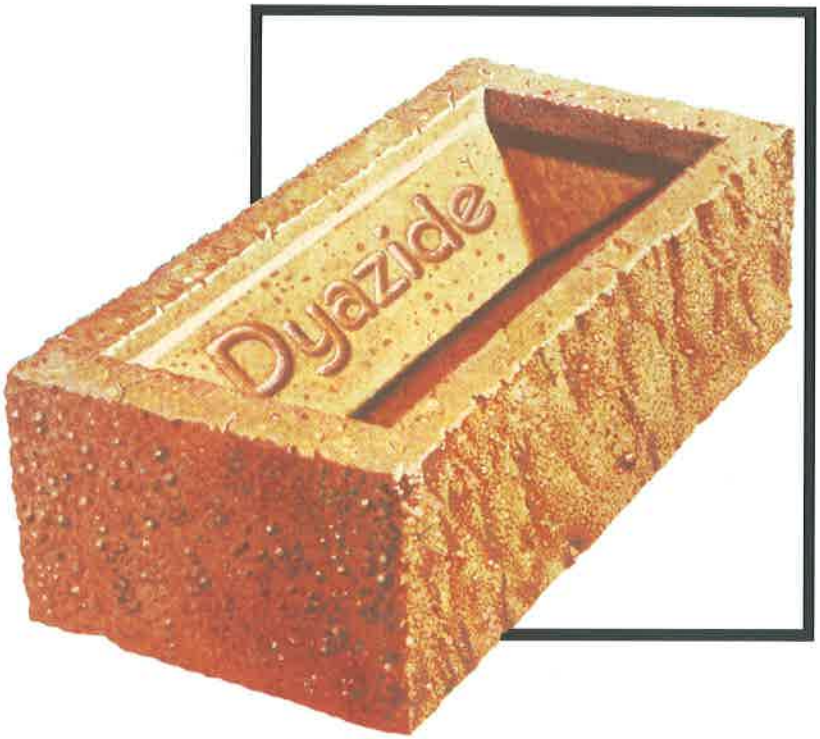

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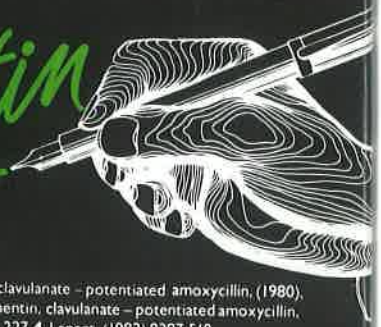

References 1. Proceedings of the 1st Symposium on Augmentin, clavulanate – potentiated amoxycillin, (1980), Excerpta Medica, **ICS 544,** 187 2. International Symposium on Augmentin, clavulanate – potentiated amoxycillin, (1981), Excerpta Medica, **ICS 590,** 65. 3. Excerpta Medica, **ICS 590,** 227. 4. Lancet, (1982) 8297, 510. 5. Excerpta Medica, **ICS 590,** 5. 6. Excerpta Medica, **ICS 544,** 173. 7. Excerpta Medica, **ICS 544,** 19.

AUGMENTIN is a product of original research from
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*Most patients presented more than one symptom

Figure 1. Results of treatment with ‹Lexotan› in 1,182 patients with functional disturbances.

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Editorial
Information needs of GPs

INTRODUCTION

Information needs must be addressed to areas where GPs are deficient. Some of these deficiencies are rooted in history and we must heed well George Santayana's dictum that those who disregard the past are doomed to repeat it.

LESSONS FROM HISTORY

In the 18th and 19th century Britain, the professions were distinguished from crafts more by social criteria than by intellectual considerations. Medicine was then considered too sordid an art for the aristocrats. Moreover it provided no opportunities for the development of social graces and leisurely pastimes. What was even worse, it demanded a total immersion in "dirty" work in a morbid environment. Hence it was relegated to a very low social status.

Literary references to medical doctors at that time were anything but laudatory. Charles Dickens, George Elliot, Jane Austen and Walter Scott portrayed them as artisans, upstarts, fumblers and physic-dispensing, pain-inflicting torturers. Rarely were they depicted as a socially acceptable species. When recognition was given, it was based more on the gentle graces rather than professional accomplishment. Thus Sir Thomas Brown was known for his "incomparable prose" and John Arbuthnot for his wit and learning.

The historian's caricature of the medical doctor was also none too kind. He was regarded as a Dandy – a term coined in the 18th century. Consider the following quotation:–

> "The enormous elongated wig, the stick with its great shapeless gilt knob, the satin coat and the haughty mien of the doctor are no more to be seen, as the new disciples of Aesculapius appear in the fashionable curled coiffure, dressed in the latest mode and carrying walking sticks and by their gallant manners they try to charm their patients."

In the 20th century until the establishment of firstly the Royal College of GPs in the U.K. and subsequently of National Colleges of GPs all over the world, family physicians were generally regarded as the lesser light when compared to their colleagues in institutional practice. Their views and opinions were never sought after and when given were politely tolerated rather than really appreciated.

THE MESSAGE

To me the historical lessons are useful. The message is clear and unmistakable. The first two historical lessons illustrate that unless members of the medical profession can measure up to the literary level of the intelligentsia of the day, they will be regarded as uncouth and unacceptable as equals. The third historical lesson illustrates that unless general practice as a medical discipline is intellectually exacting, followers of other medical disciplines will continue to tolerate rather than appreciate its disciples.

INFORMATION NEEDS IN SINGAPORE

Whether in Singapore or elsewhere, information needs of the GP are in two fundamental directions. They must be geared:–

1) To make him more effective to his patients and
2) To make his chosen discipline more intellectually exacting.

MORE EFFECTIVE GPs

How do we make people more effective?

The answer again is taken from our history books. This had to do with recruitment of able administrators in the Indian civil service during the time of British India. The selection of candidates was by a very stiff academic examination. A young man must be strongly recommended by his headmaster and by his tutor at Oxford. Up he would go to London, if he were not under 21 and over 23 years of age. There he would sit down to an examination in which 21 different papers were offered. He could try any one but none was compulsory. They ranged from Sanskrit to logic and mental philosophy. Each subject had a different value e.g. advanced mathematics was worth 900 marks and Roman history only 400. Seven papers were offered under the heading na-

tural science, and there were papers in Arabic, French, German and political science. There were set books for English literature — Shakespeare's plays, Ben Johnson's Plays, Paradise Lost, The poems of Marvell, Dryden's "Abselom & Achitophel", Bacon's essays and Browne's "Religio Medici". In addition a candidate who took this paper was expected to have a general acquaintance with 25 standard British authors including Chaucer and Macaulay. If the young man succeeded to get through, he would spend a year's probation at an English or Scottish university where a second examination followed. This time he must take compulsory papers in Indian penal code and procedures, the principal language of one of the Indian areas and the evidence and contract acts. He must also take a paper in either the code of civil procedure or Hindu and Mohamedan law, plus a choice of papers in Sanskrit, Arabic, Persian, Chinese and the history of British India. He was also tested in horsemanship to give him the necessary bearing of an administrator.

Perhaps such stringent sieving processes are not needed but GPs must acquire a wide range of knowledge. An effective GP must be a man of letters as well as a man of science.

Since the zone of health and illness is ill-defined, he should be well grounded in the psychology of health/illness with deep understanding of the psychological needs of his patients. An increasing proportion of the work load in general practice concerns social and psychological problems rather than purely physical ones. If the GP is to cope effectively with these problems, his medical education must be extended to include revelant aspects of the disciplines of psychology and sociology. The emphasis must be the study of human growth, behaviour and relationship.

The most common complaint leveled against doctors is that they fail to communicate adquately or effectively with their patients. It is sobering to note that doctors of all people are not taught how to convey bad news to patients and how to cope with the effects of this news. It is estimated that 80% of dying patients know they are dying and would wish to talk about it but 80% of doctors deny this and would like to keep them in the dark. Does the GP understand his own role as healer, counsellor, comforter and confessor? Has he the training which each role demands of him? The GP needs to be more effective in communicating with his patients in language which they understand. Is he attuned to the many occasions when non-verbal communication is conveyed to him? Does he understand the needs of the terminally ill patient? Does he know how to care for dying patients and their dependents? These are some areas where information needs are required.

The 820 substances listed in Galen's elaborate book of medicines are discovered to be therapeutically worthless. Nevertheless he and his disciples had administered these substances in various combinations which were sometimes harmless and sometimes potentially toxic, to the great satisfaction and relief of their patients. Perhaps the beneficial outcome arose from the confident way in which they were given — the placebo effect. All too often this important factor is either ignored or played down diffidently by doctors when in fact it should be capitalized and carefully made use of as an important therapeutic armamentarium.

Another area of information need is the ability to communicate within the medical profession. Charles Darwin once said that a naturalist's life would be a happy one if he had only to observe and never to write. The world would be poorer if he had just wanted to be happy. With few exceptions, GPs have long, too long, enjoyed the pleasures of just observing their patients and never learned to put their observation in writing. A modest survey of the history of medical science will reveal that there is a high degree of correlation between the ability in writing and achievement in the medical world. Reading and listening are passive experiences. Until GPs learn to write well and to speak with melifluous tongues no one will take them seriously.

Practice management has enjoyed a much neglected importance in the past. This is an area where information needs are badly required and the College of GPs, Singapore is well aware of its importance. Our readers will be reading more about this in the S.F.P. The coverage of practice management is wide and the standing committee of WONCA (World organisation of National Colleges and Academic Associations of General Practitioner/Family Physicians) has included the following under its umbrage:— Community setting for practice, multiple approaches to family practice, relationship of practitioners to their countries and their peers, preventive medicine, protocols for patient care, emergency care, clinical records systems, computer technology and microfische for data storage, patient education, staff training and practice accounts.

CORE KNOWLEDGE

Is core knowledge an information need of GPs in Singapore?

At least 8 medical journals are regularly sent out on a non-subscription basis to general prac-

titioners in Singapore. Two are general practice journals, three are journals from academic bodies and the rest from pharmaceutically oriented publishers. In 1982 these eight journals printed almost 700 medical articles. Allowing one day off a week, the GP was reading 2 articles every day of 1982.

The College of GPs, Singapore conducted two in-depth courses of instruction in 1982. In all 20 lectures were given, supplemented with 8 clinical tutorials. It also conducted a year round home study programme which consisted of 9 articles with multiple choice questions as follow-up.

In 1982, the primary health services of the Ministry of Health conducted averagely 4 lunch-time courses per week at different polyclinics. There were desultory dinner-lectures given by emminent medical personnel passing through Singapore enroute to other medical centres. Numerous seminars and workshops were organised by affiliated specialty societies of the SMA throughout the year.

We have heard of the effects of information deprivation. The converse is equally detrimental. An organism's ability to cope with sensory input is dependent on its physiological structure. The nature of its sense organs and the speed with which impulses flow through its neural system set biological limits on the quantity of sensory data it can accept. Overstimulation at the sensory level increases the distortion with which we perceive reality. Cognitive overstimulation interferes with our ability to think.

The core knowledge menu is more than adequate and the prudent GP must choose the relevant courses to meet with his own needs. Selectivity must be exercised because what is sauce for the goose is not sauce for the gander.

CONCLUSION

Information needs of the GP must be geared to enable him to relate more effectively:—

1) With his patients and
2) With his peers, those in society as well as those in the medical profession.

We should heed the solemn exhortation which Saint Bernardino made in the great Campo at Siena to students who were restless and anxious to rush off to Paris to imbibe new knowledge. "... learn from the animals who eat and store up and ruminate, little by little". The green grass of information must be eaten, stored up and ruminated before it can be turned to intellectual milk.

VC

Views expressed in the Editorial are not necessarily the official views of the College.

General Practice Profile

The Research Committee of the College of General Practitioners, Singapore*.

INTRODUCTION

The General Practitioner provides front line medical/health care. However very little is known about him, his work, his contribution and role in the total medical/health delivery system of our country. Our project is a modest one. We hope to etch him a little more distinctly so that his profile may be better appreciated.

AIMS AND OBJECTIVES

The aims and objectives of this Research Project are to study the following:

(1) the qualifications and experience of practising General Practitioners (GPs),
(2) the In-house facilities available to GPs for conducting laboratory investigations.
(3) the type of external facilities that GPs make use of to investigate their patients' medical problems, and
(4) the procedures done by GPs in their own clinics and those that were referred out.

METHODOLOGY

A questionaire was drawn up and circulated to both College and non-College members known to be in General Practice in April 1982. Of the 656 questionaires sent out, only 130 replies were received, giving a response rate of 20.0% (i.e. 130/656). Four had to be rejected because of incomplete recording of data.

The data in the remaining 126 were coded, fed into a microcomputer (BMC if 800, model 20) and analysed, using a Condor 20 package software programme.

RESULTS

1. Qualifications and Experience of GPs

1.1 Age, Sex, Ratio

Of the 126 doctors who responded, 21 were lady doctors, giving a male/female ratio of 5:1. The age and sex distribution is shown in Table 1. Table 1A shows our cohort of GPs classified by the number of years in general practice.

* Research Committee:

Dr Paul S M Chan
Dr V P Nair
Dr T M Chong
Dr L G Goh

Table 1 Distribution of GPs by Age and Sex

Age Group	Sex		Total
	Male	Female	
20 – 29 +	1	4	5
30 – 39 +	41	8	49
40 – 49 +	40	6	49
50 – 59 +	15	1	16
60 – 69 +	5	2	7
70 & above	3	–	3
Total	105	21	126

Table 1A
GPs as classified by number of years in general practice.

Years in Practice	Number GPs	%
Unspecified	3	2.4
Up to 5 Yr	26	20.6
5 – 9 Yr +	33	26.2
10 – 14 Yr +	22	17.4
15 – 19 Yr +	16	12.7
20 – 24 Yr +	11	8.7
25 – 29 Yr +	5	4.0
30 – 34 Yr +	5	4.0
35 – 39 Yr +	3	2.4
40 and above	2	1.6
Total	126	100

Established GPs, defined as those with 5 and more years' of GP-experience, form 77.0% (97/126) of the GPs in our survey.

1.2 Country of medical education/qualification

79.4% (100/126) were graduates from Singapore. Table 2 shows the distribution of doctors by country of medical qualification.

Table 2 Distribution of GPs by Country of Qualification

Country	Number
Singapore:	
King Edward VII College	3
University of Malaya in Singapore	19
University of Singapore	78
United Kingdom	7
Australia	6
Hongkong	6
India	6
Malaysia	1
Total	126

1.3 Postgraduate Qualification

Table 3 shows the breakdown of postgraduate qualification of GPs in the Survey. 27.8% (35/126) have at least one postgraduate qualification. 13.5% (17/126) have the MGCP Diploma.

Table 3 Distribution of Postgraduate Qualifications amongst GPs

Postgraduate Qualification	Number
MCGP (S'pore)	15
DCH	4
DRCOG	4
FCGP (S'pore)	2
MCGP (Malaysia)	2
DO	2
DTM & H	2
MRCP	2
MMed (Paediatrics)	1
Am Bd (Paediatrics)	1
MRACP	1
M Sc (Public Health)	1
DIH	1
MD Singapore	1
Total	39*

* 4 GPs have two additional medical qualifications

1.4 Service in Government Hospitals

Figure 1 shows the distribution of GPs by years of service in Government Hospitals. It shows that:

(1) 66.7% (84 of the 126) GPs in our survey completed at least two years of Government service before entering into General Practice,
(2) 33.3% (42 of the 126) GPs worked for less than two years in Government service,
(3) 11.2% (14 of the 126) served for 6 years and more in Government service.

1.5 Service in Government Outpatient Clinics (GOC)

Table 4 shows two interesting features:

(1) 57.9% (73 of the 126) GPs had no working experience in GOC,
(2) 15.9% (20 of the 126) GPs worked in GOC for more than a year.

Table 4 Distribution of GPs by years in GOC

Years in GOC	Number of GPs	Percentage of Sample
Nil	73	57.9
Up to 1 Yr	33	26.2
1 Yr +	3	2.4
2 Yrs +	5	3.9
3 Yrs +	3	2.4
4 Yrs +	1	0.8
5 – 9 Yrs	6	4.8
10 Yrs and more	2	1.6
TOTAL	126	100.0

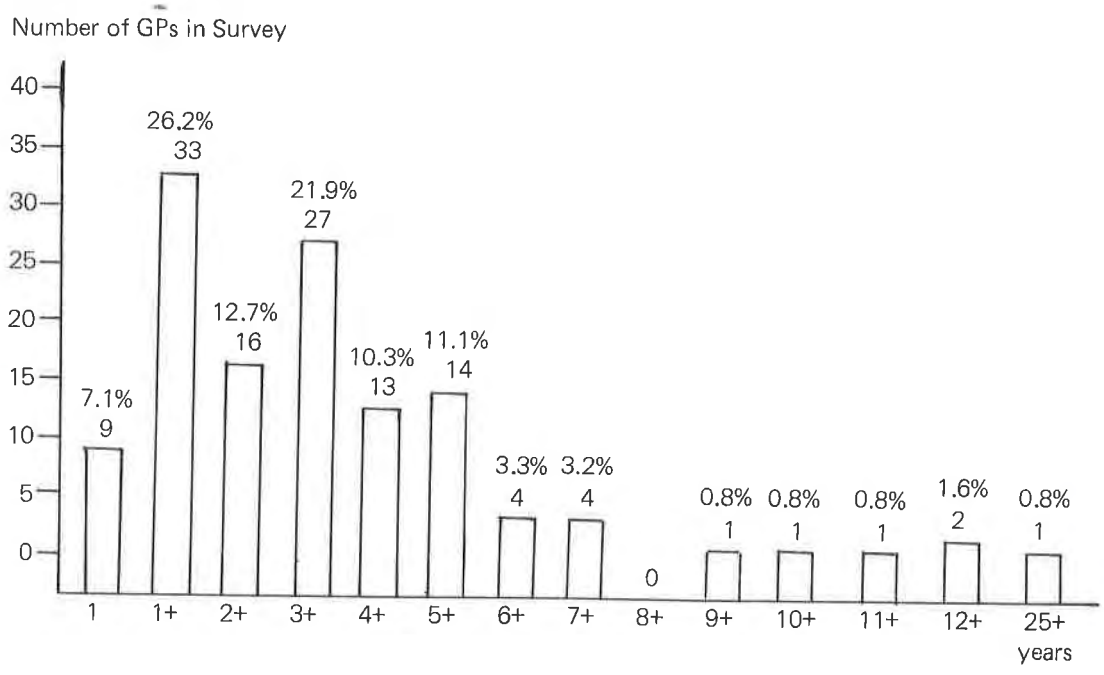
Table 5 Distribution of Hospital Postings Done Against Postings Found Useful By General Practitioners

Posting	Number Did Posting (a)	% Sample Did Posting 100a/126	Number Found Posting Useful (b)	% Found Useful 100b/a
General Medicine	103	86.7	91	88.3
Obstet & Gynae	81	69.3	67	82.7
Paediatrics	59	46.8	47	79.7
General Surgical	78	61.9	55	70.5
Orthopaedics	37	29.4	26	70.3
A & E	53	42.1	31	58.5
Psychiatry	12	9.5	7	58.3
GOC	45	35.7	22	48.9
ENT	11	8.7	5	45.5
Eye	11	8.7	3	27.3
Public Health	13	10.3	2	15.4

1.6 Postings Regarded As Useful By Practising GPs

Table 5 shows the distribution of hospital postings done against percentage of doctors who found a particular posting useful. The five postings regarded by GPs to be most useful are – General Medicine, Obstetrics & Gynaecology, Paediatrics, General Surgery and Orthopaedics in that order.

Figure 1 – Distribution of GPs with completed years of service in Government Hospitals.



1.7 GPs with Different Combinations of Hospital Postings

The distribution of GPs who had various combinations of postings was also studied. This is shown in Table 6. Only 4% of GPs did all the 5 postings perceived to be most useful. There is therefore a place for advocating a rotation programme covering postings regarded as most useful (namely, General Medicine, Obstetrics & Gynaecology, Paediatrics, General Surgery and Orthopaedics) for doctors intending to go into General Practice later.

Table 6 GPs With Different Combinations of Hospital Postings

Posting Combinations	Number Of GPs	Sample Size	Percentage
Gen Med/Surg	65	126	51.6
Gen Med/Surg/Paed	28	126	22.2
Gen Med/Surg/Paed/O&G	20	126	15.9
Gen Med/Surg/Paed/O&G/Orth	5	126	4.0

2. Type of Practice and Type of Patients Seen

2.1 Type of Practice

Table 7 shows the distribution of GP clinics by type of practice. Two features stand out:

(1) Solo practices outnumber group practices by 71 to 55. In HDB estates 64.5% (40/62) are solo practices compared to only 36.1% (13.36) in the City Limits.
(2) 49.2% (62/126) of all GPs surveyed practised in HDB housing estates.

2.2 Type of Patients Seen

Table 8 shows the distribution of GPs by type of patients seen.

(1) Only 7.9% (10/126) of GPs see only private patients.
(2) Only 12.7% (16/126) see mainly contract patients.
(3) Most GPs see an admixture of private and contract patients.

Table 7 — Distribution of GP clinics by type of practice.

Number of GPs in the practice	Locality				Total
	HDB	CITY	Others	Unspecified	
Single	40	13	13	5	71
Two	5	4	4	1	14
Three	3	2	2	—	7
Four	3	1	—	—	4
Five	1	—	1	—	2
Six	3	1	—	—	4
Seven	—	1	—	—	1
Eight	2	1	—	—	3
Eleven	—	6	—	—	6
Eighteen	—	1	—	—	1
Unspecified No.	5	6	2	—	13
Total	62	36	22	6	126

Table 8 – Distribution of GPs by Patient type

	Mainly Private Patients	Mainly Contract Patients	Only Private Patients	Mixed	Unspecified	Total
HDB	42	5	6	6	3	62
City	15	11	3	5	2	36
Others	21	–	1	–	–	22
Unspecified	–	–	–	–	6	6
Total	78	16	10	11	11	126

3. In-house Investigations Available In GP Clinics

Table 9 shows the distribution of in-house investigations available.

The following can be noted:

(1) Practically every GP had dipstick urine tests and urine pregnancy tests.

(2) Some 40-50% of practices have facilities for doing Hb estimations, blood glucose and ECG tests.

(3) About 25-35% of practices had facilities for urine microscopy, stool for occult blood testing, PAP smear, and VDRL or RPR tests.

(4) Less commonly available facilities include stool for ova & cysts examination, blood grouping, PBF smears, blood counts, blood urea estimations, skin scraping for fungal or bacterial smears; only 15-25% of practices had such in-house investigations.

(5) Only 15% had facilities for simple lung function tests (peak flow meter) and X-rays.

Table 9 -- In-house Investigations available in GP – clinics

Type of Investigations	No. of Clinics with facilities	% of Sample studied based on 126 GP-clinics
1. Dipstick urine exam.	124	98.4
2. Urine preg. test	121	96.0
3. Blood glucose	65	51.6
4. E.C.G.	53	42.1
5. Hb-estimation	51	40.5
6. VDRL or RPR	42	33.3
7. Stool for occult blood	42	33.3
8. Urine microscopy	41	32.5
9. PAP smear	35	27.8
10. Skin scrapings for fungus	30	23.8
11. Stool for ova/cysts	28	22.2
12. Blood grouping	26	20.6
13. Bacterial smears	26	20.6
14. Peripheral blood film	25	19.8
15. Blood urea	23	18.3
16. Blood count	19	15.1
17. X-rays	19	15.1
18. Simple lung function test (Peak flow meter)	19	15.1
19. Urine dipstick bacterial culture	12	9.5

4. External Facilities Utilised By GPs To Complement Their Services

4.1 Hospital Admitting Privileges

Only 31.7% (40/126) of the respondents had private hospital admitting privileges.

Of the remaining 68.3% (86/126) of the respondents, reasons given for not having admitting privileges are given in Table 10. Pre-occupation in clinic work was the most commonly quoted reason.

4.2 External Facilities Used By GPs

The distribution of External Facilities Used by GPs are shown in Fig 2a and 2b.

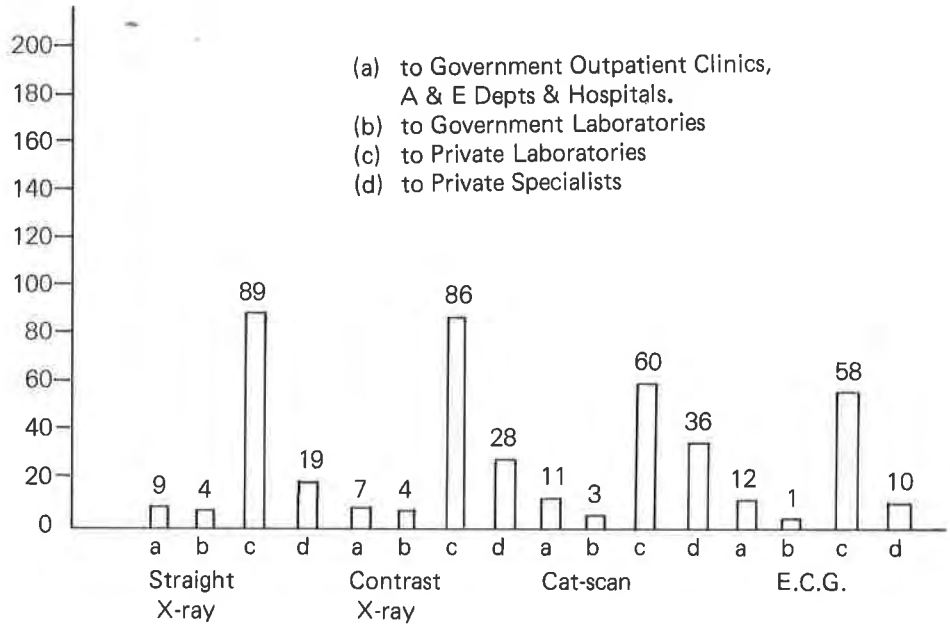
It can be seen that:

(1) For straight X-rays, contrast X-rays, CAT-scans and ECG, the majority of such investigations were referred directly to private laboratories, although a few were sent to private specialists. (Fig 2a)

Table 10 — Reasons given for not having hospital admitting privileges

Reason	Number
Preoccupied with clinic work	36
See no need or significance for it	26
Unable to obtain such facilities	8
Other reasons	8
No reason given	8
Total	86

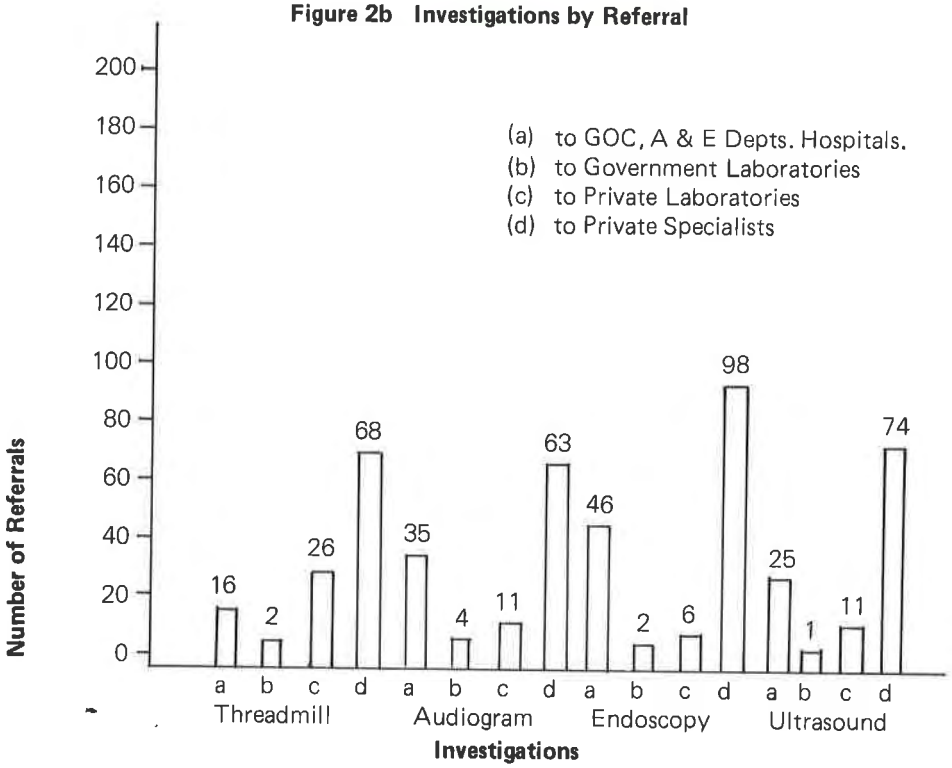
Fig. 2a Investigations by Referral



(2) Investigations sent almost entirely to private laboratories included the following: GTT, Haematological tests, Biochemical tests, Culture & Sensitivity tests and Histology.

(3) Threadmill tests, Audiograms, Endoscopy & Ultrasound examinations were referred to both Private and Specialists as well as Government Hospitals via GOC, A & E Depts. or directly. Referrals directly to private laboratories were negligible (Fig 2 b) Endoscopy, Ultrasound, Threadmill tests and audiograms required specialists skills. This will explain the negligible participation by private laboratories.

Figure 2b Investigations by Referral



5. Medical and Surgical Procedures Done By GPs and Procedures Referred Out

Four patterns can be discerned:

(1) The following procedures were done almost entirely by GPs in their clinics: Toilet and Suture of simple wound, excision of lumps, incision and drainage of abscess, nail avulsion, minor cosmetic surgery, injection of keloid, ear syringing and immunisations. See Fig 3 a

(2) The following procedures were done by about half of the GPs in their clinics: Removal of foreign body from the eye and other parts of the body, intra-articular joint injections and catherisation of the urinary bladder. The remaining half of the GPs referred such cases to GOC, A & E Departments and Hospitals predominantly. This group of procedures represents those procedures that could be handled by GPs if they were given sufficient vocational training in these areas. See Fig 3 b.

(3) The following conditions were almost entirely referred out, and predominantly to Government outpatient clinics, A & E Departments and Government Hospitals. Wound with tendon or muscle injury, partial finger amputation, fractures and dislocations. This is not unexpected since these are traumatic injuries, some of which may be of emergency nature and all would require facilities beyond those in the clinic. See Fig 3c.

(4) The following procedures were referred by GPs almost equally to Government and Private specialists: Circuumcision, injection or ligation of haemorrhoids, diagnostic or therapeutic D & C, insertion of IUCD and cautery of cervix. See Fig 3 d.

SUMMARY

1. A survey was conducted to study the available facilities and procedures in General Practitioners' clinics in Singapore. Of the 656 questionnaires sent out, 130 replies were received, giving a response rate of 20% (130/656) 4 were incomplete and therefore rejected.
2. Of the 126 respondents, 79.4% (100/126) were local graduates. 77% (97/126) are established GPs. 27.8% (35/126) have at least one postgraduate qualifications. 67.5% (85/126) have completed at least two years government service in different hospital postings. 57.9% (73/126) of the GPs have not worked in a government outpatient clinic before entry into general practice.
3. Nearly half of the GPs work in HDB clinics, slightly more than a quarter in city practices and the rest in private housing estates and other suburban parts of Singapore.
   44% (55/126) are engaged in group practices and the rest (56%) in solo practices.
4. In HDB-based practices, only 8.1% (5/62) serve mainly contract patients compared to 30.5% (11/36) in city-based practices. Private patients account for 67.7% (42/62) of HDB-based practices compared to 41.7% (15/36) of city-based practices.
5. Almost all doctors have dip-stick tests (98.4%) and urine pregnancy tests (96.0%) in their clinics. 40-50% doctors have facilities for Hb estimation, blood glucose estimation and E.C.G. About 30% doctors have facilities for urine microscopy, occult blood testing, V.D.R.L. or R.P.R. tests. Only 15% doctors have X-ray facilities.
6. 31.7% (40/126) of GPs have private hospital admitting privileges. Of the 86 who do not have such privileges, half gave the reason that they are too preoccupied in clinic work to look after patients in hospitals.
7. GPs provide their patients with a wide range of procedures e.g. T & S of simple wounds excision of lumps, incision and drainage of abscesses, nail avulsion, minor cosmetics surgery (eg. ear-piercing), injection of keloid, ear syringing and immunisation. More complicated procedures are referred either to government or private hospitals.
8. Investigations such as the following are sent almost entirely to private laboratories: G.T.T., blood tests, biochemical tests, immunological studies, C & S studies and histology.
9. Investigations like threadmill testing, audiogram, endoscopy and ultrasound require some capital investment besides training and the expertise needed in the interpretation of results. Therefore it is not surprising that most GPs referred all these investigations to be done by Government or private specialists.
10. There is considerable cooperative efforts between GPs and their specialist colleagues both in Government and the private sector showing that the referral system is working well. The usefulness of private clinical and Xray laboratories has been demonstrated in our survey.

CONCLUSION

The following conclusions can be drawn from this study despite a GP response rate of 20%.

1) Facilities and procedures offered by the group of general practitioners in this study are wide ranging and generally adequate.
2) More sophisticated facilities and procedures are referred to clinical laboratories and specialists in either the public sector or the private sector based presumably on the socio-economic status of each patient.
3) The postgraduate experience of doctors on entry into general practice varies in terms of:-
   a) previous length of service in government hospitals/institutions (experience in time) and
   b) medical disciplines covered (experience in the spectrum of diseases).
4) Almost a third of our sample have at least an additional medical qualification at the time of the survey. It is noteworthy that 13.5% have acquired the MCGP DIPLOMA whilst in general practice.

RECOMMENDATION

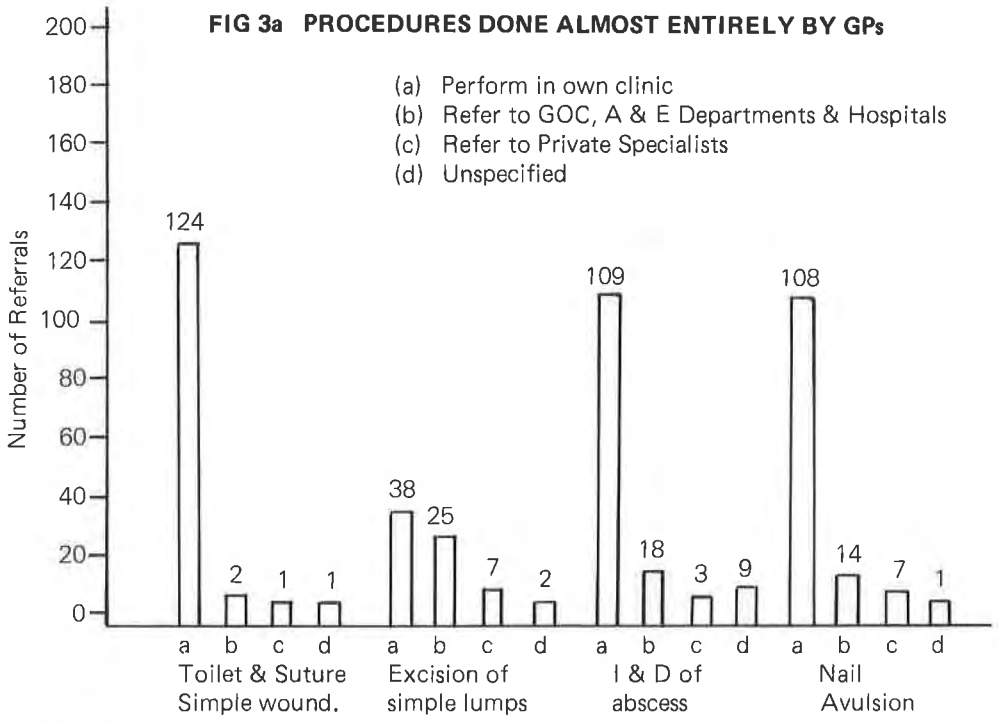
Our study does suggest that there is a place for a hospital-based rotational posting programme so that doctors will be better equipped for general practice. This will help to redress some of the deficiencies which our study has made evident.

A second feature worth studying is the trend towards a small group practice even in HDB Estates. This is a step in the right direction as this pooling of resources allows the practising doctors the chance to work, rest and educate himself further.

ACKNOWLEDGEMENT

Members of the Research Committee, College of General Practitioners Singapore would like to express their gratitude to the 130 GPs who participated in this survey. As this survey was a confidential one, we are unable to acknowledge their names. Our grateful thanks also go to Mr F B Vaz and Mariam Rose Hoon for their secretarial assistance.

FIG 3a PROCEDURES DONE ALMOST ENTIRELY BY GPs



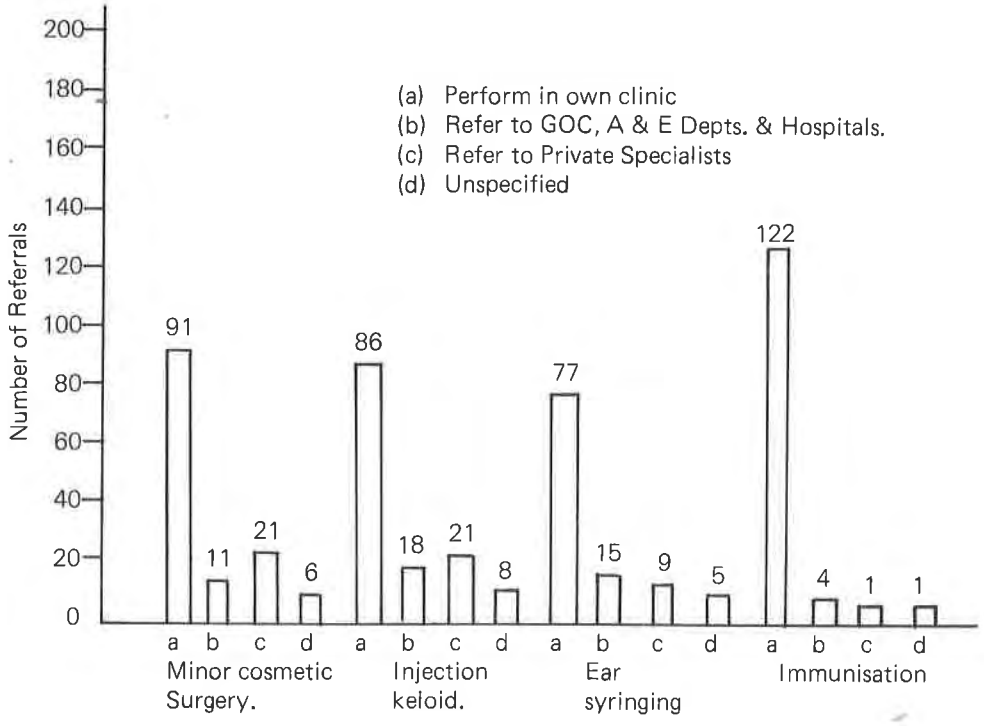


FIG 3 b PROCEDURES DONE BY ABOUT HALF OF GPs

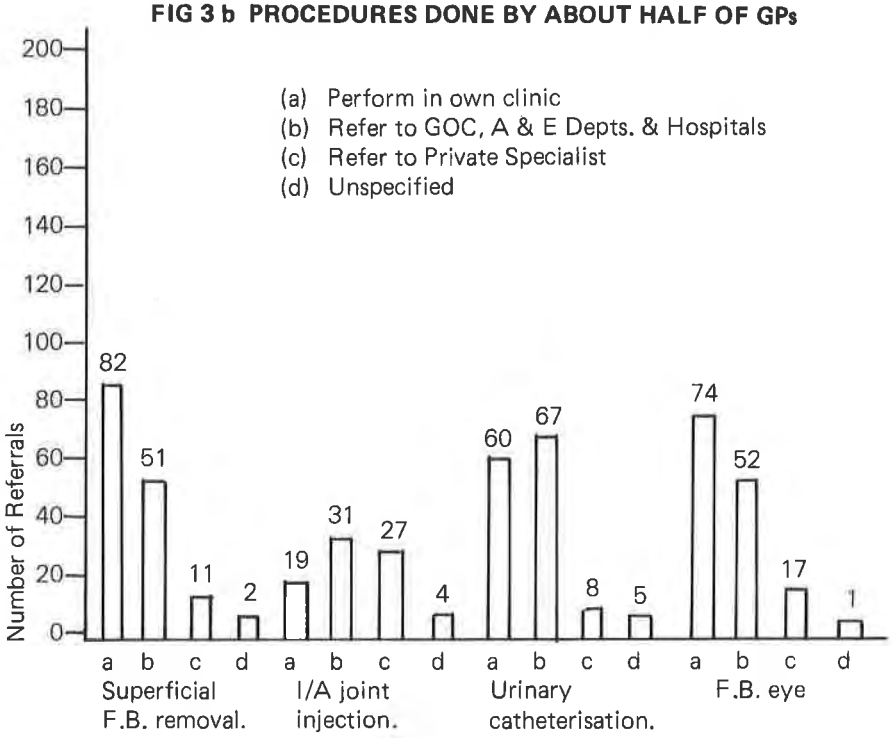


FIG 3 c CONDITIONS REFERRED OUT PREDOMINANTLY TO GOVERNMENT OUTPATIENT CLINICS, A & E DEPTS AND HOSPITALS

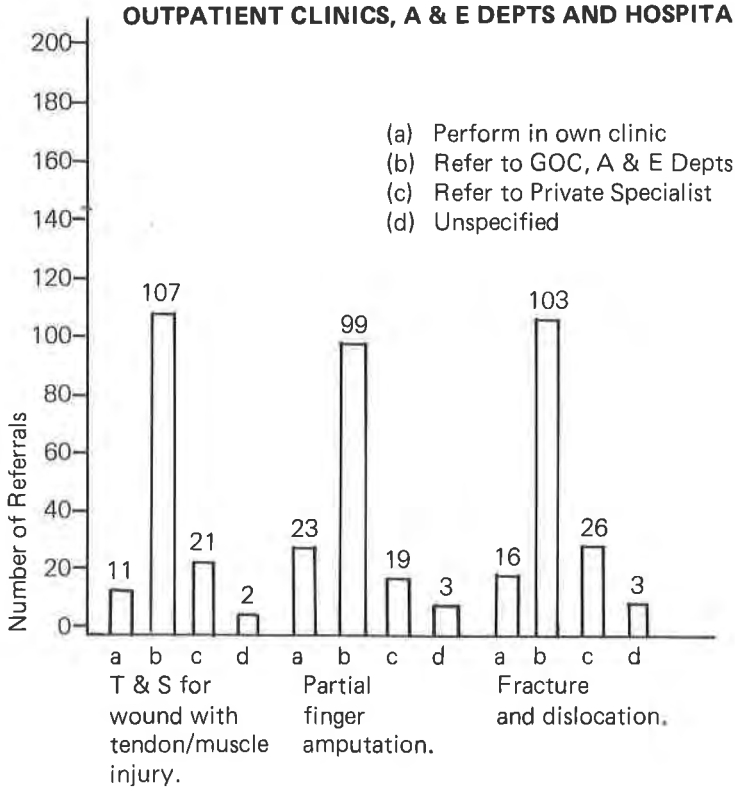
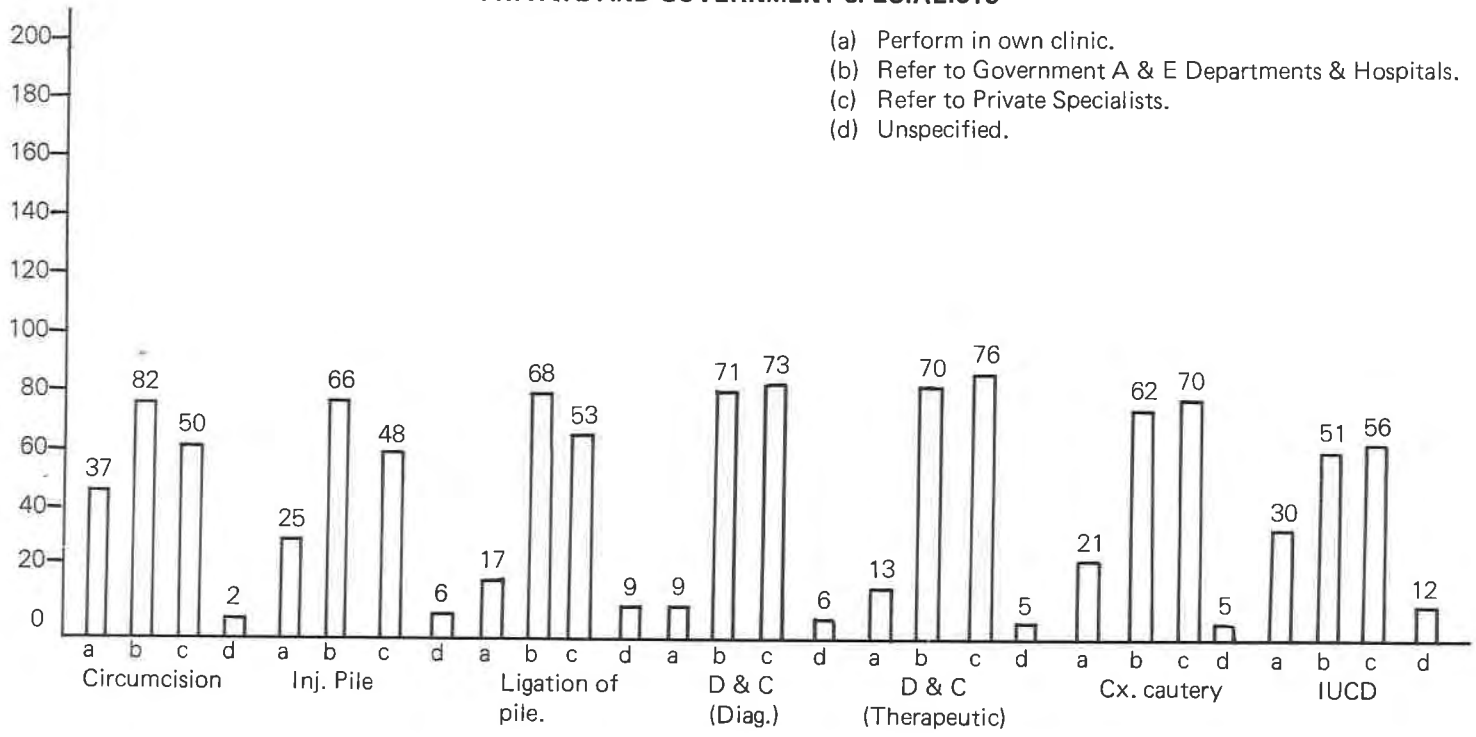


FIG 3 d PROCEDURES REFERRED OUT ALMOST EQUALLY TO PRIVATE AND GOVERNMENT SPECIALISTS

(a) Perform in own clinic.
(b) Refer to Government A & E Departments & Hospitals.
(c) Refer to Private Specialists.
(d) Unspecified.



Calcium Antagonists: A recent advance in Cardiovascular Therapy

DR L P LOW,
MBBS, FRACP, FACC
Consultant Cardiologist
Mt. Elizabeth Medical Centre

In the 1970's the pharmacological treatment of angina and hypertension was dominated by the beta adrenergic blocking agents. The 1980's promise to be the decade of the Calcium antagonists, a new class of pharmacological agents in cardiovascular therapy.

The main clinical applications of calcium antagonists are currently in the treatment of angina pectoris, arrhythmias and hypertension. This paper reviews the physiological basis of these applications.

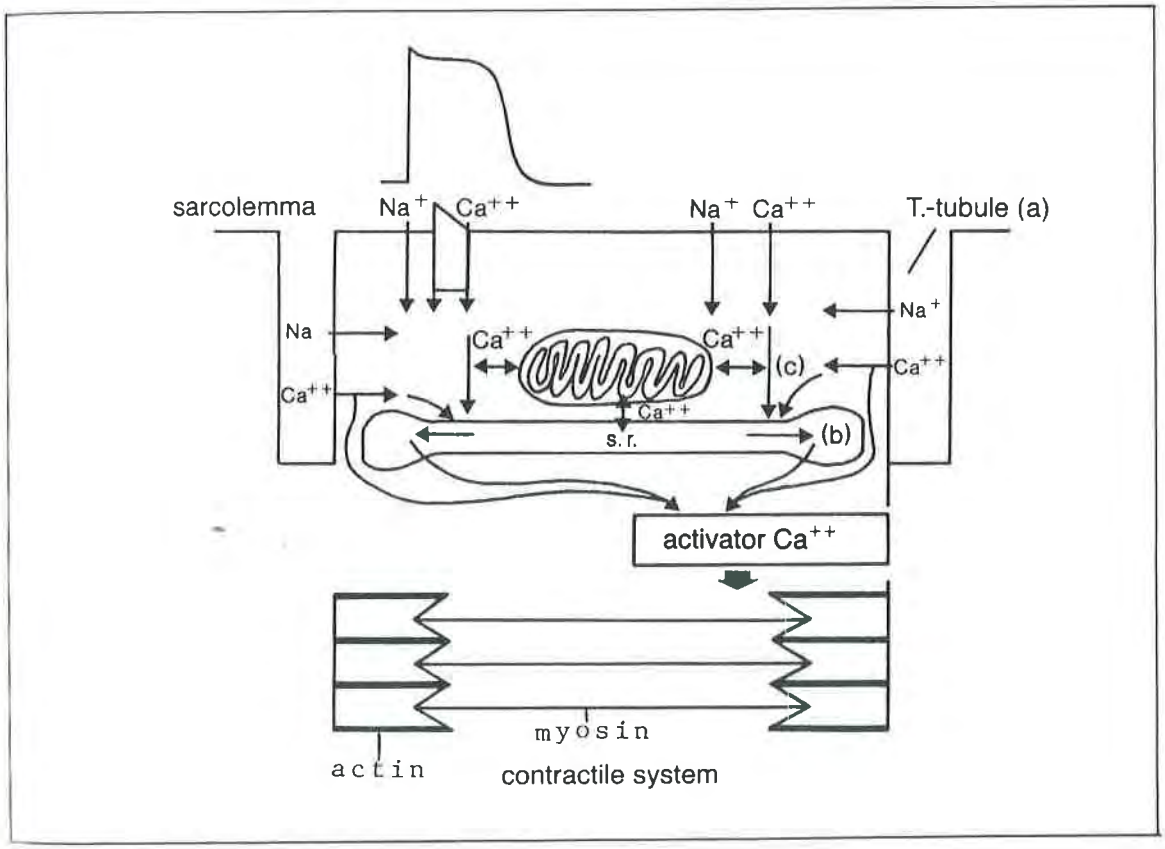


Fig 1. Schematic presentation of the influx of ions into the myocardial cell
(By courtesy from KNOLL)

The Role of Calcium in Excitation-Contraction Coupling

The myocardial fibre has a cell membrane, the sarcolemma, within which are many myofibrils. The basic functional unit of the myofibril is the sarcomere.

Each sarcomere is made up of 2 principal types of filaments viz myosin and actin arranged in a regularly interlocked structure. The interaction of these filaments are modified by 2 so-called "regular proteins" viz troponin and tropomyosin.

During contraction, cross bridges develop between the actin and myosin filaments, which cause

the actin filaments to slide in between the myosin filaments, leading to a shortening of the sarcomere. This contractile process occurs in response to a stimulus which initiates depolarisation of the muscle cell membrane. During depolarisation, an abrupt increase in membrane potential which produces the action potential, is associated with a rapid influx of Na+ ions and a slower influx of Ca ++ ions into the cell.

The influx of Ca++ ions probably occurs through channels, which have been termed "Calcium slow channels", found predominately in the area of the T-tubules of the cell membrane. Some of the calcium act directly on the myofilaments but most trigger the release of much larger quantities of calcium from the terminal cristerns of the sarcoplasmic reticulum, thus raising its concentration to a critical level of 10^{-7} mole at which calcium binds to troponin.

In the resting state the troponin-tropomyosin complex inhibits interaction between actin and myosin. With calcium-binding of troponin, the configuration of the troponin-tropomyosin complex is altered, actin is now able to activate myosin-ATPase, which splits ATP and provides the energy for the actin-myosin interaction, resulting in contraction. Not only is calcium necessary for this process, but the strength of contraction appears to be determined by its concentration.

Following contraction calcium is returned to the sarcoplasmic reticulum by ATP-dependent enzymatic pumps, the actin-myosin overlap is reduced and the muscle starts to relax. During the resting phase, the same amount of Ca++ions leave the cell as entered it during activation.

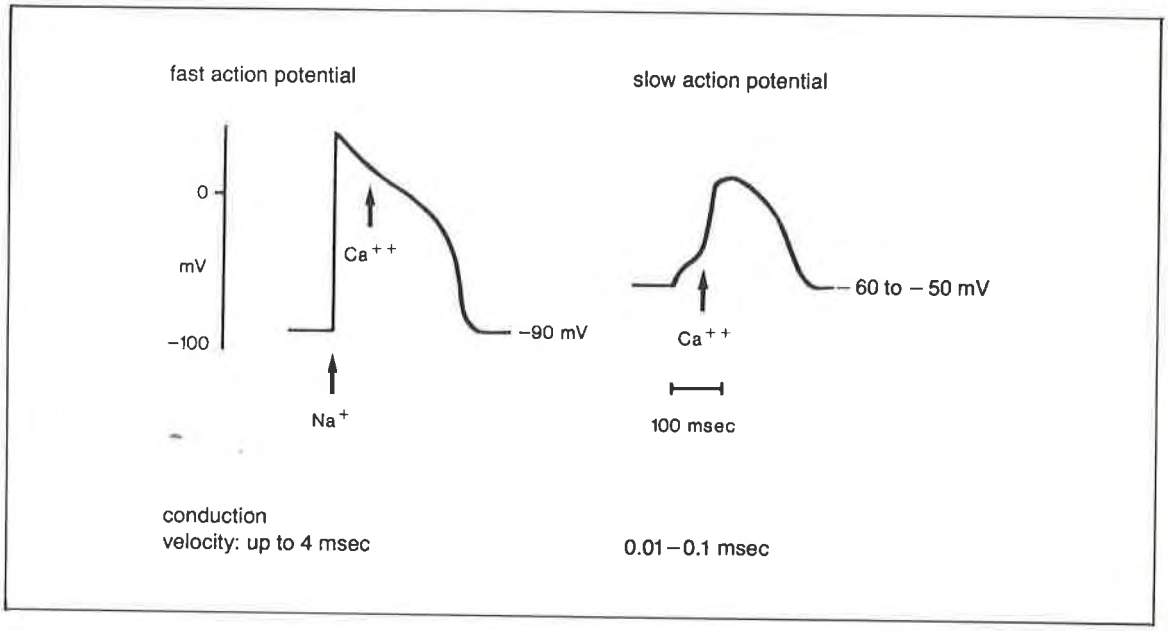


Fig. 2 Comparison of the electrophysiological properties of a fast and a slow myocardial action potential.
(By courtesy from KNOLL)

The Role of Calcium in The Action Potential

The above description of Ca++ion function has to be modified in discussing automaticity and conduction in the various types of cardiac fibres. During depolarisation, whether spontaneous or following stimulation, an action potential is generated. Two types of action potential have been observed.

The "Fast response" action potential characterised by a rapid upstroke velocity and propagation rate is predominantly due to rapid Na+ current movements. It forms the basis for the depolarisation of most normal conducting tissues in the heart, viz. atrial and ventricular muscle, His-Purkinje fibres, atrial internodal pathways and anomalous tracts in the pre-excitation pathways.

The "Slow response" action potential, characterised by a slow upstroke velocity and propagation rate, is predominately due to the slow channel Ca++ influx already described. This type of action potential is found in the sinoatrial and atrioventricular nodes.

Selective inhibition of slow response fibre activity is the basis of the anti-arrhythmic effect of the calcium antagonists.

Calcium in Smooth Muscle

The role of Ca++ ion in myocardial contraction apply in general also to smooth muscle, in particular that in arteries and veins, where muscle contraction is responsible for maintaining tone and is dependent on available intracellular Ca++. Some workers have postulated that vascular smooth muscle contraction is mediated by 2 types of adrenoreceptors viz. α 1 and α 2, and that the α 2 receptors are much more dependent than α 1 receptors on Ca++ ion availability. Alternatively, differences in degrees of Ca++ dependency may be explained by functional differentation of vascular smooth muscle cells.

Inhibition of Calcium++ ion influx in vascular smooth muscle causes vasodilation.

CALCIUM ANTAGONISTS

The Calcium Antagonists or Calcium Blocking Agents are drugs acting at the cell membrance level to specifically block, by competitive inhibition, the calcium slow channels, resulting in a decreased influx of Ca++ into the cell during the active stage.

The available agents at present may be divided into 2 groups:

a. The calcium antagonists of greater efficacy and specificity; including the 3 most widely used clinically at present viz. verapamil, nifedipine and diltiazem. These do not interfere with transmembrane Mg++ conductivity.

b. The less potent and specific agents, including prenylamine and perhexiline, which are therefore not so widely used. They reduce Mg ++ influx.

Although all the 3 Group (a) agents are clinically potent in blocking calcium in the myocardial fibres and vascular smooth muscle, only verapamil and diltiazem have potent effects on blocking "slow response" fibres, while nifedipine has virtually no effect.

The choice of the agent depends on the particular clinical circumstances.

Clinical Applications

The role of calcium described above in myocardial fibres and vascular smooth muscle, provides a rational basis for the following applications of calcium antagonism or blockade.

1. **Angina**

Calcium antagonism in the myocardial cells reduces contractility and therefore myocardial work and oxygen demand, in a similar fashion to beta blockade.

Unlike beta adrenergic blockade which may impair coronary and peripheral vasodilation calcium blockade facilitates both and therefore improves blood supply as well as lower blood pressure. These effects viz. reduced myocardial oxygen demand, increased coronary blood flow and reduced blood pressure, relieve angina.

Peripheral vasodilatation tends to cause reflex tachycardia which may aggravate angina, but in the case of verapamil and diltiazem, this is offset by depression of sino-atrial nodal automaticity.

Calcium antagonism is particularly indicated in angina due to coronary artery spasm, a situation which may in some instances be aggravated by beta adrenergic blockade.

2. **Arrhythmias**

The anti-arrhythmic effect of calcium antagonists is attributed to depression of nomotopic pacemaker automaticity, prolongation of atrioventricular conduction velocity and depression of automaticity of ectopic arising from altered myocardial fibres. Verapamil and diliazem have been used for terminating acute episodes and preventing recurrences of paroxysmal supraventricular tachycardia and slowing the ventricular respond in atrial flutter and fibrillation.

A recent significant observation is that calcium antagonists increase the threshold for experimentally induced ventricular fibrillation in animals following coronary occlusion. This may have an important clinical application in the treatment of acute myocardial infarction and other myocardial ischaemic states.

3. **Hypertension**

In early essential hypertension, some patients show a high cardiac output, but in most patients with established hypertension, the total peripheral resistance is elevated due to an increase in vascular smooth tone. Vasodilators have been used either alone or in combination

with other agents, in antihypertensive therapy to try and reverse this pathophysiological mechanism. The mechanism of action of many of these drugs is still unclear, but it has been suggested that agents like diazoxide, minoxidil and sodium nitroprusside may interfere in some way with intracellular Ca++ in vascular smooth muscle.

Interestingly, studies in spotaneously hypertensive rats suggest that there may be a systemic disorder leading to a great increase in the intracellular free Ca++ of vascular smooth muscle in these animals, and it has been postulated that a similar disorder may be present in essential hypertension.

It is not surprising that there has recently been a great deal of interest in the antihypertensive effect of calcium antagonists. Nifedipine, diltiazem and verapamil have all been shown to be effective in the oral therapy of hypertension, particularly of mild or moderate degree. Severe hypertension responds less well to these agents alone, but may respond to combination of these drugs with other hypertensive medications.

Sublingual nifedipine has a rapid onset of action and is useful when prompt lowering of blood pressure without parenteral therapy, as well as quick relief of angina, is required. It has the advantage of being applicable when the oral route cannot be used. IV verapamil is likewise effective in the rapid control of severe hypertension.

Calcium antagonists may be used to treat hypertension in the following situations:

1. As an alternative antihypertensive agent in "Monotherapy".
2. As an alternative vasodilator in combined therapy.
3. In special circumstances viz.

a. When angina is also present.
b. When an arrhythmia, especially supraventricular tachyarrhythmia, is also present, verapamil and diltiazem may be useful for both conditions.
c. When a beta-blocker is contraindicated e.g. in asthma.
d. When an oral agent cannot be administered e.g. peri-operative states or gastric aspiration in progress, sublingual nifedipine may be used.
e. For after-load reduction in heart failure, especially with use of nifedipine.

It is likely that in future, calcium antagonists will be widely used in antihypertensive therapy.

4. **Other Applications**

a. In ischaemia or excessive sympathetic stimulation, excessive Ca++ influx into myocardial cells is a decisive factor contributing to necrosis. Calcium antagonists have been shown experimentally to be effective in protecting against or reducing such injury, and it has been proposed that they may be useful clinically in limiting the extent of myocardial injury and infarction due to ischaemia.
b. Vasodilators have been used in recent years for the "after load reduction" treatment of heart failure. Calcium antagonists especially nifedipine may be used in certain circumstances for this purpose.
c. Peripheral and cerebral vasospastic states may respond to calcium antagonists and have been used with success for the treatment of Raynaud s phenomenon.
d. Hypertrophic cardiomyopathy is a situation in which there is excessive myocardial contractility. Calcium antagonists, in particular verapamil, have been shown to be beneficial in some patients but the long-term benefit in reducing mortality remains to be proven.

PRACTICAL CONSIDERATIONS

1. **Dosage**

The usual therapeutic dosage range of the 3 most widely used calcium antagonists are

a. **Verapamil**

i. IV 5 to 10mg (o.15mg/kg body weight) for termination of supraventricular tachycardia, repeated in 5 to 10 minutes if necessary.
ii. Oral 40 to 120mg 6 to 8 hourly.
Higher doses are used by some workers.

b. **Nifedipine**

Orally or sublingually, 10-30mg 6 to 8 hourly.

c. **Diltiazem**

i. IV 0.25mg/kg body weight over 60 seconds.
ii. Orally 30-80mg 6 hourly.

2. **Major Adverse Effects**

Verapamil and diltiazem may cause bradycardia and conduction defects, while nifedipine

may cause reflex tachycardia. All may cause headache, flushing and dizziness due to vasodilation, and hypotension Oedema due to sodium retention may occur with all. Constipation is most often observed with verapamil.

3. **Precautions and Contraindications**

Caution should be exercised in the use of calcium antagonists in the following situations:

a. Impaired myocardial function, especially in combination with beta adrenergic blocking agents.

b. In combination with digitalis, especially if impaired AV conduction is present.

The principal contraindications are:

a. Severe left ventriuclar dysfunction.
b. Unstable 2^{o} or 3^{o} AV block.
c. Sick sinus syndrome, unless a pacemaker has been inserted.
d. Hypotension i.e. systolic BP less than 90mmHg.
e. Atrial flutter of fibrillation complicating Wolff-Parkinson-White Syndrome.

What is AIDS?

DR C L GOH,
MBBS, M. Med (Int. Med) MRCP
Senior Registrar, Middle Road Hospital, Singapore.

INTRODUCTION:

There has been much publicity about AIDS or Acquired Immunodeficiency Syndrome recently. What is this condition called AIDS? To most this is a deadly condition most commonly associated with homosexual males who after acquiring the disease succumb to opportunistic infections.

The condition was first recorded by Gottlieb and co-workers[1] in June 1981 when they reported 5 young homosexual men, who previously in good health developed Pneumocystis carinni pneumonia. Soon more case reports[2,3,4] of young homosexual males who were previously completely well and who developed an unusually severe form of Kaposi's sarcoma involving the skin and lymphoid organs were noted. These patients also developed opportunist infections such as pneumocystis pneumonia, cryptococcosis, or severe candidiasis which are usually seen only in the severely immunocompromised host.

Since then further epidemiological informations were collected and new immunological abnormalities noted. The exact cause of the disease remains unknown but it is hoped that this may be elucidated soon just like the 'mysterious' legionnaires' disease.

RISK FACTORS:

It is now known in the United States that at least 4 major population groups are at risk. About 75% of patients are homosexual males, 13% are intravenous drug abusers with no history of homosexuality, 6% are Haitian immigrants who are not homosexual and do not abuse drugs, 0.7% are hemophiliacs, and about 5% have no apparent risk factors.[5]

OPPORTUNISTIC INFECTIONS:

The list of opportunist infections has expanded to include most of the bacterial, fungal and parasitic agents customarily associated with cellular immunodeficiency. Recently a prodrome of AIDS consisting of the syndrome of fever, weight loss and generalised lymphadenopathy has been noted in the same at-risk population.[6]

TRANSMISSION:

The mode of transmission of the disease remains unknown and several postulates have been suggested. It is generally believed to be a disease transmissible through sexual intercourse, transfusions and transplacental means. On the notion that AIDS may be due to an infectious agent, the U.S. Centers for Disease Control have published recommendations for strict precautions by clinical and laboratory workers[7]. They should avoid direct contact of skin and mucous membranes with blood, blood products, excretions, secretions, and tissues of persons likely to have the syndrome.

MORTALITY:

The overall mortality from AIDS is 40%. Even those who recover initially die subsequently from malignant disease or overwhelming infection later[8]

PATHOGENESIS:

Here cellular immunodepression is severe. Lymphopenia and skin-test anergy are common and the in-vitro responsiveness of T cells to a range of mitogens is greatly depressed. Many patients show a striking reversal of the T-helper/T-suppressor ratio reflecting a reduction in the T-helper subset[10]. This reversal of ratio may be a useful indicator to early detection of the condition. In contrast, humoral immunity generally remains intact and in fact hypergammaglobulinaemia is the rule. The complement components are normal. Viral agents have been mentioned as possible triggering or contributing factors in AIDS. Severe hezpes simplex virus infections have been described[9]. There is also widespread seropositivity for Epstein-Barr virus and cytomegalovirus. The titre for the latter is usually high denoting recent infection or reactivation[11]. The overall assault leads to depressed cellular immunity ending in opportunistic infections and death.

SURVEILLANCE:

Until facilities for laboratory investigations of

patients with suspected AIDS becomes available the best thing to do is to be alert on the possibility of such a diagnosis. If the condition is really infectious then with time the condition will present itself all over the world. The relevant clinical features to look for is the sudden deterioration of previously healthy individuals in the high risk group (as mentioned above), the development of Kaposi sarcoma like lesions on the skin (these appear as disrete bluish nodules). The presence of lymphadenopathy and the presence of lymphopenia. Where available the antibody titre for Cytomegalovirus may be helpful. All patients will require inpatient treatment.

References

1. Gottlieb MS, Schanker HM, Fan PT, Saxon A, Weisman JD, Pozalski I,: Morbid Mortal Weekly Rep: 1981:30:250-52.
2. Friedman-kien A, Laubenstein L, Marmor M et al: Morbid Mortal Weekly Rep 1981: 30:305-08.
3. Hymes KB, Greene JB, Marcus A. et al: Lancet 1981:ii:598-600
4. Friedman SM, Felman YM. Rothenberg R, et al: Morbid Mortal Weekly Rep: 1981:30:409-10.
5. Centers for Disease Control: Morbid Mortal Weekly Report 1982:31:507-14
6. Centers for Disease Control: Morbid Mortal Weekly Report 1982:31:249-51.
7. Editorial: Lancet: 1983:i:164.
8. Editorial: Lancet: 1983:i:162-64.
9. Siegal FP, Lopez C Hammer GS, et al: N Engl J Med 1981:305:1439-44.
10. Fauci AS: Ann Intern Med 1982:96:777-79.

Cancer Research Grant

The Singapore Cancer Society will consider applications for cancer research grant from those engaged in cancer research. Application forms are obtainable from the Singapore Cancer Society, #06-03 & 04, Realty Centre, 15 Enggor Street, S'pore 0207.

All applicants will have to submit their application forms giving full details of the project and personal particulars to the Administrative Officer, Singapore Cancer Society through their respective heads of departments.

Closing date of application will be 30 September 1983.

Non-Specific Genital Infection (NSGI) of the Female

DR K B LIM
MRCP, DIp Ven, Middle Road Hospital, Singapore.

The term non-specific genital infection (NSGI) implies an infection of the genital tract for which no cause can be found. NSGI of the male is a urethritis and is therefore commonly called non-specific urethritis (NSU) or non-gonococcal urethritis (NGU). Although both terms are often used synonymously, this is incorrect as non-gonococcal means that only gonorrhoea has been excluded, while non-specific strictly implies that all known possible causes have been excluded, though this is rarely the case. The latter term is adopted here as it has become widely used despite the inaccuracy. NSGI of the female generally implies an infection of the cervix, but rarely the urethra may be the site of primary involvement. In contrast to NSU, which is characterized by a urethral discharge containing significant numbers of polymorphonuclear leucocytes in the absence of N. gonorrhoeae, NSGI of the female may be totally asymptomatic with only minimal signs. Thus, the diagnosis is made on historical evidence of sexual contact with a male partner suffering from NSU or on the culture of possible aetiologic agents from the cervix or in a patient with consistent genital signs in the absence of N. gonorrhoeae, Candida albicans or Trichomonas vaginalis.

This article discusses the difficulties associated with the diagnosis of NSGI in the female and attempts to bring together information that may help in its diagnosis. NSGI of the female is a rather vague entity and as such, its diagnosis is likely to be missed by the unaware. The complications are also considered in some detail to highlight the potential risks of not making the diagnosis. It is hoped that the reader's index of suspicion can be enhanced. This may halt or even reverse the upswing in the incidence of NSU now being seen in many countries.

Aetiology

C. trachomatis is probably responsible for a proportion of cases of NSGI in women. A number of studies report the incidence of chlamydial infection of the cervix in women attending clinics and the higher incidence found when they attended because the partner had NSU[1,2]. Tait et al[3] reported the clinical findings associated with chlamydial infection of the cervix and noted that the signs regressed after treatment. The isolation of C. trachomatis from the cervix and fallopian tubes of patients with acute salpingitis suggests that it plays an aetiologic role in pelvic inflammatory disease (PID).[5]

There is also evidence to suggest that Mycoplasma hominis may be implicated in some cases of PID. The role of Ureaplasma urealyticum (T. strain of mycoplasma) in PID is however, less clear.

Epidemiology

Impressions in the diagnosis of NSGI in the female hinders estimation of its true incidence. Indeed, prior to the isolation of C. trachomatis from the female genital tract, some physicians even disputed its existence as a clinical entity in the sense of a sexually transmitted disease. In England, where there has been nationwide recording of incidence data since 1951, a total of 27,410 new cases of NSGI in females and 86,896 cases of NSU in men were recorded in 1980. The male:female ratio was 3:1. This discrepancy is too great even after allowing for the few cases or urethritis due to non-infective causes, eg urethral stricture and the few cases of spontaneously recurrent NSU. If we accept that NSGI is a primarily sexually transmitted disease (STD), then the above figures suggest underdiagnosis or underreporting and worse still, failure to treat. Failure to treat may partly account for the continual rise in the incidence of NSU in the U.K. Furthermore, it exposes the female to the risk of complications (see below).

Clinical features

It is perhaps most frustrating to patients and to clinicians working in STD clinics that women should so commonly have asymptomatic infections for they not only represent a source of infection but are also a risk to themselves (from complications) and to the neonate during parturition. Like uncomplicated gonorrhoea, there may be no distinctive features of NSGI in women and the patient may be totally asymptomatic

with minimal signs. Even when symptoms are present; they are usually mild and include dysuria or a vaginal discharge. Examination commonly reveals no abnormality although occasionally there may be a cervical erosion with oedema and congestion or a mucopurulent cervical discharge. Unfortunately, none of these signs are diagnostic and chlamydial infection of the cervix may not always result in clinically apparent disease[4,6]. Colposcopic examination of the cervix may however show characteristic follicles similar to those seen in chlamydial infection of the eye[7]. A mucopurulent cervical exudate is also non-specific and is commonly seen in patients fitted with an I.U.D. Similarly, erosions are especially common among females taking oral contraceptives. Nevertheless, Tait et al[3] were able to establish an association between chlamydial isolation and the presence of a hypertrophic cervical erosion. 19% of women with such erosions and only 3% of women without erosions were chlamydia positive.

Diagnosis

Although there are imperfections of making the diagnosis of NSU in men, all venereologists now acknowledge its existence as a definite disease entity. This is not the case in NSGI in women. Adler[8], in surveying the diagnostic criteria for non-specific genital infection in STD clinics in England and Wales, found that only 60% of clinics for female patients recognised the condition as a distinct clinical entity even though the patient was not a sexual contact. He found that the two most commonly used criteria in such clinics were positive microscopical findings of leucocytes on the cervical slide (77% of the clinics) and positive findings on clinical examination such as cervicitis or mucopurulent or purulent cervical discharge or both (53% of the clinics). However, there was a variation between clinics in the number of leucocytes per high power field (HPF) that were used and in most clinics the number of leucocytes was not quantified.

Attempts have been made to establish criteria for the diagnosis of NSGI in women. Simmons and Vosmik[9], Burns et al[10] suggested that inflammatory changes on cytology might help in the diagnosis. Fox[11] suggested 50 or more leucocytes in several HPFs on the cervical slide in conjunction with inflammatory changes on cervical cytology as a means of diagnosis. However as women with gonorrhoea, trichomoniasis and to lesser extent, candidosis can also exhibit inflammatory changes on cytology, these conditions should be carefully excluded first. The presence of pus cells on the cervix is physiological and varies with the phase of the menstrual cycle. Thus microscopy of the gram stained cervical slide is of limited value although its presence in the absence of other STDs should raise the possibility of NSGI. Like in NSU, the number of leucocytes per HPF that constitute a positive microscopic criteria remains to be established.

Ideally, cultures for C. trachomatis, M. hominis and U. urealyticum should be undertaken but such facilities are available only to a few clinics. Moreover, even if they are available, the problem is only partially solved as there will remain those cases of NSGI where none of these organisms can be isolated.

Thus, the diagnosis of NSGI in women remains problematic and in practice, depends on a history of sexual contact with a male patient with NSU or positive cultures for C. trachomatis, M. hominis or U. urealyticum from the cervix. In the absence of these, the diagnosis may be made on the basis of clinical signs with consideration of microscopic findings on the gram stained cervical slide and cervical cytology. The imperfections of these have already been discussed but the clinician faced with a symptomatic patient often has to make a decision on even scanty evidence. Empirical treatment of such cases may be justified (see below).

Management of NSGI

Most clinicians working in STD clinic treat any patient who has been in contact with a man with proven or presumptive NSU. As it is not uncommon for STDs to coexist, an attempt should be made to exclude other STDs. In particular, female contacts should have smears and cultures taken for N. gonorrhoeae as the diagnosis in the male may be mistaken. If facilities are available cervical cultures should be undertaken for C. trachomatis, M. hominis and U. urealyticum. Serological lists for syphilis should also be performed.

In some cases, where no cause can be found for the patient's symptoms or signs, it may be useful to check the male partner for evidence or urethritis. This will provide indirect evidence of NSGI in the patient. Where this is not possible, for example, after a casual encounter it is reasonable to treat the patient empirically.

Treatment

Although many women receive needless treatment, it would be prudent under the prevailing state of knowledge to treat all NSU contacts

epidemiologically. It is possible that undertreatment is one reason for the continuing increase in NSU over recent years in western countries.

Treatment reduces the likelihood of complications developing and prevents reinfection of the male partner. In addition, it removes a possible source of infection to other male partners.

The same antimicrobials as for NSU may be used in the treatment of NSGI. A fourteen day course of treatment with one of the following antibiotics is usually adequate – oxytetracycline or tetracycline 250-500 mg qds; doxycycline 100 mg bd, minocycline 100 mg bd or erythromycin 250-500 mg qds. Erythromycin should be given to pregnant women. The patient should be advised against sexual intercourse until the male partner has been evaluated and treated.

Complications

Complications may develop in the untreated patient or in neonates born to such patients. Except for neonatal chlamydial pneumonia, these are equivalent to the complications of gonorrhoea.

Bartholinitis:

C. trachomatis have been isolated from exudates from Bartholin's ducts[12]. Chlamydial bartholinitis should therefore be considered when other causes of bartholinitis cannot be identified. A three week course of the above antibiotics is usually sufficient.

Pelvic inflammatory disease:

In western countries PID is more often non-gonococcal than gonococcal. Most of the studies on PID have been conducted in Sweden, where the diagnosis in the majority of cases was verified on laparoscopy. It has been found that PID in women 25 years or younger was associated with gonococci, mycoplasma and chlamydiae. Mardh et at[5], found C. trachomatis in the cervix of 19 of 53 patients with acute salpingitis and in material from the fallopian tubes of 6 of 20 valid specimens and concluded that chlamydia is a common aetiologic agent in this condition. Treatment consists of rest and anti-microbials. In severe cases a prolonged course of antibiotics may be required. Delays in diagnosis and treatment are often followed by considerable morbidity in the form of dyspareunia, chronic pelvic pain and infertility.

Chlamydial eye infection in the newborn:

This is an important complication of genital chlamydial infection. Infection occurs during parturition leading to non gonococcal ophthalmia neonatorum. The incubation period is 5-14 days but may be shorter. The condition is generally milder than gonococcal ophthalmia neonatorum, but, both conditions may coexist emphasizing the importance of obtaining cultures for both organisms. Contrary to popular belief, scarring and pannus formation may occur in apparently mild cases if left untreated. 40% of babies born to mothers from whom chlamydia has been cultured from cervical material will develop ocular infection. The infection should be treated with 1% tetracycline eye ointment applied to the lower lid qds and erythromycin suspension 40 mg/kg/day in divided doses for 3 weeks.

Neonatal chlamydial pneumonia:

This condition was first described by Beem and Saxon[13] in black infants. The pneumonia typically starts at the age of 2-3 weeks but is only usually diagnosed at 6 weeks or more. The features include a characteristic staccato cough, a mucoid nasal discharge in a usually afebrile child. Respiratory distress may develop and apnoeic attacks may occur. Inspiratory rales are present on auscultation but there are no or minimal expiratory rhonchi. Chest x-ray shows diffuse infiltration and hyperinflation of the lungs and the diagnosis can be confirmed by isolating C. trachomatis in nasopharyngeal secretions or by the finding of high titres of antichlamydial serum antibodies by the micro IF test. Treatment is with physiotherapy and erythromycin 40 mg/kg/day in divided doses for 3 weeks.

Conclusion

The incidence of NSU has increased dramatically over the past two decades. Statistics from the U.K. show that from 1951 to 1965, gonorrhoea was more frequent. However, from 1965 onwards the incidence of NSU has become greater than that of gonorrhoea in men, and the gap continues to widen. One reason for the increase may be the failure to detect and treat asymptomatic infections in the female.

In Singapore there is evidence to suggest that NSU is on the increase. In 1982 there were 499 cases as compared to only 252 in 1981. These has therefore been a nearly two-fold increase in incidence. As NSGI in the female is often asympto-

matic, all female contacts of men with NSU should be enthusiastically traced, evaluated and treated. In spite of the lack of diagnostic features, clinicians should recognise its existence in women who are not themselves contacts of men with NSU and actively look for evidence of NSGI. Vigorous attempts should also be made to trace male contacts of such patients. Such measures have to be implemented if we are to prevent NSU from reaching the proportions now being seen in many western countries.

References

1. Hilton A L, Richmond S J, Milne D et al Chlamydia A in the female genital tract Brit J Vener Dis 1974; 50:1-10.
2. Oriel J D, Povis P A, Reeve P et al Chlamydial Infection of the Cervix Brit J Vener Dis 1974; 50:11-16.
3. Tait I A, Rees E, Hobson D et al Chlamydial isolation of the cervix in contacts of men with non gonococcal urethritis Brit J Vener Dis 1980; 56:37-45.
4. Rees E, Tait A, Hobson A et al Chlamydia in relation to cervical infection and pelvic inflammatory disease. In: Hobson D and Holmes K K (eds) Non gonococcal urethritis and related infections, American Society for Microbology, Washington, D C 1977a, 67-76.
5. Mardh P A, Ripa T, Mitchell S H Chlamydia trachomatis infection in patients with acute salpingitis New Eng. J Med 1977; 296:1377-9.
6. Oriel J D, Johnson A L, Barlow D et al. Infection of the uterine cervix with C. trachomatis J. Infect Dis 1978; 137:443-51.
7. Dunlop E M C, Hare M J, Darougar S et al Infection of the eye, genitalia and rectum by a new serotype (Type G) of TRIC agent Brit J Vener Dis 1973; 49, 301-5.
8. Adler M W Diagnosis, treatment and reporting criteria for non-specific genital infection in sexually transmitted disease clinics in England and Wales, 1: Diagnosis Brit J Vener Dis 1978; 54:422-7.
9. Simmons P D, Vosmik F Cervical cytology in non-specific genital infection: an aid to diagnosis. Brit J Vener Dis 1975; 50:313-4.
10. Burns D C, MacD, Darougar S et al. Isolation of chlamydia from women attending a clinic for sexually transmitted disease Brit J Vener Dis 1975; 51: 314-8.
11. Fox H Non-specific genital infection in a general practice Brit J Vener Dis 1974; 50:125-31.
12. Davies, J A, Rees E, Hobson D et al Isolation of chlamydia trachomatis from Bartholin's ducts Brit J Vener Dis 1978; 54:409-13.
13. Beem M O, Saxon E M Respiratory tract colonization and a distinctive syndrome in infants infected by chlamydia trachomatis New Eng J. Med. 1977a; 296:306-10.

Epiloque

The Tenth WONCA World Conference on Family Medicine

The Tenth WONCA World Conference has come and gone. It was of special significance to us because the College was the host of this World meeting. Judging from the favourable comments of our delegates and the standing ovation received by the Host Organising Committee at the Closing Ceremony, we can infer that we had not done badly in the organising of this Conference.

The Tenth WONCA World Conference on Family Medicine was to date, the largest medical meeting held in Singapore. A total of 1,315 delegates and 534 accompanying persons attended. It was also a Conference of other "firsts". It was the first time that an audio-visual "curtain raiser" was introduced into the Opening Ceremony in a Medical Conference in Singapore. It was the first WONCA Conference that had its Conference proceedings ready before the end of the Conference and it was the first time that a WONCA Conference had a daily bulletin throughout the duration of the Conference.

All these achievements were made possible through the dedication of the band of doctors both directly and indirectly involved in the organisation of this Conference.

Perhaps the best testimonial is that given by Dr Edward J Kowalewski, WONCA's Conference Planning Committee Liaison Person:

> "The Host Organising Committee has made this meeting possible. It has demonstrated dedication, knowledge of what the contents of the Scientific meeting should be, administrative and financial expertise. They have set a high standard in the Scientific programme. Underlying all these is the extremely hardworking and energetic Host Organising Committee. This Host Organising Committee is by far the most professional of the non-professionals in the organisation of such a meeting."

It may not be out of place here to recount the key factors that contributed to the successful outcome of this Conference.

The Host Organising Committee was entrusted by the College the task of organising this Conference. It consisted of the following members: the Chairman, Deputy Chairman and Treasurer, Organising Secretary, and four Sub-Committees, each under a Sub-Committee Chairman. The Sub-Committees were the Scientific, Social/Publicity, Publications and Exhibition Sub-Committees. The Host Organising Committee was linked to WONCA through the Conference Planning Committee which consisted of three persons, (the Host Organising Committee Chairman, the Secretary and Treasurer of WONCA and the WONCA Conference Liaison Person.)

The Scientific Sub-Committee was entrusted the task of drawing up a Scientific programme of sufficient depth and content befitting the image of WONCA. Efforts were made to ensure a worldwide participation and to have papers of geo-political spread as well as different levels of development of family medicine.

The Scientific programme was made up of five segments, consisting of (1) a keynote address, (2) 4 plenary sessions and 3 plenary workshops, (3) 10 WONCA Standing Committee Open Forums, (4) 16 Free Paper sessions delivering a total of 111 Free Standing Papers and (5) 3 Workshops/Seminars.

A micro-computer was found very useful in the tabulation of the speakers, their countries of origin, their topics as well as keeping track of speakers yet to submit their papers. To achieve date, time and place of delivery of their papers, a packaged software programme, the CONDOR 20 Database Management Programme, was used. The micro-computer also made it very easy to send out a final confirmation letter to each speaker, giving information of the date, the time, the venue and the title of his paper. This took two hours to prepare compared to two days if done manually.

The Social/Publicity Sub-Committee was responsible for putting together an interesting programme for the delegates and accompanying persons. The participants were given a treat at the Opening Ceremony to an audio-visual curtain raiser on the work of the Family Doctor, as well as a buffet of our cosmopolitan cuisine. Then there were South East Asian arts and crafts on display. For the Closing Ceremony, there was a non-stop variety show. This drew huge applause.

Through Orient Vacations, a wide variety of tours and shows were also made available to the delegates and accompanying persons to make their stay in Singapore a memorable one. Credit must be given to the good service provided by Orient Vacations, the official conference travel agent.

The Social Sub-Committee had also to publicise the Conference. A total of three mailings were conducted to woo the potential delegates.

The Publications Sub-Committee took charge of the production of the Conference proceedings as well as the Daily News Bulletin. Our publisher, Mr Benett Austin, worked very hard to make the publications possible. In fact, for each edition of the Newsletter, the type-setting was completed at around midnight, the printing completed the next day by about 4 a.m. and thereafter for circulation at 8 a.m. The News Bulletin concentrated on three areas, current conference news, features on WONCA's activities and features of interest to the accompanying persons on shopping, sightseeing and fashion.

Through "staged calls", the last being in March, for the submissions of full papers, a total of 139 out of 168 papers presented were received in time for inclusion in the Proceedings.

The Exhibition Sub-Committee staged the Meditech '83 Medical Trade Exhibition. Coming at a time of general economic recession as well as competition from Medic Asia, the Committee had to work very hard to put up the 36 booths of Trade Exhibition. The success of the Exhibition however, more than paid for the labour put in by the Sub-Committee.

Perhaps, what was often overlooked but nevertheless of great importance was the administrative work put in by the Secretariat to ensure the Conference went on without a hitch. It was the Host Organising Committee's policy to keep member countries well informed of the latest developments in the Conference. The response was most encouraging. This enabled the Committee to function without any significant hitches. We were also fortunate to have a very able Administrative Secretary in Miss Janet Ho. Mention also must be made of Miss Teo Siew Gek and Miss Teresa Loke who helped out during the last few months before the Conference. They were unbeatable in efficiency.

In the course of the organising some hard decisions had to be made. First, the stipends and "benefits" of the speakers had to be finalised. Then there was the problem of simultaneous translation of English into languages like French and Spanish. This could not be catered for because of the prohibitive cost. The non-English speaking countries were co-operative in allowing English to be the only Conference language. Perhaps, the hardest decision was whether to stage the Closing Ceremony in one or two venues, because of the large number of delegates and accompanying persons attending this Conference. As it was not possible to know the exact number of guests attending until the very last day, the decision had to be based on prediction of the number of people likely to attend. It was just as well we banked on two venues. Both were packed!

Finally, a word or two must be said about Conference manpower. The number of people we had was very small. We were indeed grateful for the wives of many of the Host Organising Committee members, who chipped in to fill the Conference satchels and handle the reception duties. Secondly, the small numbers of the Organising Committee allowed for better interaction between members and for decision making.

The Tenth WONCA Conference provided Singapore the opportunity to make itself known a little better to the world platform of primary care doctors.

It has also benefited Singapore in another way. Hosting the Conference here allowed us the opportunity of sharing at first hand the experience and thinking of top family doctors from all over the world. This coming at a time when Singapore is looking into the incorporation of Family Medicine into the undergraduate and postgraduate curricula is timely. The post WONCA Conference challenge has just begun.

ALFRED W T LOH

Practice Management

An Introduction

A new area of medical practice thinking is coming into focus. This is Practice Management. In keeping with the Council of the General Practitioners, Singapore has set up a Practice Management Committee.

Terms of Reference

The Committee has set itself the following terms of reference.

1. To define the scope of Practice Management
2. To collect a dosier of information on the key aspects of Practice Management.
3. To disseminate useful collected information through the Singapore Family Physician and similar avenues.
4. To stimulate exchange of ideas through seminars and workshops on key aspects of Practice Management.
5. To conduct relevant research on Practice Management.
6. To consider the set-up of a model practice.

Definition of Practice Management

As a working definition, Practice Management may be defined as the study into ways and means whereby the Practice can be run efficiently and cost effectively in the delivery of its medical services.

Scope of Practice Management

The scope of Practice Management is outlined in Appendix I. Arbitrarily, the various activities of Practice Management can be grouped into 4 areas namely:

1. Activities related to the organisation of the Practice to provide medical services.
2. Activities related to practice administration.
3. Activities related to the practice's outside relationship.
4. Applied Research.

Practice Management Section

In forthcoming issues of the Singapore Family Physician, the Practice Management Committee will be contributing a Section on Practice Management thought, ideas and innovations.

An Invitation

We invite readers to send in their comments, ideas and feedback on this Practice Management Section.

Send your communications to:
The Chairman,
Practice Management Committee,
c/o College of General Practitioners, Singapore
4-A College Road, Singapore 0315.

Appendix I
SCOPE OF PRACTICE MANAGEMENT

1. Organisation of the Practice to Provide Medical Services
- * Setting up Practice
- * Types of Practices
- * Emergency Medical Services
- * Doctor's Call Bag
- * Medical Equipment
- * Dispensing
- * Medical Records
- * Proctocols For Patient Care & SMEs
- * Keeping Abreast
  - Journal Reading
  - Information Dosier on
    - Drug dosages & interactions
    - Normal values
    - Procedures
- * Starting a small medical library
- * Organisation to train students

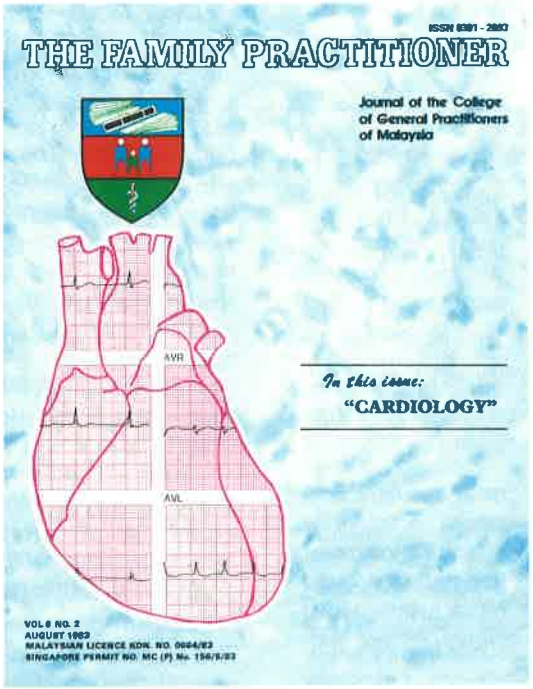
2. Practice Administration
- * Office Management
  - Reception
  - Appointment
  - Recall
  - Storage & Retrieval of Records
  - Correspondence
- * Practice Staff Management
  - Recruitment
  - Induction Training
  - Vocational Training
  - Supervisory Training
  - Motivation & Career Development
  - Terms of Employment
  - Management of Change
- * Financial Management
  - Practice Accounts
  - Billing & Accounts Receivable
  - Equipment – purchase and lease
  - Taxation
  - Investment
  - Insurance
- * Inventory Control

3. The Practice's Outside Relationships
- * Medical Practice Laws
- * Counselling & Health Education
- * The Health Care Team

4. Applied Research
- * Communication Problems
- * Manpower
- * Practice Characteristics

GLG

The Family Practitioner



'The Family Practitioner' is the official journal of the College of General Practitioners of Malaysia. It is published three times a year. The inaugural issue appeared in June 1973. There are a number of regular features:

(1) **Symposium:** Each issue is devoted to a certain theme of interest to family doctors eg. the August, 1983 issue features 'Cardiology'. Although at present the majority of the articles is contributed by specialists who are authorities in their particular field, we try to strike a balance of inviting family doctors to write certain articles. Eventually we hope family doctors will contribute 50% of the articles in our symposia. This will reflect the state of development of Family Medicine in our country.

(2) **Articles and Papers:** In this section we publish original papers and review articles. This section is open to all members of the medical profession, as long as their articles are of interest to family doctors. We hope that this will serve as an inducement for family doctors to write scientific articles.

(3) **College News**

Family Practice Around the World: Our journal also serves as a newsletter to disseminate information on Family Medicine both at home and abroad.

Cover:

The cover features the College Crest with the motto DENGAN TATANGAN ILMI, which means 'Care with Knowledge' in Bahasa Malaysia.

There is also a photograph/drawing illustrating the theme. The background is blue and is actually an enlargement of a clinical slide.

Although at present 'The Family Practitioner' is primarily a journal of information, and features mainly the clinical content, on 'care knowledge' of Family Medicine, we hope that as family doctors contribute more articles, we will be able to present the progress of Family Medicine in our country.

"The Family Practitioner" write-up is contributed by its present Editor Dr TM Ho to whom the SFP is indebted. ED.

HOME STUDY SECTION

Pleural Effusion

Dr W C TAN

MBBS (Aberdeen); MRCP, FRACP,
Senior Lecturer,
Department of Medicine,
National University of Singapore

Introduction

Accumulation of excessive amounts of liquid in the pleural space is a frequent manifestation of serious thoracic disease usually cardiac or pulmonary. Often it is the first manifestation of an extrathoracic or systemic disease. Pleural effusions, regardless of size, should always be considered as an ominous warning of a serious underlying disorder.

Diagnostic Evaluation

Patients with pleural effusions are usually seen by physicians because of dyspnoea or pleurisy or symptoms related to the underlying cause of the disorder. Only occasionally are patients found to have an unsuspected pleural effusion when physical or radiographical examinations are performed for some other purpose.

The most common symptom is breathlessness, patients may describe a "heavy" sensation or a dull pain on the affected side. Classic pleuritic pain may or may not occur depending on the cause of the disorder.

Associated symptoms are often more valuable than symptoms of the effusion itself, in defining the cause. A history of exertional dyspnoea, ankle swelling, hypertension, rheumatic fever or known heart murmur points to congestive heart failure; progressive weight loss and weakness suggest malignancy; heavy smoking, cough and haemoptysis suggests a primary bronchogenic carcinoma; recent onset of high fever with chills suggest an empyema.

The signs of a pleural effusion include dullness to percussion, decreased fremitus and breath sounds. Compression of the lung just above the effusion often decreases the gas content per unit volume, thereby producing the physical findings of consolidation. Thus, bronchial breath sounds, increased transmission of whispered and spoken voice and aegophony are common manifestations of pleural effusion.

Radiographic Evaluation

The amount of fluid necessary to produce blunting of the costophrenic angle is usually 500 to 600ml. The classic density is higher laterally, the typical 'S' shaped curve. An airfluid level is due either to gas introduced into the pleural space such as after thoracocentesis, communication with the tracheobrochial tree or infection with a gas-forming microorganism.

Effusions may be trapped or loculated. Empyemas have a particular tendency to loculate posteriorly. Small effusions may be distinguished from pleural adhesions by taking films of the chest in both lateral decubitus positions.

The chest x-ray may also provide clues to the cause of the effusion such as an enlarged heart or valvular calcification suggests cardiac failure, a tumour mass indicates malignancy, and parenchymal densities point to pneumonia.

Thoracocentesis

The two important reasons for performing a thoracocentesis are:

1) to obtain fluid for diagnostic examination.
2) to remove liquid to alleviate symptoms.

Percutaneous pleural biopsy using the Abrams needle is now a routine part of the diagnostic evaluation of patients with pleural effusion of unknown origin. It is best performed when there is an ample collection of pleural fluid. Pleural biopsies are particularly rewarding in the diagnosis of tuberculous effusion and carcinoma of the pleura (one half to three quarters of the patients).

Pleural Fluid Analysis

A standard group of tests is usually performed including gross and microscopic examinations of the fluid, determination of the concentration of total proteins and bacteriological and cytological studies.

Conventionally, pleural fluid with a protein concentration of less than 3g/dl is a **transudate** and one greater than 3g/dl is an **exudate.**

The common conditions associated with transudates are left ventricular failure, associated with ascites in hepatic cirrhosis, nephrotic syndrome and renal failure.

EXUDATES

The common causes of exudate effusions are:

1. Parapneumonic: Often sterile, but the presence of viable bacteria may progress to form an empyema. The predominant cellular content is polymorphonuclear leucocytes. Effusions due to viral and mycoplasma are usually small and transient.
2. Tuberculous. The typical cytological sign of a tuberculous effusion is lymphocytosis. In the early stages of the effusion, granulocytes are often present.
   In contries where tuberculosis has become rare, a lymphocytic picture is found more commonly in lymphoma and carcinoma. Tuberculous effusion are controlled by aspiration to dryness and effective tuberculous chemotherapy.
3. Connective Tissue Disorder
   Pleuritis and pleural effusion are usually late manifestations of systemic lupus erythematous. About 25% of patients have evidence of effusion at autopsy. Pleurisy with effusion occurs in up to 5% of patients with rheumatoid arthritis and particularly in male patients with subcutaneous nodules. Spontaneous resolution is the role.
4. Hypersensitivity to drugs such as nitrofurantoin, methysergide, practalol, isoniazid and diphenylhydantoin may occasionally cause a small pleural effusion which resolve when the drugs are discontinued.
5. Haemorrhagic effusions are commonly caused by neoplasm, pulmonary infarctions, trauma and rarely by tuberculosis.
6. Chylons effusion. This milky effusion is due to exchange of lymph into the pleural cavity caused by trauma or obstruction from carcinoma or lymphoma.

Empyema

This term is used to describe a collection of pus in the pleural cavity. Sometimes it is not frankly turbid but microscopically leucocytes and pus cells are abundant. Empyemas are more characteristic of infections with some microorganisms than with others: staplylococcus aureus, streptococii (aeorbic and anaerobic), mixed anaerobic bacteria, Klebsiella pneumoniae, pseudomonas pyocyaneus, E Coli and amoeba. In general, all empyemas should be treated promptly by tube drainage and control of the infection with suitable antibiotic. Only rarely is thoracotomy with decortication necessary to remove a greatly layer on the pleural surface.

Summary

Pleurisy with effusion is not a disease entity but must be regarded as a sign of an important underlying disorder. Diagnostic efforts are always warranted, and a specific diagnosis can be expected in most patients.

References

- Mestitz P, Purves MJ, Pollard AC: Pleural biopsy in the diagnosis of pleural effusion. A report of 200 cases. Lancet 2:1349-1353, 1958.
- Von Hoff DD, Volsi VL: Diagnostic reliability of needle biopsy of the parietal pleura: a review of 272 biopsies. Am J Clin Pathol; 64:200-203, 1975.
- Pulmonary Diseases and Disorders. Alfred P Fishman, McGraw-Hill Book Company. Pleural Disease, Page 1357-1380.

Respiratory distress in infants

DR K VELLAYAPPAN MBBS, M Med(Paed), FRACP

It is not uncommon for an infant or child to have respiratory infections every two to three months in the first three years of life. Many children will overcome each episode successfully within a few days and are in excellent health in between these episodes of infection. However a certain percentage of children do not completely recover or have continuing symptoms of the respiratory tract. These children need to be evaluated carefully, so that we can diagnose their condition and treat them appropriately. They could be suffering from any of these illnesses – bronchial asthma, foreign body in the respiratory tract, bronchiectasis, pulmonary tuberculosis, cystic fibrosis, immunodeficiency, or pulmonary haemosiderosis. In newborns, respiratory distress occurs more commonly in preterm and small for date infants, in babies with congenital heart disease and congenital malformations of the respiratory tract.

NEW BORN INFANT

It is important for the physician looking after a newborn to decide whether the infant has respiratory distress and if so, the cause must be diagnosed immediately, so that definitive treatment can be given.

Breathing in the newborn is quite different from that of the child or adult. Irregularity of respiration is common, even in mature infants. The more premature the infant the more irregular it is. Periodic breathing is commonly seen in preterm babies and sometimes in term infants. In this condition periods of apnoea alternate with periods of relatively rapid breathing. The periods of apnoea do not last longer than 10 seconds. When apnoea lasts longer than 10 seconds, is frequent and is associated with bradycardia, one must always suspect a pathological process. The relatively high compliance of the infant's chestwall results in the tendency of the intercostal muscles to draw inward and the costal margin to recede during inspiration.

CAUSES OF RESPIRATORY DISTRESS IN THE NEONATE

A Congenital Malformations

i) Pierre Robin Syndrome (Receding chin, cleft palate, glossoptosis).
ii) Cloanal atresia.
iii) Tracheooesophageal fistula and oesophageal atresia.
iv) Laryngeal problems – webs, haemangioma, cyst, laryngomalacia.
v) Vascular ring.
vi) Diaphragmatic hernia.
vii) Lobar emphysema.
viii) Oesophageal duplication.

B Hyaline membrane disease.
C Aspiration Penumonia.
D Apnoea of the preterm infant.
E Respiratory infections
– intrauterine
– extrauterine
F Pulmonary haemorrhage.
G Wilson-Mikity syndrome.
H Congenital heart disease.
I Diseases of central nervous system
– CNS depression by drugs
– CNS infection
– CNS haemorrhage

The infant with respiratory distress is easily identified because of the onset of rapid breathing with or without cyanosis. A respiratory rate greater than 40 per minute after the first hour of life is regarded as tachypnoea. Subcostal retractions and expiratory grunt are indicative of moderate to severe respiratory distress. Apnoea is also a sign of respiratory distress. A neonate with any of these signs should be regarded as having a cardio respiratory problem.

The maternal history can often give a clue to the attending physician to the possible cause of the respiratory distress. If there is a maternal history of polyhydramnios, the infant may be suffering from atresia of the upper gastro intestinal

tract and aspiration pneumonia is likely in these infants. A neonate with intrauterine infection may develop respiratory distress as a result of congenital heart disease or pneumonitis. Infants of diabetic mothers are at increased risk of having congenital heart disease or developing hyaline membrane disease. If the delivery of the infant was difficult respiratory distress may develop as a consequence of meconium aspiration. If there has been prolonged rupture of membranes, respiratory distress may develop due to pneumonia or sepsis. Transient tachypnoea develops in infants delivered by Caesarean section. This is thought to be due to the insufficient removal of pulmonary fluid. Excessive maternal analgesia and anaesthesia during delivery may cause respiratory depression.

Accurate knowledge of gestational age is important in determining whether the light weight infant is either a preterm or small for date infant. A detailed physical examination using the Dubowitz criteria can distinguish between the two. Preterm infants have a high incidence of HMD and small for date infants develop respiratory distress due to meconium aspiration.

The time of onset of respiratory distress and the condition of the baby at birth may give a hint to the aetiology of the respiratory distress. Preterm infants develop respiratory distress due to HMD at or very soon after birth. On the other hand infants with meconium aspiration may not develop respiratory distress for many hours after birth. Similarly infants with congenital heart disease may be very well for many hours after birth before they develop any respiratory difficulty. Increasing respiratory distress in a neonate who had been stable with HMD may signify development of congestive heart failure due to patent ductus arteriosus.

A careful clinical examination may help to define the cause of the respiratory distress. A receding chin with glossoptosis suggests Pierre Robin Syndrome. If the neonate develops respiratory difficulty each time he is fed, a diagnosis of choanal atresia is a possibility. A scaphoid abdomen, and a 'dextrocardia' in a neonate with respiratory distress strongly suggests a diaphragmatic hernia. Presence of a significant murmur and hyperdynamic heart sounds point more towards a cardiac pathology for the respiratory distress.

A chest X-ray is a very useful and simple investigation to determine the cause of the respiratory distress.

HYALINE MEMBRANE DISEASE (HMD)

This is a disease commonly seen in preterm infants. It has been known to occur in infants of diabetic mothers, infants delivered by caesarean section, in the second twin and in asphyxiated and shocked infants. HMD is rare in the full term infant but does occur. Certain factors reduce the risk of developing HMD — intrauterine growth retardation, prolonged rupture of membranes, prenatal administration of steroids and maternal heroin addiction.

Deficiency of pulmonary surfactant with resultant collapse of the alveoli and a decrease in the functional residual capacity explains the pathophysiology of the disease. Two chemical pathways play a part in the synthesis of the pulmonary surfactant — the choline pathway and the phosphatidyl ethanolamine pathway. Synthesis of the surfactant occurs in type II alveolar penumocytes. Perinatal asphyxia added to the background of surfactant deficiency may play an equally important part in the causation of HMD.

Clinical features of HMD are the expiratory grunt which comes on very soon after birth, tachypnoea with subcostal retraction and cyanosis if the disease is severe. The clinical course is variable in terms of severity with a peak between 48 to 72 hours. Increase in severity of the disease is manifest by increase in oxygen requirement, marked subcostal and intercostal retractions and the development of frequent apnoeic spells. The expiratory grunt may disappear as the disease worsens.

A chest X-ray in HMD shows a reticular granular pattern with air bronchogram effect in the lung parenchyma.

Neonates with HMD are nursed in the incubator or under a flourescent light to give them sufficient warmth to maintain body temperature between 36 and 37°C. Gentle handling is important and adequate fluids, electrolytes and calories are provided initially by means of an indwelling umbilical catheter. Anitbiotics are usually not necessary but may have to be given in a sick infant. Oxygen therapy is essential and the goal is to maintain a PaO2 of 50-70 mm Hg and minimise the toxic effects of oxygen. Thus the F102 should be varied to maintain PaO2 within this range. Transcutaneous monitoring of oxygen is useful and is fairly accurate for this purpose. If adequate oxygenation is not achieved by high F102 (greater than 0.6), oxygen has to be administered by CPAP (Continuous positive airway pressure). Generally airway pressures between 2 and 7 cm water are recommended. Pneumothorax is a complication of this therapy and occurs three times more commonly that infants not receiving

this therapy. CPAP may also interfere with venous return to the heart resulting in a lowered cardiac output with a risk of peripheral vascular collapse. When there is no improvement with CPAP and respiratory failure is imminent assisted ventilation is necessary.

MECONIUM ASPIRATION

Meconium aspiration is usually seen in small for date infant, full term infant with foetal distress and in the postmature infant.

As a result of an intrauterine insult, usually leading to hypoxia, these infants pass meconium into the amniotic fluid and aspirate the meconium when they make gasping movements in utero.

Respiratory distress may develop immediately after birth but more commonly it comes on within 12-24 hours. Tachypnoea with hyperinflated chest is the usual clinical feature. Cyanosis is apparent if the disease is severe.

At delivery, oxygen should be administered and meconium should be aspirated by direct visualisation of the trachea. A chest X-ray must be done and it typically shows coarse streaks of atelectasis associated with hypertranslucent areas with increased A-P diameter of the chest and flattened diaphragms.

Management is supportive and it consists of giving sufficient warmth, fluids, electrolytes, calories and oxygen. Administration of broad spectrum antibiotic is essential. A deterioration in the clinical condition should suggest that a pneumothorax or pneumomediastinum has developed and a chest X-ray will readily confirm this and these complications should be treated accordingly.

APNOEA OF THE PRETERM INFANT

Cessation of respiration of more than 10 seconds in association with bradycardia is termed apnoea. Apnoea may be primary or secondary to a pathological process usually an infection. Treatment consists of cutaneous stimulation and nursing these infants in an enriched oxygen environment. In spite of these measures if apnoea is still a problem aminophylline can be administered orally. Aminophylline is a respiratory stimulant and it also increases alveolar ventilation. CPAP can be tried if treatment with aminophylline is ineffective. Pathological causes must alway be excluded and treated appropriately.

PNEUMONIA

In any newborn infant with respiratory distress, pneumonia should always be considered as a possible diagnosis. Infection in the newborn are acquired during the birth process or from a nursery personnel. Infants born after prolonged labour, premature rupture of membranes are at increased risk of developing pneumonia.

Presently the commonest organism causing neonatal pneumonia is Beta haemolytic streptococcus group B. The other organisms are staphylococcus aurens and E. coli. Viral pneumonia though rare can also occur in the newborn and the respiratory syncytial virus has been isolated in outbreaks in the newborn nursery. A broad spectrum antibiotic should be used in the treatment of neonatal pneumonia and a combination of penicillin and aminoglycoside is commonly used in many neonatal units.

CONCLUSION

In this article, I have outlined the various causes of respiratory distress in the neonate and highlighted a few of the common conditions. In any newborn with respiratory distress, a detailed maternal and perinatal history is important and a plain X-ray of the chest will help greatly in the diagnosis of the infant's problem. There is always the dilemma whether the respiratory distress is due to a disease process in the respiratory tract or in the cardiovascular system and even a careful history and clinical examination may not solve this problem. Apart from a plain chest X-ray, an EGG and Echocardiography can be of great value in determining the presence and type of cardiac condition. ■

The Outpatient Management of Bronchial Asthma

DR Y C CHENG
MBBS. M. Med. (int. Med) (S'pore), MRCP (UK)
Senior Registrar, Department of Medicine III,
Tan Tock Seng Hospital, Singapore

A. Diagnosing asthma clinically

The typical case of asthma is easily diagnosed. However a number of clinical presentations which respond well to asthma treatment are often missed. For example the isolated cough after a respiratory viral infection, after exercise, or after an occupational exposure may respond well to asthma treatment but may not to a vaporiser, antibiotic, cough medicine or expectorants. Isolated breathlessness or chest discomfort often responds to a beta-agonist. Occasionally the airway obstruction of asthma is felt as a sense of blockage in the throat. Mild obstruction may sometimes present as symptomatic hyperventilation and requires the usual treatment for airway obstruction. The smoker with chronic bronchitis should receive at least one vigorous trial of therapy with an asthma regimen including ingested adrenocortical steroid carefully monitored with pulmonary function tests. In practice, the response to a trial of therapy provides an important confirmation of the diagnosis.

Wheeze arises when air flows through narrowed large airways. It is now recognised that this airways narrowing is not commonly due to direct blockage but rather to dynamic compression during expiration, in association with small airways obstruction. Wheezing is an extremely common symptom in children, but wheezing is not a normal finding. Recurrent wheezing in children is almost always due to asthma. Wheezing in adults usually denotes airways obstruction; this possibility can be simply confirmed by observing a forced expiratory time in excess of five seconds.

The basic problem in asthma is that the bronchial tubes are too sensitive and they narrow easily in response to a number of factors. These factors which may trigger asthma include exercise, irritants like cigarette smoke, dust, sprays (e.g. hair spray), and strong odours, cold air, and other weather conditions, allergy, infection and emotion. The importance of each of these factors varies from person to person. Some may have no effect on the person at all, while others may be so important that treatment in relation to them clears the asthma altogether. It is therefore necessary to consider the triggering factors in planning treatment. If attention is paid only to the therapeutic program of symptomatic medication and the associated or inciting factors are left unattended, then the chronic state of the disease is destined to continue.

A sizeable number of asthmatic patients have associated sinusitis and they are not responsive to the usual bronchodilating drugs until proper medical or surgical care of the sinusitis is instituted(1). Based on present-day concepts of the pathogenesis of bronchial asthma, three possible mechanisms by which paranasal sinus disease could produce asthma are firstly, bacterial seeding of the lung from the sinuses. Mucopurulent material may drip into the pharynx from the infected sinus, involve the mucous membranes of the trachea and bronchi and cause secondary bronchitis resulting in asthma. Secondly, reflex bronchospasm through the parasympathetic nervous system. Thirdly, enhancement of beta-adrenergic blockade. Partial beta-adrenergic blockade is present in patients with bronchial asthma(2). Infections of the respiratory tract (and the sinuses are part of the respiratory tract) are known to enhance this blockade, resulting in an increase in the hyperirritability of the bronchial tree.

B. Help from the Laboratory

1. Spirometry

Asthma is defined as reversible airflow obstruction. A 15 to 20 per cent variability in forced expiratory volume in one second (FEVI) or peak flow rate (PFR) provides the diagnosis. This improvement can be obtained with an inhaled beta-agonist in patients with resting airway obstruction. Alternatively a 20 per cent fall in FEV1 can be provoked by exercise or inhalation of cold air, histamine or methacholine if resting flow rates are normal.

Provocation tests and spirometry also provide a quantitative assessment of severity.

2. Histamine inhalation test

An increased response of airway smooth muscle is so characteristic of asthma that it has been proposed as a criterion for the diagnosis of the disease(3) and the measurement of bronchial reactivity has been used as a diagnostic test in patients with unexplained cough or dyspnoea (4,5). The measurement of bronchial reactivity is considered by some "as essential to the diagnosis and management of asthma as the glucose tolerance test is to diabetes" (6). Airway responsiveness to histamine is either an important determinant of the severity of asthma and the medication requirements or a consequence of the severity of asthma(7). The severity of asthma is usually assessed by history, examination and measurement of spirometry or peak flow rates. Physical examination is an insensitive method of assessing severity(8). Spirometry may be normal when the person is seen in the clinic but low at other times of the day. As a result patients may inadvertently be undertreated or overtreated on the basis of clinical and spirometric information alone.

Inhalational histamine challenge to measure bronchial reactivity is done locally(9) and the side effects of the test are minimal. It takes about 20 minutes to perform and the result is known immediately. It is done as an outpatient procedure but certain antiasthmatic medications must be withheld before the test can be reliably interpreted.

3. Allergic investigation of asthma

This aims to establish whether or not the patient's asthma has an allergic component, what type of allergic reaction is present and whether there are single or multiple allergies.

Clinical history focusses on variations of symptoms on exposure to dust, domestic pets, moulds, occupational chemicals and the effect of work or visiting specific places or houses. Immediate asthmatic reactions are easily recognised by the patient but late reactions occurring 6 hours after exposure may not be easily related to allergen exposure. There is little evidence that food allergens are important in chronic asthma although foods, food additives and food preservatives can cause anaphylactic reactions of which asthma may be a part. Specific drugs that affect the arachidonic acid pathways have already been discussed.

a) Skin tests

To demonstrate specific IgE antibody, skin prick tests with a range of common inhaled allergens are the most reliable method of testing. A small drop of the allergen solution is put in the skin and the superficial layers of the skin are gently lifted with the tip of a sharp needle through the drop of solution. A positive reaction consists of a weal and a flare, (the former being more significant), that erupts within 15-20 minutes and positive results correlate extremely well with specific IgE in the blood. In Singapore, this test is also performed and details are available elsewhere(10).

Total serum IgE antibody level measured by RIST and specific IgE antibody using RAST are expensive to do and the former shows considerable overlap between the normal and atopic populations for it to be clinically useful.

Late skin reactions consisting of a large, well-defined swelling appearing 4 hours after the immediate skin reaction, reaching a maximum at 5 to 8 hours, and persisting up to 36 hours show good correlation with type III allergy (though not always confined to it) and late bronchial reactions.

b) Evidence of mediator release

Release of ECF-A from mast cells will cause eosinophilia in local tissue and in the blood. More than $0.4 \times 10^9/L$ in the blood is generally taken as abnormal. The presence of eosinophils in the sputum is always abnormal. As ECF-A is released whether the stimulus is immunological or not, eosinophils do not necessarily indicate an allergic reaction.

c) Specific bronchial challenge testing

Multiple positive skin test allergies signifying an atopic status do not necessarily correlate them as the precipitating cause of bronchospasm. Positive skin tests in the absence of a supportive history are probably of little clinical relevance. Should there be single or dual skin test positive reactions and supportive clinical history, there may arise the need to confirm the relevance of that allergen in causing the patient's asthma. In some cases of industrial asthma, it is necessary to challenge with the possible offending substance to establish a diagnosis. In these two instances, then, specific bronchial challenge testing is indicated and follows the principles of testing as for histamine inhalation.

On the basis of the above investigations, some of the cases of bronchial asthma would be labelled intrinsic or idiopathic. This may be because the extrinsic causes have not been defined as yet.

Many urban asthmatics who would have formerly been classified as "intrinsic" are now known to be allergic to cockroach antigens (11-13) or to rodent antigens(14). As the antigens used for skin testing are manufactured overseas, e.g. UK, it is possible that skin-test-negative asthmatics may have extrinsic asthma but to as yet unidentified aeroallergens in our local environment.

C. Patterns of asthma

1. Breathing patterns

Asthma causes severe airway obstruction and reduced dynamic compliance, thereby increasing the work of breathing. Furthermore, the resulting, rapid, shallow breathing is not optimal for minimal work(15). Respiratory frequency is increased during the asthmatic attack (16,17), the mechanisms of which may be vagally mediated.

There are many factors that increase the work of breathing in asthma in addition to the increase in airway resistance and the fall in dynamic lung compliance. These include hyperventilation (the partial pressure of arterial CO_2 is frequently reduced during an asthmatic attack); the fact that tidal volume and frequency are not optimal for minimal work at a given alveolar ventilation; the sustained activity of the inspiratory muscles responsible for the increase in functional residual capacity(18,19), which markedly increases the elastic work of breathing; and the distortion of the rib cage and abdomen from their configuration during relaxation(20). During breathing the respiratory muscles must perform work against viscous resistance (of airways and tissues) and elastic resistance. The amount of work done against viscous resistance is determined to a large extent by turbulent airflow that results from branching in the upper airways and bronchioles. Increased flow rates and airway obstruction cause increased turbulent airflow and hence increased work but in bronchioles, flow is laminar rather than turbulent(21) and in some asthmatics, most of the obstruction is in peripheral airways and turbulence contributes very little to airway resistance and the work of breathing(22,23).

The sum total of prolonged asthmatic attacks is the increasing probability of muscle fatigue and exhaustion. Preoccupation with "status asthmaticus" is outdated. Long before there is complete un-responsiveness to outpatient pharmacological treatment, a major increase in treatment, use of adrenocorticosteroids and referral to hospital is mandatory. These patients are hyperventilating and are agitated because they are sick, hypoxic and usually have an excess of endogenous and exogenous catecholamines circulating. They do not have psychogenic hyperventilation syndromes. They require oxygen and bronchodilation, not sedation. Asthma patients are hypocapnic and oxygen treatment for acute attacks should generally be liberal. Hypercapnoea usually implies exhaustion or extreme airways obstruction. One should be wary of acute asthmatic patients without a low P_aCO_2. They may be in a precarious position and require respiratory support.

2. Airflow obstruction patterns

It is conventional to divide asthma into the extrinsic and intrinsic types as a means of classification. This step is useful, if simply to remind us that atopy or allergy is not an essential requirement. Of more practical importance in aiding management is Turner-Warwick's suggestion that we simply go back to calling any variable (reversible) airways obstruction as "asthma" and try to distinguish empirically types of asthma depending on the pattern of variability and response to treatment. Four patterns are recognised based on peak flow monitoring(24).

a) The BRITTLE asthmatic complains of intractable asthma and of extreme, rapid variability of the wheezing. Records show that low air flow readings are rapidly reversed by bronchodilator aerosols, but attempts at stabilisation commonly fail. Recognition of this pattern is useful because the clinician learns to understand why such patients need to use their aerosols — for the asthma, and not because of "addiction".

b) The MORNING DIPPER patients maintain a rhythm of obstruction with well-maintained peak flows during the day and reduction to low levels in the early hours of the morning. Recognition of this pattern is useful because it helps us realise that repeated normal readings during office hours can occur in severe asthma, death from asthma at night is a real risk and slow-release sympathomimetic and theophylline tablets taken on retiring are likely to be required.

c) The "IRREVERSIBLE" asthmatic exhibits at least a component of airways obstruction that apparently cannot be reversed with conventional bronchodilators. Some of these patients have asthma as the cause of at least part of this apparently fixed obstruction by reversing with cor-

ticosteroids, or even with atropine alone. Recognition of this group prevents condemning patients to the diagnosis of "irreversible airways obstruction." Even patients with apparently classical emphysema can be hiding an useful element of apparently "irreversible asthma."

d) The DRIFTER shows little day to day in their obstruction but can very slowly improve with therapy as judged by careful peak flow monitoring over months.

However patients can switch from one pattern to the other and show all sorts of other patterns.

D. Control of asthma

Control of asthma can be defined as (1) normal functioning of the individual (2) no severe attacks (3) no persisting symptoms and (4) no side effects from medications.

The key to restoration of control is early recognition of worsening with the introduction of a sufficient increase in treatment to reverse the problem. A major increase in the level of treatment is needed if function is limited or there is awakening two nights in a row. Patients must be informed that an ingested adrenocorticosteroid is usually needed without delay if there is ever a need for an inhaled beta-agonist more than six times daily of if there is any shortness of breath which persists for one hour despite other treatment.

Patients need reassurance by a number of messages including: asthma is not due to emotions; does not lead to emphysema; does not lead to heart disease; usually has nothing to do with diet; is variable and even after years of persisting difficulty can subside; is reversible; and can almost invariably be controlled by present methods and present medications with virtually no side-effects. The repeated necessity for urgent treatment or repeated hospital admissions indicates inadequate maintenance treatment and/or inadequate intervention in response to early warnings of exacerbations.

Avoidance strategies have major benefits in a few circumstances; these include avoidance of exposure in occupational asthma, removal of a pet from the house when it is responsible and air conditioning if asthma is from pollen and mould spores. Household dust control measures may help but should not be overdone.

A British confidential inquiry into deaths from asthma found that 86% of the 90 deaths were potentially preventable(25) and sudden severe attacks were known to have occurred in 35 of them. The committee commented that "there was little evidence that the patients or their relatives received adequate instructions in the use of corticosteroid drugs, particularly for exacerbations of asthma, or on what action to take to get medical help in a severe attack." Discussing the routine drug treatment they suggest that some patients should have been taking higher doses of bronchodilators and of those on steroids a proportion should have been on higher doses. In an analysis of the fatal attack, in 71 of the 90 patients, the final attack lasted less than 14 hours and the general practitioner failed to send to hospital 23 patients. It was suggested that earlier hospital admission would have saved many lives.

The importance of objective measurements in the assessment of ventilatory capacity and the degree of airway obstruction is illustrated by a poor correlation of spirometry with clinical signs and symptoms(26, 27). In most instances the physician and patient underestimate the severity of the abnormality, which contributes to asthma mortality and morbidity and a plea is made for more widespread utilisation of the simple, portable peak flow meter. Even should improvement occur in peak expiratory flow rates, the arterial blood gas tensions fail to improve for several days during recovery from acute attacks of asthma(28). The mechanisms leading to the fall in P_aO_2 is only partially related to peak expiratory flow rate. It is associated with plugging of small airways, leading to a shunt effect(26,28). Persistence of small airway dysfunction with associated hypoxia but few symptoms, is well documented in asthma (29). This implies that we should not be in too great a hurry to cease treatment after attacks of acute asthma, since subtle but important abnormalities may persist even during interval phases of asthma which may not be evident on clinical examination. This small airways dysfunction has two implications. Firstly asymptomatic interval phase asthma is not necessarily equated to absence of airways pathology. Secondly, interval asthmatics may reduce their level of activities to cope with their diminished respiratory reserve and this may be subtle and unappreciated until appropriate treatment is instituted.

The residual abnormalities present due to undertreatment of asthma must be stressed. They may well from the basis for premature deterioration of lung function.

E. Medications for asthma

The first line of treatment is with drugs that bronchodilate and reduce bronchial responsive-

ness; these include theophylline and/or a beta-agonist in the United States while the reverse, a selective beta-2-agonist and/or theophylline holds true for the British school. A most recent review has been published(30). In disease, bronchodilator drugs can act in two ways: by relaxing both the circumferential and the longitudinal smooth muscle fibres of the airways through direct action and by shrinking the mucosa and diminishing secretory activity through vaso-motor effects. The three principal categories of bronchodilators are the sympathomimetic agents (usually beta-2-agonists), the xanthines and the anticholinergic drugs.

Locally, a selective beta-2-agonist will usually be the first choice bronchodilator for both the rapid relief of an asthmatic attack and for the maintenance treatment of chronic asthma. Though best given by pressurised aerosol inhaler to minimise side effects of muscle tremor, tachycardia and anxiety, because inhalation technique is important and somehow local patients find difficulty in coordinating the puffs, tablets are still widely prescribed. Alternative methods of inhalant therapy using a dry powder inhaler (e.g. Ventolin Rotacaps) or a tube spacer (e.g. Bricanyl) have not caught on either.

Theophylline has a low toxi/therapeutic ratio and its plasma half-life is reduced in smokers but prolonged by concurrent treatment with cimetidine, and in patients with cirrhosis, congestive heart failure, chronic obstructive lung disease, cor pulmonale and in acute febrile episodes. Toxicity may manifest early as nausea and vomiting. Theophylline plasma concentrations have yet to be measured locally as a routine test but levels of 6-10 ug/ml in the average adult gives few side effects (nausea, vomiting, headaches, insomnia and agitation). However concentrations of 10-20 ug/ml give maximal therapeutic bronchodilatation but side effects are also more prevalent.

It is often stated that xanthines act by inhibiting phosphodiesterase, which subsequently increases the levels of CAMP in bronchial muscle and thereby relax them. This would imply that the combination of a beta-2-agonist with theophylline would be synergistic in producing bronchodilatation. Phosphodiesterase inhibition moreover requires theophylline concentrations some ten times higher than those causing bronchodilatation in man in vivo(31) and most studies in man fail to show this expected synergism(32,33). Nonetheless, oral theophylline does act as a bronchodilator to spare high dosages of beta-2-agonists (especially when given by mouth) and diminish undesirable muscle tremor. Theophylline's other beneficial actions include potentiating diuretics, increasing cardiac output and possibly pulmonary vasodilatation. Recently, it has been shown that it may also improve the mechanical performance of the diaphragm and render it less susceptible to fatigue(34).

Slow-release preparations of both beta-2-agonists (eg Ventolin spandets, Meptin) and theophyllines (eg Nuelin, Euphyllin retard) are available and are preferred as they provide relatively constant plasma concentrations of the drug over at least 4-8 hours and this may be important in preventing the early "morning dip" in some chronic asthmatics.

Oral steroids (prednisolone) are still the mainstay of treatment for severe asthmatics in addition to the above drugs. Inhaled steroids (Becotide or Rotacaps) provide effective prevention and can replace oral steroids but regular administration is essential. After sustained use of bronchodilators, decreased responsiveness may appear and is often associated with severe respiratory acidosis. In certain patients a refractory form of asthma, the so-called locked lung syndrome, has been described. This syndrome may be due to a blockade of beta-adrenergic receptors by a metabolite of isoprenaline(35,36). However the blocking activity of this substance may also be weak so that the locked lung syndrome or other forms of refractory asthma may be due to tolerance to adrenergic aerosols(37).

Tolerance, desensitisation, tachyphylaxis or refractoriness is the phenomenon where continued exposure to a drug or hormonal agonist often leads to a blunted response to that agonist. Desensitisation is often accompanied by a decrease in the affinity of the receptors for the substance (uncoupling) followed by a decrease in the number of receptors (down-regulation). Down-regulation of beta-adrenergic receptors on white cells has been convincingly demonstrated(38-41) but methylprednisolone treatment can rapidly reverse terbutaline-induced down-regulation of lymphocyte beta-adrenergic receptors(39). Also, patients with asthma who are receiving long-term steroid therapy have a normal number of white blood cell beta-adrenergic receptors(42). Glucocorticoids have been found to cause an acute increase in beta-adrenergic receptors within 4 hours of administration(43).

Sodium cromoglycate by powder inhalation or aerosol when used regularly is effective in preventing asthma when the situation is stable. Long-term use actually reduces the airway hyperreactivity and significantly reduces the frequency and severity of asthma attacks(44,45). Bronchodilators only relieve the symptoms. Beta-agonists by inhaler and cromoglycate are valuable in pre-

venting exercise-induced asthma if taken before hand. However, Hambleton et al have shown that the protective effect of theophylline in allergic asthma is significantly stronger than that of cromoglycate(46). The powder in spincaps tends to clump in the tropics and again, the inhalation manoevre is a set back to its use locally.

Finally to mention the newer drugs and immunotherapy. Ipratropium bromide, an atropine derivative, is a powerful anticholinergic inhibitor of vagally-mediated bronchomotor tone. Given by aerosol (it is not available locally). There are no systemic atropine-like effects and no inhibition of mucociliary clearance(47). It has been used in treating acute asthma(48) and produces further bronchodilatation by interaction with beta-2-agonists or theophylline.

Ketotifen is a new oral agent, for preventing asthma, with anti-histamine and antiphylactic actions but it takes several weeks of treatment to show effect. It is also reported to posses phospodiesterase-inhibiting properties and functional SRS-A antagonism(49). Given twice daily, drowsiness is the main side effect. Details of its efficacy have been summarised recently(30).

Calcium ions are involved in the constriction of smooth muscle and mediator release. Inhibition of transmembrane calcium ion influx should be beneficial in asthma which involves both contraction of bronchial smooth muscle and secretion of bronchoconstriction-mediators from mast cells(50). Nifedipine is a potent antagonist of calcium ion influx across cell membranes and it appears to inhibit exercise-induced asthma(51,52) and exhibit a protective effect against histamine-induced bronchoconstriction in stable asthmatic subjects(53).

Allergen injection treatment (hyposensitisation, immunotherapy) reduces responsiveness to the antigens injected. Efficacy is well proven in pollen hay fever but less well in asthma. In recent years, treatment with medication has outdistanced immunologic strategies. Controlled trials have shown that hyposensitisation to house dust and house dust mite result in clinical improvement in carefully selected asthmatic children(54) but failed for grass-pollen asthmatics(55).

F. Summary

It is important to make the correct diagnosis of asthma especially in those who may already have other lung disease but with a concomitant asthmatic element. Precipitating and triggering factors should be avoided where possible and practical but modern-day therapeutic modalities are highly effective and acceptable to patients. Knowing the patterns of asthma enables proper timing of medication to be prescribed, and decreases the probability of undertreating patients who appear clinically well when seen in the day. Beta-2-agonists are freely utilised because the effect is rapid and the patient relieved of symptoms. Aerosols should be the preferred route. There should be no hesistancy to administer parenteral and/or oral corticosteroids for short periods when the patient's asthma is out of control, resorting to inhaled preparations only when the asthma has stabilished. Sodium cromoglycate has several strong points to recommend its more widespread use if oral preparations were effective. Of the newer drugs, more studies are required to prove their usefulness.

REFERENCES

1. Slavin RG, Cannon RE, Friedman NH, Palitang E, Sundaram M. Sinusitis and bronchial asthma. J. Allergy Clin Immunol 1980;66:250-7.
2. Szentivanyi A. The beta-adrenergic theory of the atopic abnormality in bronchial asthma. J Allergy 1968;42:203-32.
3. American Thoracic Society Committee on Diagnostic Standards for Nontuberculous Disease. Definitions and classification of chronic bronchitis, asthma and pulmonary emphysema. Am REV Resp Dis 1962;85:762-9.
4. Cockcroft DW, Killian DN, Mellon JJA, Hargreave FE. Bronchial reactivity to inhaled histamine: a clinical survey. Clin Allergy 1977;7:235-43.
5. Corrao WN, Braman SS, Irwin RS. Chronic cough as the sole presenting manifestation of bronchial asthma. N Engl J Med 1979;300:633-7.
6. Woolcock A. Airway reactivity. Mechanism and Clinical relevance. In Hargreave FE. ed. Proceedings of symposium, McMaster University 1979. Mississauga, Ontario, Astra 1980.
7. Juniper EF, Frith PA, Hargreave FE. Airway responsiveness to histamine and methacholine; relationship to minimum treatment to control symptoms of asthma. Thorax 1981;36:575-9.
8. Shim CS, Williams MH Jr, Evaluation of the severity of asthma: patients versus physicians. Am J med 1980;68:11-3.
9. Chee YC. Measurement and clinical significance of bronchial responsiveness to histamine. Sing Med J 1982;23:306-10.
10. Chee YC. Skin prick tests for atopy in asthmatic adults in Singapore. ASEAN J Clin Sci 1982;3:259-64.
11. Kang B. Study on cockroach antigen as a probable causative agent in bronchial asthma. J Allergy Clin Immunol 1976;58:357-65.
12. Twarog FJ, Picone FJ, Strunk RS, So J, Colten HR. Immediate hypersensitivity to cockroach; isolation and purification of the major antigens. J Allergy Clin Immunol 1977;59:154-60.

13. Kang B, Sulit N. A comparative study of prevalence of skin hypersensitivity to cockroach and house dust antigens. Ann Allergy 1978; 41:333-6.
14. Lim DT, Ganju A. Reaginic sensitivity to mus musculus extract in a group of Chicago urban asthmatics. Ann Allergy 1980; 44:267-72.
15. Roussos C, Macklem PT. The respiratory muscles. N Engl J Med 1982; 307:786-97.
16. McIlroy MB, Marshall R. The mechanical properties of the lungs in asthma. Clin Sci 1956; 15:345-51.
17. Hedstrand U. The optimal frequency of breathing in bronchial asthma. Scand J Respir Dis 1971; 52:217-21.
18. Martin J, Powell E, Shore S, Emrich J, Engel LA. The role of respiratory muscles in the hyperinflation of bronchial asthma. Am Rev Resp Dis 1980; 121:441-7.
19. Muller N, Bryan AC, Zamel N. Tonic inspiratory muscle activity as a cause of hyperinflation in histamine-induced asthma. J Appl Physiol 1980; 49:869-74.
20. Goldman MD, Grimby G, Mead J. Mechanical work of breathing derived from rib cage and abdominal V-P partitioning. J Appl Physiol 1976; 41:752-63.
21. Wood LDH, Engel LA, Griffin P, Despas P, Macklem PT. Effect of gas physical properties and flow on lower pulmonary resistance. J Appl Physiol 1976; 41: 234-44.
22. Despas PJ, Leroux M, Macklem PT. Site of airway obstruction in asthma as determined by measuring maximal expiratory flow breathing air and a helium-oxygen mixture. J Clin Invest 1972:3235-43.
23. Lisboa C, Jardimi J, Angus E, Macklem PT. Is extrathoracic airway obstruction important in asthma? Am Rev Resp Dis 1980; 122:115-21.
24. Turner-Warwick M. On observing patterns of airflow obstruction in chronic asthma. Br J Dis Chest 1977; 71:73-85.
25. British Thoracic Association. Deaths from asthma in two regions of England. Br Med J 1982; 285: 1251-6.
26. McFadden EF, Kiser R, deGroot WJ. Acute bronchial asthma. New Engl J Med 1973; 288:221-5.
27. Rubinfeld AR, Pain MC. How mild is mild asthma? Thorax 1977; 32: 177-81.
28. Rebuck AS, Read J. Assessment and management of severe asthma. Am J Med 1971; 51:788-98.
29. Cochrane GM, Benatar SR, Davis J, Collins JV, Clark TJH. Correlation between tests of small airway function. Thorax 1974; 29: 172-8.
30. Flenley DC. New drugs in respiratory disorders I. Br Med J 1983; 286: 871-5.
31. Kolbeck RC, Speir WA, Carrier GO, Bransome ED. Apparent irrelevance of cyclic nucleotides in the relaxation of tracheal smooth muscle induced by theophylline. Lung 1979; 156:173-83.
32. Vermeire P. Combined use of theophylline with beta-2 agonists in asthma. Eur J Resp Dis 1982; 63: 372-5.
33. Isles AF, McLeod SM, Levison H. Theophylline. New thoughts about the old drug. Chest 1982; 82:49S-54S.
34. Ambier M, De Troyer A, Sampson M, Macklem PT, Roussos C. Aminophylline improves disphragmatic contractility. N Engl J Med 1981; 305:249-52.
35. Van Metre TE. Death in asthmatics. Trans Am Clin Climatol Assoc 1966; 78:58-69.
36. Bierman WC, Pierson WE. Hand nebulisers and asthma therapy in children and adolescents. Pediatrics 1973; 54: 668-70.
37. Dollery CT. Absorption and fate of isoproterenol and fluorocarbons inhaled from pressurised aerosols. Ann Allergy 1973; 31: 25-30.
38. Galant SP, Duriseti L, Underwood S, Insel PA. Decreased beta-adrenergic receptors on polymorphonuclear leukocytes after adrenergic therapy. N Engl J Med 1978; 299:933-6.
39. Tashkin DP, Conolly ME, Deutsch RI et al. Subsensitisation of beta-adrenergic receptors in airways and lymphocytes of healthy and asthmatic subjects. Am Rev Resp Dis 1982; 125: 185-93.
40. Krall JF, Connelly M, Tuck ML. Acute regulation of beta-adrenergic catecholamine sensitivity in human lymphocytes. J Pharmacol Exp Ther 1980; 214:554-60.
41. Tohmeh JF, Cryer PE. Biphasic adrenergic modulation of beta-adrenergic receptors in man: agonist-induced early increment and late decrement in beta-adrenergic receptor number. J Clin Invest 1980; 65:836-40.
42. Galant SP, Duriseti L, Underwoods, Allred S, Insel PA. Beta-adrenergic receptors of polymorphonuclear particulates in bronchial asthma. J Clin Invest 1980; 65:577-85.
43. Davies AO, Lefkowitz RJ. Corticosteroid-induced differential regulation of beta-adrenergic receptors in circulating human polymorphonuclear leukocytes and mononuclear leukocytes. J Clin Endocrinol Metab 1980; 51:599-605;
44. Altounyan REC. Review of the clinical activity and modes of action of sodium cromoglycate, in Pepys J, Edwards AM eds: The mast cell: its role in health and disease. Kent, England. Pitman Medical 1979, pp 199-216.
45. Hyde JS, Isenberg PD, Floro LD. Short-and-long-term prophylaxis with cromolyn sodium in chronic asthma. Chest 1973; 63:875-80.
46. Hambleton G, Weinberger M, Taylor J et al. Comparison of cromoglycate and theophylline in controlling symptoms of chronic asthma. Lancet 1977; 1:381-5.
47. Rebuck AS, Chapman KR, Braude AC. Anticholinergic therapy of asthma. Chest 1982; 82: 55S-7S.
48. Ward MJ, Fenthem PN, Roderick-Smith WH, Davies D. Ipratropium bromide in acute asthma. Br Med J 1981; 282:598-600.
49. MaClay WP, Kennedy GR. Drugs for asthma: Mast-cell stabilisers. Br Med J. 1981; 282: 1072-3.
50. Middleton E. Antiasthmatic drug therapy and calcium ions; review of pathogenesis and the role of calcium. J Pharm Sci 1980; 69:243-51.
51. Cerrina J, Denjean A, Alexandre G, Lockhart A, Duroux P. Inhibition of exercise-induced asthma by a calcium antagonist, nifedipine. Am Rev Resp Dis 1981; 123:156-60.
52. Barnes PJ, Wilson NM, Brown MJ. A calcium antagonist, nifedipine, modifies exercise-induced asthma. Thorax 1981; 36:726-30.
53. Williams DO, Barnes PJ, Vickers HP, Rudolf M. Effect of nifedipine on bronchomotor tone and histamine reactivity in asthma. Br Med J 1981; 283: 348-9.
54. Warner JO, Price JF, Soothill JF, Hey EN. Controlled trial of hyposensitisation to Dermatophagoides

pteronyssinus in children with asthma. Lancet 1978; 2; 912-5.
55. Hill DJ, Hosking CS, Shelton MJ, Turner MW. Failure of hyposensitisation in treatment of children with grass-pollen asthma. Br Med J 1982; 284: 306-9.

General Reading

1. Asthma Series No 1 to 6 by Sandoz publications
2. Antiasthmatic drugs today: A seminar
Medical Progress April 1982
Antiasthmatic therapy pg 76
Sympathomimetric and anticholinergic bronchodilators pg 79
Theophylline pg 85
Sodium cromoglycate pg 90
Corticosteroids pg 96
(has good MediGuide for each section, useful for patient instructions)

Home Study Programme MCQs

MCQs on articles in Vol. IX No. 2 (April/June 1983). Answers on page 158

Starting with this issue of The Singapore Family Physician, answers to MCQs will be published in the same issue as the MCQs, but no unfair peeping. If your score is not fully satisfactory, how about reading the articles again?

1. Which of the following statements about pneumonia are true?
   - A In pneumonia, fever may be absent in elderly patients and patients on steroids
   - B A hospital patient developing pneumonia is likely to be infected by staphylococcus aureus
   - C Anaerobic infections are commonly seen in alcoholics and diabetics
   - D Haemophilus influenzae is a cause of serious pneumonia in children below the age of 5 years
   - E Small abscesses and pneumatocoeles in the chest radiograph indicate staphylococcal infection

2. Pneumonia is more likely to be due to a non-bacterial infection if:
   - A Respiratory symptoms precede and overshadow constitutional symptoms
   - B Physical signs in the chest are absent or inconspicuous
   - C Mucoid sputum is produced
   - D The spleen is palpable and the white cell count is normal
   - E A pleural effusion develops

3. Pneumococcal pneumonia is characterised by:
   - A A sudden onset of chills followed by rigors and high fever
   - B A high incidence of associated herpes labialis
   - C Peak frequencies in infants and the aged
   - D Early onset of pleuritic pain
   - E Homogenous consolidation of one or more broncho-pulmonary segments

4. Which of the following statements about aspiration pneumonia are true?
   - A Anaerobic infections are common
   - B The dependent parts of the lungs are involved
   - C Oesophageal disease is a predisposing factor
   - D It is a recognised complication of chronic sinusitis
   - E As a complication of coryza, it typically causes bilateral symmetrical radiographic opacities

5. The following statements are correct:
   - A Staphylococcal pneumonia is a known complication of influenza
   - B Chicken-pox pneumonia is commoner in children than in adults
   - C Patients with mycoplasma pneumoniae infections may develop haemolysis and other extra-pulmonary manifestations
   - D Severe pneumonia with red, tenacious sputum is caused by Klebsiella pneumoniae
   - E In Legionnaire's Disease, the clue to diagnosis is a serious pneumonia with multi-system involvement

6. In patients with pneumonia:
   - A Skin test is useful when fungal infection (e.g. aspergillosis) is suspected
   - B The Chest X-ray will show involvement of the upper lobes with massive consolidation, bulging of the fissures and cavitation and even pleural effusion in Klebsiella pneumoniae infection
   - C The mortality rate is 50-80% if the infection is caused by gram-positive organisms like Klebsiella or Pseudomonas
   - D Diagnosis of Legionnaires' Disease is confirmed by Immunofluorescent Antibody studies
   - E Jaundice is a not uncommon complication of severe pneumonia

7. Lung abscess may result from:
   - A Bacterial infection of a collapsed lobe
   - B Infection of a pulmonary infarct
   - C Obstruction of a bronchus by carcinoma
   - D Amoebic infection in the liver
   - E Trauma to the lung

8. In pulmonary abscess:
   A A high swinging fever is typical
   B Putrid sputum is due to anaerobic infection
   C Aspiration is suggested as the cause when the pulmonary opacity in the Chest X-ray is situated in the apical segment of the right lower lobe or the posterior segment of the right upper lobe
   D Cavitated lung tumour is an important differential diagnosis
   E Most patients will require surgical treatment

9. Which of the following statements about bronchiectasis are true?
   A It is seen most frequently in the middle lobe
   B It is a recognised complication of measles, varicella, pertussis and tuberculosis
   C It typically causes a cough, worse in the night, productive of copious foul-smelling, yellow sputum
   D It is a recognised complication of cystic fibrosis
   E Hypogammaglobulinaemia is a predisposing factor

10. The clinical features of bronchiectasis include:
   A Night sweats
   B Haemoptysis
   C Recurrent episodes of pneumonia or pleurisy
   D Clubbing of the fingers
   E Crepitations audible over the affected areas of the lung

11. Which of the following findings may be helpful in elucidating the aetiological basis of bronchiectasis in a patient?
   A A history of long-standing diarrhoea with pale stools
   B A history of a severe attack of whooping cough
   C Dextrocardia and sinusitis
   D Calcified broncho-pulmonary lymph nodes seen on Chest X-ray
   E A positive precipitin reaction to Aspergillus niger

12. In bronchiectasis:
   A Dyspnoea and wheezing may be present
   B Postural drainage and long-term antibiotic therapy is indicated
   C Cerebral abscess and amyloidosis are possible complications
   D Failure of medical treatment is a clear indication for surgery
   E Surgical treatment must be preceded by bronchography

13. Which of the following pairs of occupations and diseases caused are correct?
   A Pottery and ceramics making – silicosis
   B Coal mining – anthracosis
   C Tin mining – siderosis
   D Cane-sugar industry – byssinosis
   E Cork industry – suberosis

14. Which of these statements about silicosis are correct?
   A Silicosis results from prolonged inhalation of coal dust
   B Symptoms appear as soon as the fibrogenic response of the lung commences
   C Finger clubbing is a common sign
   D Tuberculosis is a common complication
   E "Egg-shell" calcification in the hilar lymph-nodes is a characteristic feature

15. In silicosis:
   A A purely restrictive disease is produced
   B Haemoptysis may occur
   C The pleurae are characteristically thickened and adherent
   D There is an increased incidence of lung cancer
   E The rheumatoid factor may be present without any arthritis

16. Which of the following clinical signs are common in asbestosis?
   A Clubbing of the fingers
   B Fine crepitations
   C Cyanosis
   D Painful corns of warts on the hands
   E Emphysema

17. Which of the following statements about asbestosis are true?
   A Presence of "asbestos bodies" in the sputum is diagnostic of the disease
   B On X-ray, the abnormalities are most marked in the upper zones
   C Bilateral pleural plaques are often present
   D Bronchial carcinoma is a complication
   E Peritoneal mesothelioma may follow trivial exposure to blue asbestos

18. Coal-workers' pneumoconiosis:
   A Is more common in mines producing hard

coal than soft coal
B May be complicated by progressive massive fibrosis
C Is associated with an increased incidence of lung cancer
D Is often associated with silicosis
E In association with rheumatoid arthritis is known as Caplan's Syndrome

19. In Farmers' Lung:
A There is a seasonal incidence during summer
B Crepitations would be an expected finding
C The patient suffers from attacks of acute dyspnoea
D Cough with profuse expectoration if common
E Examination of the peripheral blood would reveal a neutrophilic leucocytosis

20. Pulmonary cedema seen on Chest X-ray may be caused by:
A Heroin over-dosage
B Sarcoidosis
C Fat emboli
D Haemosiderosis
E Eosinophilic Lung Syndrome

21. Miliary calcification of the lungs on X-ray may occur in:
A Chicken-pox
B Asbestosis
C Histoplasmosis
D Sarcoidosis
E Farmers' Lung

22. Causes of acute ventilatory failure include:
A Encephalitis
B Poliomyelitis
C Guillain-Barre Syndrome
D Myasthenia gravis
E Opiate and barbiturate overdose

23. In acute ventilatory failure:
A The serum bicarbonate is normal or slightly raised
B The patient may exhibit paradoxic inward motion of the abdomen during inspiration
C The arterial $PcCo_2$ is greater than 80 mm. Hg.
D Diazepam is the drug of choice if sedation is required
E Mechanical ventilation can be discontinued as soon as the disturbances in blood pH and gases have been corrected

24. Spontaneous pneumothorax:
A Typically results from rupture of a subpleural bulla
B May not produce any abnormal physical signs
C With a fluid level at the costo-phrenic angle on Chest X-ray indicates a haemothorax
D May recur on the same side in 30% of patients
E May not require any active treatment

25. Predisposing factors for pulmonary embolism include:
A Oral contraceptive use
B Congestive cardiac failure
C Lung abscess
D Chronically ill, elderly patients
E Chronic bronchitis

26. Which of the following statements are correct?
A Pulmonary infarction occurs in 50% of patients with pulmonary embolism
B Gross haemoptysis occurs in all cases of pulmonary embolism
C A bloody pleural effusion is seen only when pulmonary infarction accompanies pulmonary embolism
D The treatment of pulmonary effusion consists of anticoagulants and bronchodilators
E Pulmonary infarction requires surgical removal of the pulmonary embolus

27. The Adult Respiratory Distress Syndrome:
A Occurs in persons with previously normal lungs
B Chest X-ray shows the picture of pulmonary oedema
C Non-thoracic trauma may be the initiating event
D May be a complication of acute pancreatitis
E Positive end expiratory pressure (PEEP) ventilation with 100% oxygen is usually necessary in the treatment

Book Review

FAMILY PRACTICE — An International Perspective in Developed Countries

Editors: John P Geyman, John Fry

Appleton-Century-Crofts/Norwalk (1983) Connecticut

The editors Geyman and Fry, together with 7 contributors have put together the results of an in-depth research and evaluation of the health care systems of the United States, Canada, the United Kingdom, Scandinavia, Australia, New Zealand, Japan and South Africa, representing a broad spectrum of traditions of medical education and patterns of health care delivery. They are to be congratulated for the work they have done.

The past 10-15 years have witnessed a rediscovery in many developed countries of the essential role of the family physician. In spite of the considerable differences in the medical education and health care systems from one country to another parallel developments have taken place in the renaissance of general/family practice and its emergence as a distinct discipline. Much can be learned by describing and comparing the experience in countries that are leaders in this renaissance.

Specifically, this book has 3 objectives:

(1) It seeks to bring together in one book the trends in family practice in various developed countries;

(2) It seeks to present an analysis of the evolution and present state of family practice in selected countries to illustrate common problems and issues; and

(3) It seeks to consider future needs for family practice in developed countries.

This book has ten chapters. Chapter 1 describes the background of Family Practice past and present. Chapters 2 to 9 are devoted to in-depth analysis of health care system organisation, training and research in each of the eight countries selected for this comparative study. The last chapter wraps up the whole study with the authors making comparisons and contrasts of the various aspects of each country's experience during the last 10-15 years. The remaining paragraphs give a bird's eye view of what they found.

The evolution of family practice:

Family Practice is being regarded by some as a specialty. It is interesting to note that those countries where family practice has made the most progress as a recognised "specialty" are those which went through a cycle of increasing medical specialization, only to discover that there is the need to rebuild the primary care base. Perhaps the specialist attitude towards medical care is now being transferred to Family Medicine.

The role of family practice organisations in the renaissance of family practice presents some contrasts. In Canada, a single organisation, the College of Family Physicians of Canada, has assumed all of the following responsibilities: approval and accreditation of family medicine residency programs, sponsorship and conduct of continuing education programs, and both certification and recertification by examination of candidates for specialty certification. Several years ago, it formed a section for teachers of family medicine. In the United States, on the other hand, a group of organisations is responsible for these functions: the American Board of Family Practice (certification and recertification), the Society of Teachers of Family Medicine (teaching and academic development), and the American Medical Association (accreditation of residency programs).

The current status of Family Practice:

Those countries with high proportions of generalists (e.g., United Kingdom, Canada, Australia) have relatively clear-cut divisions of responsibility among specialties for primary care, exemplified most clearly by the firmly established gatekeeper role in the United Kingdom. Countries with comparatively low proportions of generalists (e.g., United States, Japan) have diluted and fragmented primary care responsibilities among many specialty groups and settings. This is illustrated by the so-called hidden system of primary

care (actually a nonsystem) in the United States, where non-primary care specialties (e.g., surgery) may provide limited primary care services for which they are often poorly prepared and little interested.

Patterns of Practice:

Although solo practice was the predominant form of practice in the countries studied, an increasing number are now practising in groups, usually small groups of two to four family physicians.

In the United States, Canada, Australia, and South Africa substantial hospital care is part of one's everyday practice. For the rest there is a striking lack of any significant role in hospital practice.

The structure of primary care system:

There is a considerable variation in the structure of the primary care system from one country to another, ranging from the full gatekeeper role in the United Kingdom (whereby the patient cannot obtain care by a consultant without referral from the family physician) to the largely unstructured system in Japan, where a hospital system of inpatient-outpatient care is in active competition with a myriad of private practice clinic-based physicians in all specialties.

The reimbursement system:

The most common predominant reimbursement system used is some type of fee-for-service arrangement, but there are important variations from one country to another in how reimbursement levels are determined and applied. A full capitation method of reimbursement, based upon provision of primary care services to a defined population as a gatekeeper to the health care system, is found today only in the United Kingdom and Denmark.

Education:

The extent of development of family practice in the medical schools appear to be strongly correlated with the progress made in each country. Thus, in the US and Canada, a typical department of family practice has focused around a departmental teaching practice located either at the University or in affiliated sites in the community; active teaching programs are conducted for both medical students and residents. In Australia, all of the medical schools have departments of family practice; all of these are involved in medical student teaching, but none are involved in residency training. In South Africa, the departments of family practice in the medical schools have extensive service commitments, including responsibility for the emergency room, general outpatient clinics, and community clinics; teaching in these departments is limited to medical students, since residency training is not provided on an organized basis.

Residency training has been developed as a major part of family practice in those countries where it is best established. Several countries are pressing for completion of formal family practice residency training before the graduate can be certified as a family physician, even for practice. To date, only the United Kingdom has called for mandatory residency training in preparation for general/family practice. Although there is a considerable variation in the structure of residency training around the world, most programs average three years in length. They usually comprise a combination of hospital-based teaching rotations (usually 18 to 24 months) (e.g., medicine, paediatrics, obstetrics-gynacology, and surgery) and supervised practice experience in a teaching general/family practice. In the United States there is a strong emphasis on continuity of care during the residents training, so that the resident's time is divided between hospital/ambulatory teaching rotations and the family practice centre throughout the three year programme. In most other countries, there is a sharp separation between block rotations in the other specialties and general/family practice experience. There are no such studies yet available that compare the educational outcomes of these different types of residency programmes.

Fundamental differences are readily apparent in the organisation structure and control of residency training in several of the developed countries. In the United States, the national family practice organisations have no operational responsibility for these programmes, and accreditation authority is vested with the American Medical Association. On the other end of the organisational spectrum is the Australian scheme, whereby a single decentralised national residency programme is conducted by the Royal Australian College of General Practioners in a variety of settings without any ties to departments family practice in the medical schools.

Research:

The development of teaching programmes in family practice has been the first priority in most of the developed countries, so that research development has tended to lag behind. The United Kingdom is the exception to this observation. The Royal College of General Practitioners has strongly supported research for 15 years, and excellent population-based research has been carried out in the defined populations of a number of community practices. During the past ten years, the North American Primary Care Research Group has provided an effective forum for the sharing of research techniques and approaches. Various international groups have further supported family practice research, as for example, the Research Committee of the World Organisation of National Colleges, Academies and Academic Associations of General Practitioners/Family Physicians (WONCA).

The 1970s saw an increasingly effective research effort in family practice being mounted in a number of the developed countries. The content of family practice, studies related to health services and the delivery of care, clinical decision making, the natural history of disease, quality and outcomes of care, and behavioural/family issues. The increasing number, diversity, and quality of citations in the index Medicus in recent years under "Family Practice" provides documentation of the importance of this effort.

Common Weaknesses of Family Practice World Wide:

There is still a general lack of realistic understanding both within and without the medical profession of both the content and demands of Family Practice, and the extent of training needed to serve in that role.

The clinical, teaching and research base for Family Practice is still weak in most of the developed countries. Some of the family units are small appendages of other departments, such as community medicine. Many are not full departments of Family Practice in their own right.

Although not a major problem in some countries, there is still a relative lack of organised continuing medical education for family physicians in most of the developed countries.

Another general weakness is the lack of regular quality control mechanisms as an integral and accepted part of Family Practice.

Action

Organised Family Medicine needs to take co-ordinated and concerted actions to address these weaknesses. There is a need for closer collaboration among Family Practitioners to develop practical and non-threatening self-checking auditing exercises that can be used to compare and contrast practice locally, regionally, nationally and internationally, as well as collaboration with hospital based specialists for the creation of broad and basic consensus guidelines for the care of certain important clinical conditions jointly. Nationally, the leaders of Family Medicine must define national problems and issues and take positive steps to resolve them and internationally, there is much to be done to share experiences and address common problems. The authors of this book have made a good start.

This book is a must on the bookshelves of those involved in family medicine, be they department heads, teachers, medical historians or just family practitioners. To those in countries developing their family medicine departments, this book offers within its covers a sum total of 10 to 15 **years of experience multiplied by eightfold.**

GLG

News from the Council

1) **Ninth College Council 1983-85**

At the 12th Annual General Meeting of the College, held on Sunday, 26 June 1983, the following were elected into office for the 9th College Council 1983-85:

President	Dr Wong Heck Sing
Vice President	Dr Victor L Fernandez
Honorary Secretary	Dr Alfred W T Loh
Honorary Treasurer	Dr Lim Kim Leong
Council Members	Dr Paul S M Chan
	Dr Goh Lee Gan
	Dr Michael P Y Loh
	Dr Frederick Samuel
	Dr Henry P H Yeo

At the first meeting of the 9th Council, held after the A.G.M., Dr James Chang Ming Yu and Dr Leong Vie Chung were unanimously elected Censor-in-Chief and Honorary Editor respectively.

At the second meeting Dr Frederick Samuel resigned from Council and Dr Moti H. Vaswani was subsequently co-opted.

2) **College Fellow**

At the 12th Annual General Meeting of the College, Dr Lim Kim Leong was elected a Fellow of the College.

3) **The 9th College Convocation and 6th Sreenivasan Oration** will be held at Sir Stamford Room, mezzanine level, Hyatt Regency Singapore, on Sunday, 6 November 1983, at 7.00 p.m. Dr Victor L Fernandez, Immediate Past President of the College will deliver the Oration. This will be followed by the College's Annual Dinner, which will be held at the Crystal Ballroom (2nd Level) Hyatt Regency Singapore.

4) **New Members**

The following have been accepted by Council from July/September 1983 into membership of the College:

Dr Ang Poh Kit	Associate Member
Dr Chan Wah Mei	—do—
Dr Cheng Soo Hong	—do—
Dr Cheong Pak Yean	—do—
Dr Chia Chai Nah, Patricia	—do—
Dr Daswani, Chandra Ramchand	—do—
Dr Lim Yong Teeng	—do—
Dr Marrett, Anny	—do—
Dr Soh Eng Liong, Lawrence	—do—
Dr Tan Keng Chiew	—do—
Dr Tan Seok Leng	—do—
Dr Toh Soo Ling, Serene	—do—
Dr Yap Chiew Fah, Ivy	—do—

We welcome them to the College and hope they will participate fully in all activities of the College.

5) **Psychological Medicine Update**

The College's Continuing Medical Education Unit is organising the above course, commencing 16 September 1983. The programme is as follows:

THEORY SESSIONS:

Date	Topic:	Lecturer:
16. 9.83	Schizophrenia	Prof Tsoi Wing Foo FRCP (G), DPM, MRC Psych, FRANZCP
23. 9.83	Depression	Dr Chia Boon Hock MD, DPM, FRANZCP
30. 9.83	Neurosis	Dr Chee Kuan Tsee DPM, MRC Psych
7.10.83	Psychological & Psychiatric Problems among National Servicemen	Col (Dr) Fong Yeng Hoi DPM, MRC Psych
14.10.83	Psychological & Psychiatric Problems of the Aged	Dr Lim Hsin Loh DPM, MRC Psych
21.10.83	Dying and Bereavement:	
	A psychiatrist's viewpoint	Dr R Nagulendran DPM, FRANZCP
	A Family Physician's viewpoint	Dr Leong Vie Chung FCGPS
28.10.83	Sexual Disorders	Dr Kok Lee Peng DPM, MRC Psych
3.11.83	Psychological & Psychiatric Problems among children.	Dr Goh Choo Voon DPM, MRC Psych

CLINICAL DEMONSTRATIONS:

18. 9.83	Case Demonstrations – Schizophrenia	Prof Tsoi Wing Foo FRCP (G), DPM, MRC Psych, FRANZCP
2.10.83	Case Demonstrations – Neurosis.	Dr Chee Kuan Tsee DPM, MRC Psych

HOME STUDY PROGRAMME – Answers to:

MCQs published in Vol. IX No. 1 (January/March 1983)

1. A, B, C, E
2. A, B, D, E
3. A, C, D, E
4. B, C
5. A, B, D, E
6. B, C, E
7. B, D, E
8. D
9. A, D, E
10. A, B, C, D, E
11. A, B, D, E
12. A, D, E
13. A, B, D
14. B, C, D
15. B, C
16. A, C, D, E
17. A, B, C, D, E
18. A, B, C, D
19. A, B, C
20. B, E
21. A, C, E

MCQs published in Vol. IX No. 2 (April/June 1983)

1. A, B, C, D, E
2. B, C, D
3. A, B, C, D, E
4. A, B, C, D
5. A, C, D, E
6. A, B, D, E
7. A, B, C, D, E
8. A, B, C, D
9. D, E
10. A, B, C, D, E
11. A, B, C, D
12. A, C
13. A, B, E
14. D, E
15. B, C, E
16. A, B
17. C, D
18. A, B, D, E
19. B, C, E
20. A, C, D, E
21. A, C, D, E
22. A, B, C, D, E
23. A, B, D
24. A, B, D, E
25. A, B, D
26. C
27. A, B, C, D

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V. SUNDARARAJAN B SC, MBBS, FRCP(E), FRCP(G), DIP DERMATOLOGY, PPA
24 NOVEMBER 1934 – 15 JULY 1983

Professor V Sundararajan, V S Rajan to his friends, Director of the Middle Road Hospital, Associate Clinical Professor of Dermato-Venereology, National University of Singapore, died suddenly and unexpectedly on 15 July 1983, after a very brief illness, at the age of 49, still in his prime of life. His untimely departure from this world was felt by Singaporeans of all walks from the lowest to the highest in the Nation, his professional colleagues, his friends, his family, his patients and the like. Fate in one cruel blow removed a person who had made immense contribution and from whom more was expected, not only towards his chosen profession but to the community at large. It is unfortunate that he had to depart from this world forever with so many things still expected of him. The large number of people from all walks of life who came to pay their respects during his funeral is a tribute to the affection and esteem in which he was held by everyone.

Rajan was born in 1934 at the beautiful Perak town of Taiping, Peninsular Malaysia. He was the second of two sons. He finished his early education at Anderson School, Ipoh. He went to Madras, India in 1946 where he completed upper secondary education and obtained the school leaving certificate in 1950. He went on to University of Madras in 1951 and obtained his B Sc in 1954. He decided to follow the medical tradition of his father. He came as a medical student to the then University of Malaya in Singapore in October 1954, where he studied and obtained his MBBS in 1960. In 1966 he was sent on a Government Scholarship to United Kingdom where he obtained the Diploma in Dermatology of the University of London, and the MRCP (Edinburgh) in 1967.

After graduating in 1960 he did the usual round of postings in various hospitals of the Ministry of Health and ended up in the Middle Road Hospital in 1964. He took a liking to the specialty of dermato-venereology and decided to specialise in it. His rise in his professional career was meteoric. After he returned from his postgraduate studies, he was made the Senior Registrar in 1968, and Superscale Grade 'G' in 1970; becoming a Director and Consultant Dermatologist (Grade E) of Middle Road Hospital in 1976. He was promoted to Superscale Grade D in 1980. He was awarded the Public Administration Gold Medal the same year by the Singapore Government. The College of General Practitioners Singapore honoured him with the prestigeous Certificate of Appreciation in 1980.

Dr Rajan had many facets and his activities covered many fields both in the profession and in community service. Firstly, I would like to talk about his specialty – dermato-venereology. When Dr Rajan and I were students, the specialities of venereology and dermatology were not given sufficient emphasis by part-time teachers; and medical students also gave it mere passing cursory attention. Dr Rajan was committed and dedicated to his specialty. He recognised the importance and he conscientiously wanted to cultivate and upgrade the general knowledge in the discipline of dermatology and venereology amongst not only medical students but also postgraduate students and more important, general practitioners. He ran many in-depth courses and lecture-demonstrations. He took personal interest in every course and lecture conducted by Middle Road Hospital. He strove very hard for increased teaching time and ensured that undergraduate and postgraduate students attending his Hospital for training received a high standard of training in both fields. He also built up relevant teaching materials, in-

cluding audio-visual aids. All this keen interest resulted in the growing number of medical graduates specialising in dermatology and the widespread acceptance of dermatology as a specialty and the average general practitioner's knowledge of this field improved. Members of the College will remember the numerous refresher courses and update sessions he held for our members. It is a personal attribute that not only did he ensure that during these courses sufficient lectures of great depth were delivered, but also suitable clinical materials were brought in for demonstration. Lastly, he also made sure that after a long session at Middle Road Hospital, our members went with a fair bit of good quality refreshment, drinks and cakes. It is a fitting and glowing tribute to his dedication to teaching and upgrading the status of dermato-venereology that the National University of Singapore made him the first Associate Clinical Professor in this subject/discipline. The facet of his personality, that he presented to his patients is another thing. He was a thorough, concerned doctor who was full of empathy for their problems. However, some of his patients have remarked to me that he rarely smiled or looked benevolently. He had a stern and serious countenance. However, they all got better with his treatment, appreciated his professional competence in his field and always recommended him to others and always went back to him. Dr Rajan was a no-nonsense man when it came to work or in dealing with patients and he did not waste time with much ado about nothing in dealings with staff. He always went straight to the crux of the problem.

Dr Rajan was internationally renowned for his work in venereology and dermatology. He had sat on many Expert Committees. Dr Rajan held a number of short-term consultant appointments for WHO, as Consultant in STD and Epidemiology Services in Fiji and also Consultant in STD Control for Kuala Lumpur in 1981. Besides, he was a member of the Technical Committee of the International Union against V D and Treponematoses, Technical Adviser of the National Workshop on STD Control in Kuala Lumpur. He was on the Advisory Committee of the Third International Symposium on Psoriasis at Stanford University, 1981. He was a Committee Member of the American Venereal Disease Association. He was a Member of the WHO Expert Advisory Panel on Venereal Diseases, Treponematoses and Neisserie infections. Besides these, he was on the editorial boards of a number of professional publications, including the Asean Journal of Clinical Sciences, Asian Journal of Infectious Diseases, European Journal of Sexually Transmitted Diseases. In 1982 he was a member of two WHO Scientific Groups on STD attending their meetings in Geneva and Washington, D.C.

His was not an activity fully devoted to his professional field. He was also very active in community service. He was a Member of the Hindu Advisory Board. He took an active role in the work of the Tamil Language & Cultural Society of Singapore, the Tamil Representative Council of Singapore and the Singapore Indian Fine Arts Society. He was for a long time, Member of the Board of Governors of the Academy of Indian Fine Arts. He was a founder member and Vice President of the Singapore Indian Education Trust.

I first came to know Dr Rajan 29 years ago when he came to join as a medical student at the then University of Malaya in Singapore. We were classmates. I had received a special message to look after him and help him to settle down in his new environment at Dunearn Road Hostel. I was told that he was just recuperating from a recent illness and that he was a vegetarian as well. All these called for a great degree of adaptation. The late Dr Rajan was a shy, retiring and reserved person. He had very few close friends in University, and later in life. No doubt he mixed very freely with all his professional and other colleagues with whom he established very good working relationships. But only within the confines of his narrow circle of very close friends would be really open up and occasionally let himself go, let his hair down. He was a very committed and loyal friend as well as a very devoted family man. Notwithstanding this, he also gave so much of himself to the common cause tirelessly. It is sometimes said that God calls to Himself those whom He loves best or those who are close to Him.

Dr Rajan is survived by an aged mother, a young son and a wife, to whom we offer our sincere heartfelt condolences and sympathy. We, in the College share their loss tremendously.

S. Kumarapathy

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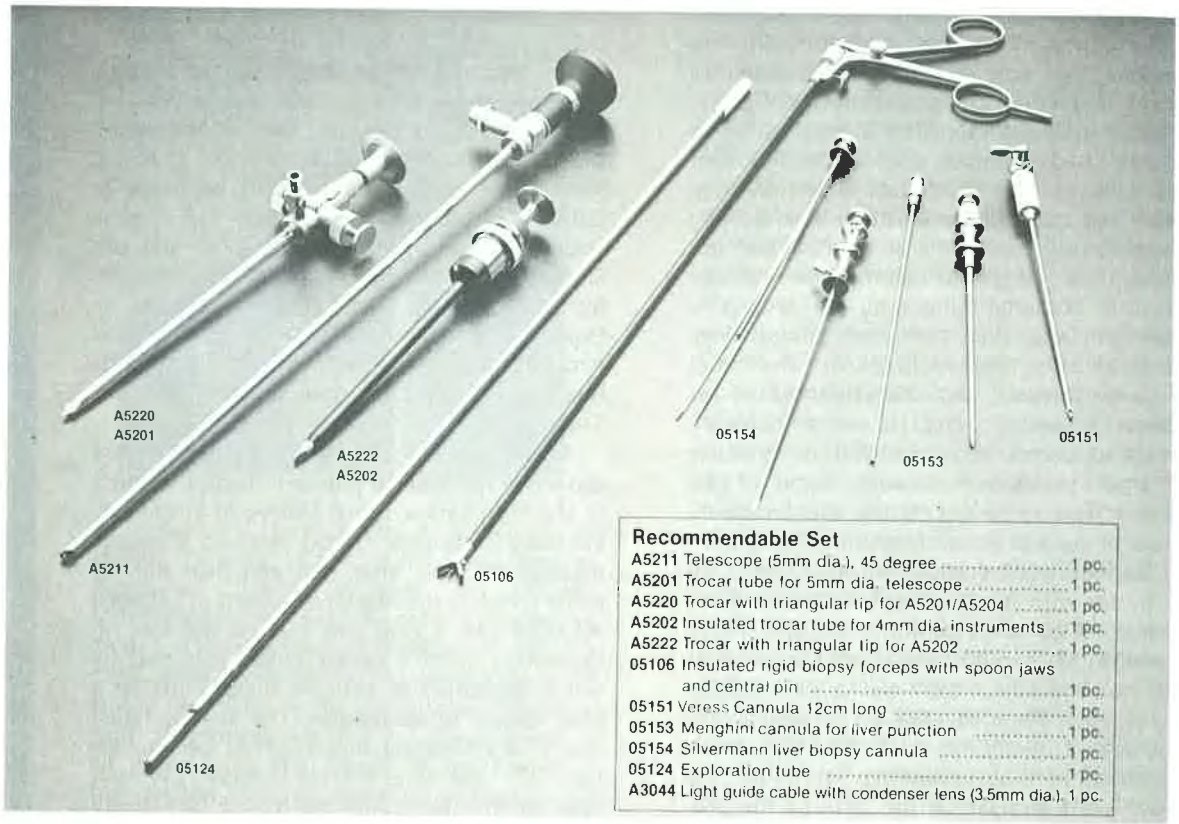

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* WHO — International Code of Marketing of Breast Milk Substitutes, WHA 34.22, May 1981.

* *Codex Alimentarius Commission Joint FAO/WHO food standards for foods for infants and children. CAC/RS 72/74-1976. Rome: Secretariat of the joint FAO/WHO food standards programme 1976.*

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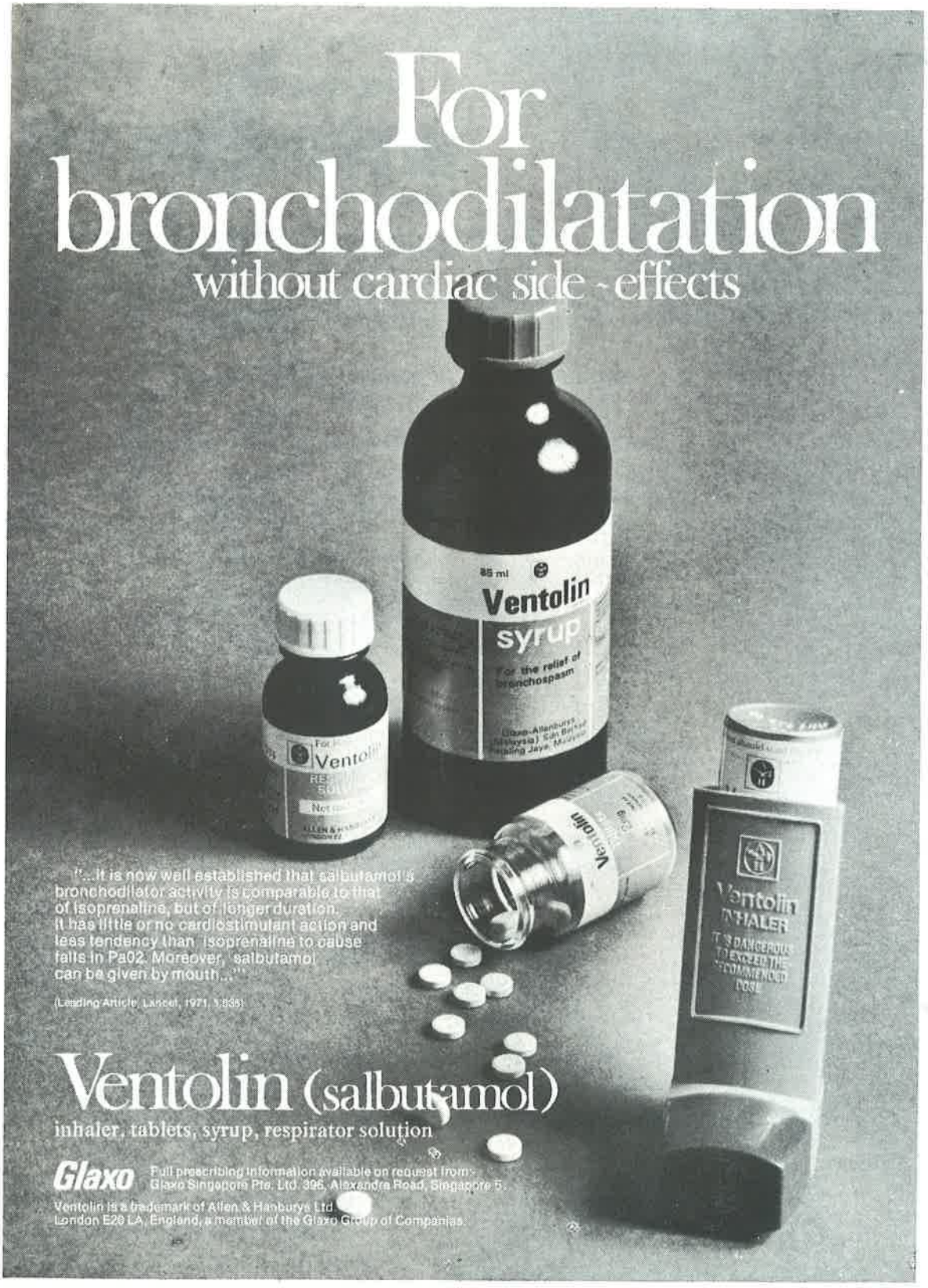
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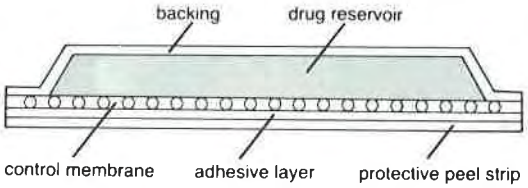

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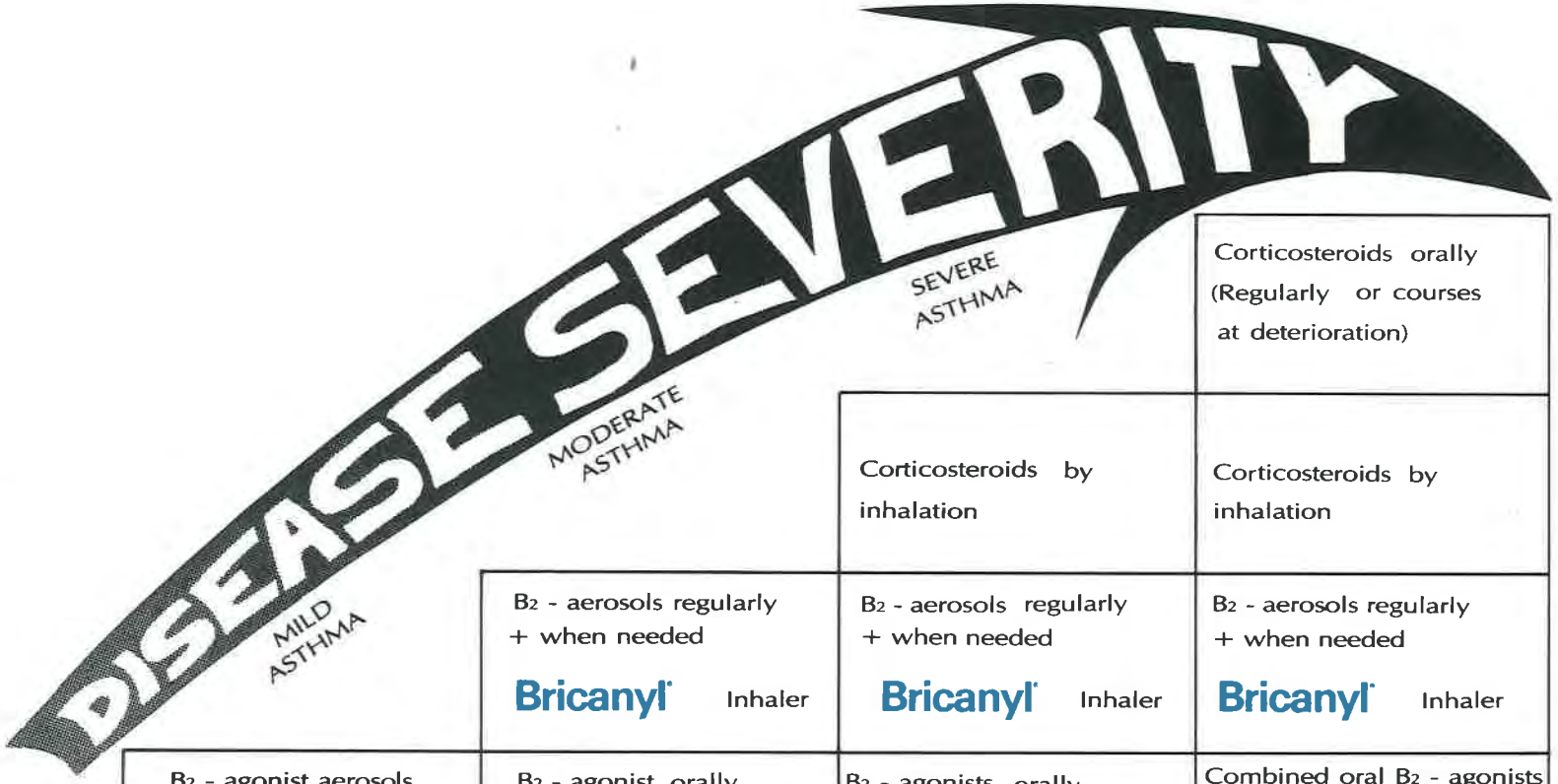

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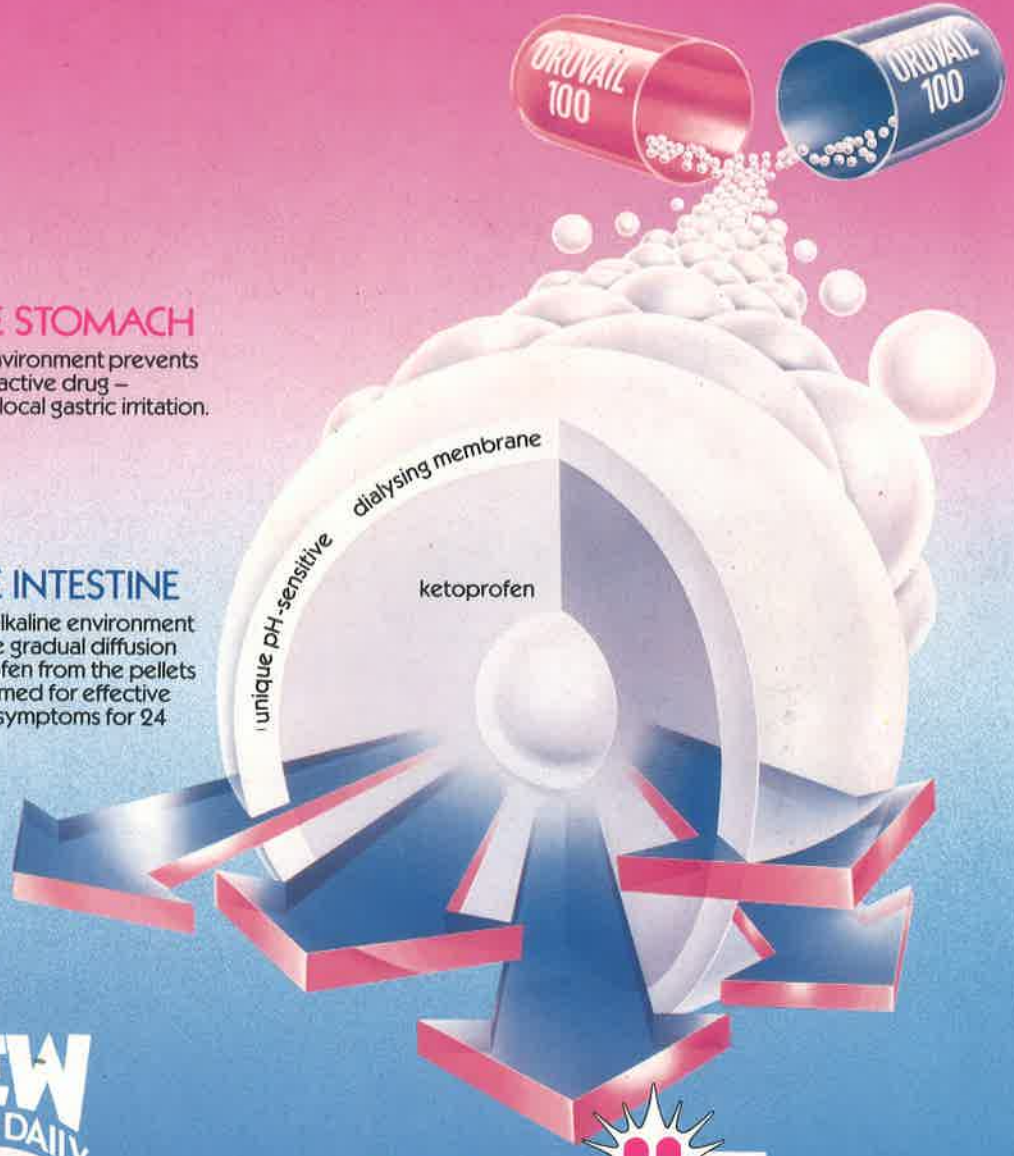

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