

# RES MEDICA

Journal of the Royal Medical Society



## Contents

EDITORIAL	5
OBESITY John F. Munro	6
SUDDEN INFANT DEATH SYNDROME Wm. A Branford	10
THE AUSTRALIA (HEPATITIS-ASSOCIATED) ANTIGEN Christopher Smith, M.B., Ch.B., M.R.C.P.E.	16
OLD MEDICAL BOOKS IN EDINBURGH LIBRARIES: THE ROYAL MEDICAL SOCIETY CATALOGUE PROJECT Geoffrey D. Hargreaves	25
THE DOCTOR AND THE ELDERLY James Williamson	29
THE CONTRIBUTORS	33
SOCIETY NEWS	34
TRAVEL FUND REPORTS John Parker and P.M.A. Calverley	35
BOOKS	38

Copyright Royal Medical Society. All rights reserved. The copyright is retained by the author and the Royal Medical Society, except where explicitly otherwise stated. Scans have been produced by the Digital Imaging Unit at Edinburgh University Library. Res Medica is supported by the University of Edinburgh's Journal Hosting Service: <http://journals.ed.ac.uk>

ISSN: 2051-7580 (Online) ISSN: 0482-3206 (Print)

Res Medica is published by the Royal Medical Society, 5/5 Bristo Square, Edinburgh, EH8 9AL

Res Medica, Autumn 1972, 7(1)

doi:[10.2218/resmedica.v7i1.1972](https://doi.org/10.2218/resmedica.v7i1.1972)

Vol. VII. No. 1

AUTUMN 1972

res  
medica

# JOURNAL

*of the*

ROYAL COLLEGE

*of*

SURGEONS

*of*

EDINBURGH

---

*Published Bimonthly: January, March, May, July,  
September, November*

*Annual Subscription: £5.00 post free*

**Editor: Sir JOHN BRUCE, C.B.E., T.D., F.R.C.S.E., F.R.S.E.**

# *res medica*

## *Journal of The Royal Medical Society*

VOL. VII. No. 1.

AUTUMN 1972

### **Editorial Committee**

#### **Honorary**

*Sir John Bruce, C.B.E*  
*Sir Derrick Dunlop, M.D.*  
*R. J. Kellar, C.B.E.*

#### **Editor**

*Hugh E. Barnes*

#### **Sales**

*William A. Branford*

#### **Business**

*Peter D. Angus*

### **Advertising**

*Mackenzie & Arthur Ltd.*  
*53 George St., Edinburgh, 2.*

PUBLISHED TWICE YEARLY BY THE ROYAL MEDICAL SOCIETY

**IMPORTANT NEW BOOKS  
FOR STUDENTS**

**Publications  
Blackwell Scientific**  
Oxford . London . Edinburgh  
Melbourne

**A COMPANION TO MEDICAL STUDIES**

Editors-in-chief: R. Passmore and J. S. Robson

**Volume 1. Anatomy Biochemistry, Physiology and Related Subjects.** 1968 (Revised Third Printing 1971). 1142 pages, 1056 illustrations, 22 plates (5 colour). Cloth £7.00, limp £5.50

**Volume 2. Pharmacology, Microbiology, General Pathology and Related Subjects.** 1970 (Reprinted 1970). 836 pages, 420 illustrations, 30 plates (14 colour). Cloth £6.00, limp £4.50

**Volume 3. Medicine, Surgery, Obstetrics and Social Medicine.** Spring 1973. 2200 pages, 750 illustrations. Two volumes: cloth about £12.00 each, limp about £7.50 each

**Essential Immunology**  
Roitt. £1.70

**A Dictionary of Immunology**  
Herbert and Wilkinson  
Cloth £2.25, Paper £1.50

**Lecture Notes on General Surgery**  
Ellis and Caine  
4th edn, £2.75

**Lecture Notes on Diseases of the Ear, Nose and Throat**  
Foxen. 3rd edn, £1.50

**Lecture Notes on Dermatology**  
Solomons. 3rd edn, £2.25

**Human Embryology and Genetics**  
Beck. £3.75

**Lecture Notes on Psychiatry**  
Willis. 3rd edn, £1.00

**Diseases of the Nervous System**  
Matthews and Miller. £3.75

**Ocular Pathology**  
Greer. 2nd edn, £5.00

**General Urology**  
Smith. 7th edn, £4.15

**Review of Medical Microbiology**  
Jawetz. 10th edn, £3.90

**Review of Medical Pharmacology**  
Meyers. 3rd edn, £4.15  
**Handbook of Surgery**  
Wilson. 5th edn, £3.20

**H. K. LEWIS PUBLICATIONS**

**BAILEY and LOVE'S**

**SHORT PRACTICE OF SURGERY**

Edited by **A. J. HARDING RAINS, M.S., F.R.C.S.,** and  
**W. MELVILLE CAPPER, F.R.C.S.**

Fifteenth Edition (Reprinted). Pp. xii + 1296. 1592 illustrations. £7.00 net

**MATERIALS AND CLOTHING IN HEALTH AND DISEASE**

By **E. T. RENBOURN, M.D.**

Pp. 612.

£9.50 net.

**THE SIGNIFICANCE OF PHYSICAL SIGNS IN MEDICINE**

By **PETER MILLS, F.R.C.P.** Pp. 106. £2.25 net.

**PRACTICAL PROCEDURES IN DIAGNOSTIC RADIOLOGY**

By **H. M. SAXTON, M.B., M.R.C.P., F.F.R., D.M.R.D.,** and **BASIL STRICKLAND, M.B., M.R.C.P., D.Ch., F.F.R., D.M.R.D.**  
Second Edition. 48 illustrations. £5.00 net.

**PREVENTIVE MEDICINE IN MEDICAL CARE**

By **K. SCHWARZ, M.B., B.Ch., M.R.C.P., D.P.H.** £2.50 net.

**LAW RELATING TO HOSPITALS AND KINDRED INSTITUTIONS**

By **S. R. SPELLER, O.B.E., LL.B., Ph.D.,**  
Fifth Edition. £9.50 net.

**H. K. LEWIS & CO. LTD.**

**136 GOWER STREET, LONDON, WC1E 6BS**

# Contents

## EDITORIAL

OBESITY JOHN F. MUNRO

SUDDEN INFANT DEATH SYNDROME WILLIAM A. BRANFORD

THE AUSTRALIA  
(HEPATITIS ASSOCIATED) ANTIGEN CHRISTOPHER SMITH

OLD MEDICAL BOOKS IN  
EDINBURGH LIBRARIES GEOFFREY D. HARGREAVES

THE DOCTOR AND THE ELDERLY JAMES WILLIAMSON

## SOCIETY NEWS

TRAVEL FUND REPORTS JOHN PARKER &  
PETER CALVERLEY

## BOOK REVIEWS

# Beta Blockade

Sympathetic innervation is mediated through

(a) ALPHA - RECEPTORS (b) BETA - RECEPTORS (c) ALPHA AND BETA - RECEPTORS

Beta - blocking compounds

(a) ACCELERATE HEART RATE (b) DECREASE HEART RATE  
(c) ONLY AFFECT BLOOD PRESSURE

Stimulation of bronchial beta-receptors causes

(a) BRONCHODILATATION (b) BRONCHOCONSTRICTION (c) NO RESPONSE

Beta-blockers must not be used with digitalis

(a) TRUE (b) FALSE

Even if you gave the answers correctly (c, b, a, b), you may be interested in further information on beta-blockade, and on the beta-blocking compounds.

**Inderal**  
TRADE MARK  
PROPRANOLOL  
The standard, reference beta-blocker.

**Eraldin**  
TRADE MARK  
PRACTOLOL  
The only cardioselective beta-blocker.



THE QUEEN'S  
AWARD  
TO INDUSTRY  
1967 1968 1969  
1970 1972

Please write to:-  
Imperial Chemical Industries Limited,  
Pharmaceuticals Division,  
U.K. Medical Products Department,  
Alderley Park, Macclesfield, Cheshire.

Name

Address

Medical School

R.M.(i)

PH794

## EDITORIAL

There has been a heartening increase of discussion recently in medical and other literature, about the "side-effects" of industrial and medical progress and expansion — namely pollution and over-population. It is particularly good to see the medical profession ruminate a little less, and start to call for and take action over the population problem.

Most people, including the Government, tend to feel over-population is a problem mainly confined to the "underdeveloped" countries. It is certainly true that from sheer weight of numbers their problem is that much greater: also, because 40-45% of their population are under 15 years, their populations will continue to rise for some forty years after the fertility level has fallen to the replacement level. But Britain must nonetheless do her share towards population stability, not only for the world's sake but also for her own. We, the U.K., are the eighth most densely populated country; England and Wales alone would be second only to Formosa; and currently births are exceeding deaths by 300,000 every year. The problems of overcrowding and pollution, with all their attendant detriments to health (both physical and mental) are not unknown to us already, and can but increase as the population rises. Ultimately, of course, shortage of food and other natural resources can only lead to malnutrition, starvation, and loss of necessities such as heat and power. The integrated rate of increase of ecological demand on agriculture, industry and mining is now 5.6%/year, and the population increase 2% — we cannot with impunity continue to use up natural resources at such an increasing rate.

And both rates are exponential in type, giving them a somewhat benign appearance at present, but shattering in effect before too long.

The medical profession can do much in the way of prevention. It is also in some ways responsible — not only because better medical care has contributed to the problem but also because the burden of a sick populace will fall on the profession if no prevention is taken. Doctors are a body of highly intelligent people in a particularly good position to influence the public and Government: we should use this influence fully, and now, while success can still be achieved.

The most important means of prevention that should be taken is education. A full-scale campaign is needed, employing all the means of mass education available — television and radio, newspapers and journals, instruction at schools and universities, and possibly even material incentives as already given in India. In support of such education the Government has a vital role: to ensure the continuing liberal nature of the abortion laws, while ruling out regional differences; to extend family planning facilities in the N.H.S., with prescription of free contraceptives; to extend the number of beds for abortion, vasectomy and sterilisation; and, not irrelevantly, to legislate in favour of better career prospects for women. The medical profession must take upon itself the job of bantering the Government until such steps are taken, and itself initiate mass education with all the means at its disposal.

Some people get fanatical about these matters, and cry Doom. Doom is only one alternative — let's take the other.



# OBESITY

John F. Munro

## INTRODUCTION

During last year about a quarter of the adult population of the United Kingdom tried to lose weight, demonstrating that our society is becoming increasingly aware of the problems of obesity. This may partly be cultural and attributable to Twiggy and the miniskirt but may also reflect the relatively recent medical appreciation that not only is obesity the commonest nutritional disorder in the United Kingdom but also a major health hazard. An American male 20% in excess of ideal weight carries an increased mortality of 30% or, in other words it is as dangerous to be 10 lbs. overweight as it is to smoke 25 cigarettes a day.

## DEFINITION & INCIDENCE

Obesity is an excessive accumulation of body fat — but unfortunately it is difficult to make easy and accurate measurements of body fat. The practised fingers are probably as accurate as calipers in measuring skin fold thickness and although weighing by total body immersion may be accurate the facilities to undertake this are not readily available at Chemists or Airports. It seems likely in the meantime that obesity will be related to Ideal Weight which is a Life Insurance Company concept whereby weight is related to optimum life expectancy. This has obvious limitations; one

has only to watch the caponised caperings of modern shot-putters or even the front-row of Scotland's present scrum to appreciate that not everybody who is overweight is obese; likewise some flabby women may carry considerable excesses of fat but remain within a few pounds of their ideal. In spite of these limitations it is usually accepted that patients who are more than 10% in excess of their ideal weight are "OVERWEIGHT" and those more than 20% in excess are "OBESE".

Using these criteria it has been roughly estimated that 1 in 5 of the adult population of the United Kingdom is obese.

## CAUSES.

There are a number of rare endocrine causes for obesity; these include Hypothyroidism, Hypogonadal eunuchism, Cushing's Syndrome and Steroid therapy, Stein-Leventhal Syndrome, and various pituitary and hypothalamic disorders. The vast majority of patients suffer from non-endocrine or "Simple" Obesity — a totally inappropriate adjective but it cannot be denied that people with simple obesity are eating or have eaten more calories than their metabolic requirements. This may arise in a number of ways.

1. *Gluttony.* A few people eat excessively because they enjoy food; I can recall one who admitted to eating 12,000 calories a day but whose problem was his alcohol consumption.

A pint of draught export contains about 200 calories. Bearing this in mind it is remarkable how few medical students are overweight.

2. *Psychological.* Many overeat in order to obtain psychological relief during periods of idleness, boredom, anxiety or depression. In some the cause of that depression may be their obesity and to others this is an additional factor. Thus a vicious circle can arise with obesity leading to depression causing overeating with further weight gain. An extreme example of psychological overeating is the rare syndrome of nocturnal gorging.

3. *Altered Homeostasis.* A normal person has a number of homeostatic mechanisms whereby he can avoid or burn off excess calorie consumption. Thus, lean subjects can accurately evaluate the nutritive content of a liquid diet and only take their daily requirements; if deliberately overfed the resultant weight gain is less than anticipated. Obese patients either lose or never possess these homeostatic mechanisms and cannot assess nutritive values nor burn off excessive calories which are inevitably converted into body fat. To eat an extra apple a day produces a theoretical weight gain of  $\frac{1}{2}$  stone a year.

### METABOLIC CONSEQUENCES

Simple obesity produces a complex chain of metabolic changes. These include increase in serum insulin but reduction in peripheral insulin activity causing reduced Glucose assimilation by muscle; growth hormone levels tend to fall but adrenal cortical activity increases. Patients are resistant to ketosis.

The net effect of these changes is that the patient becomes metabolically more efficient and thus a second vicious circle situation develops, obesity itself tending to perpetuate the obese state. Although it seems probable that complex enzyme mechanisms are involved one simple minded way of explaining the situation to a patient is to say that subcutaneous fat acts like double-glazing and by retaining body heat reduces the daily caloric requirements. Although these changes may be responsible for aggravating and perpetuating obesity they are not the direct cause and are corrected by successful weight reduction. Recently, however, it has been demonstrated in obese children that those who were overweight as infants possess more adipose cells than those who subsequently gain weight. It is possible that this is of long-term prognostic significance in that the number of fat cells is related

to eating habit in infancy, and the more fat cells one possesses the more difficult it is to reduce. Puppyfat is not 'benign' and the vast majority of obese children become obese adults. In view of the rarity of mal- or sub-nutrition and the hazards of childhood obesity the recent fuss about free school milk appears totally inappropriate.

### SIGNIFICANCE

In addition to the very considerable increase in mortality almost exclusively due to ischaemic heart disease, obesity is also associated with considerable morbidity. Examining from the toes upwards it may cause flat feet, ankle oedema, varicose veins, knock knees, osteoarthritis of the hips and other weight bearing joints, genital prolapse, menstrual irregularities, possibly urinary tract infections, gallstones, hiatus hernia, Pickwickian Syndrome, and a liability to respiratory infections in childhood. It also aggravates the symptoms of any co-existing respiratory or cardiac disease, is associated with diabetes and increases the incidence of post-operative and obstetric complications.

In addition the grossly obese are invariably psychologically disturbed and the cheerful ever-smiling "fatty" is wearing a mask to protect himself from his feeling of inadequacy which may be the product of years of inappropriate ridicule.

### TREATMENT

#### *General Considerations.*

The treatment of obesity comprises two phases, an initial period of weight loss and subsequent prevention of weight regain. The second phase is often neglected but temporary weight loss is only of temporary benefit and many find it even more difficult to reduce a second time. It is thus very important to explain to patients that once they have reached an acceptable weight they will have "to watch their diet" and this usually involves a basic alteration in eating habit. Some patients will lose weight remarkably easily but others, particularly those with psychological problems, will require considerable encouragement and support. Unsuccessful attempts at weight loss may harm the more severely disturbed and it is essential to ensure that the patient is appropriately motivated before commencing treatment. Patients should also have a realistic concept of the rate of weight reduction. Many anticipate a 2 week miracle but Magic Wands are not available on the N.H.S. To gain

weight at the rate of 1 stone per year is disastrous but to reduce at 1 lb. per week may appear unacceptably slow yet it produces a weight loss of nearly 4 st. a year. Many give up because they expect too much and become disheartened by perfectly acceptable progress. Body fat stores will only be reduced when calorie expenditure exceeds calorie intake.

#### Exercise.

The exercise required to burn off one pound of fat is equivalent to playing 20 sets of tennis or walking from Glasgow to Edinburgh. Such activities are often associated with increased calorie consumption, but in spite of this, physical exercise is an important if subsidiary and somewhat neglected adjunct to weight reduction. It is noteworthy that obese adolescents tend to take much less exercise than their lean counterparts.

#### Diet.

The sheet anchor of treatment must be dietary restriction. Numerous different dietary regimens have been fashionable but a calorie is a calorie and the dramatic early weight losses associated with high protein, or high fat diets merely reflect the initial water diuresis that follows restriction of carbohydrate. This medical fact has been converted into financial gain by the exponents of the "Grapefruit Diet" and other off-beat regimens.

For a diet to be effective it must be reasonably cheap, easy to follow and provide flexibility and variety. The normal intake in the United Kingdom is such that these aims and total calorie restriction can be achieved most easily by carbohydrate restriction, the simplest of which comprises 3 groups of food, one forbidden, one to be taken in unlimited quantities and the third in restricted quantities — the number of portions permitted depending upon age, occupation, etc.

Failure to lose weight implies lack of dietary adherence but sometimes this is unintentional as occurred with the patient who was seduced by T.V. into thinking that Lucozade was good for her. Those who cannot adhere are either "nibbling" between meals or "gorging" at meals or doing both. Some find it easier to adhere to more rigid regimes. These include "Two meals per day" which reduces opportunities of temptation and the "Five meals per day" diet which makes use of the increased calorie expenditure that occurs after meals.

Others may respond to formula diets using Metarcal or Limmits, Trimmets etc. though it

must be emphasised that these replace rather than supplement meals.

#### Drug Therapy.

Various drugs have been advocated; but should only be administered in combination with dietary control and only when this is failing to produce weight loss. They include Thyroxine which is contra-indicated except in hypothyroidism and the bulk agents such as Methyl Cellulose — a substance remarkably free from side — and therapeutic effects. Amphetamine and the amphetamine derivatives are C.N.S. stimulants with definite weight reducing properties. Their potential danger as drugs of addiction is reflected by the price they command on the Black Market, and there is considerable pressure to ban their use. This appears extreme but they should only be prescribed under close medical control. They lose their weight reducing effect after 6 to 8 weeks, should be avoided in the psychologically disturbed and are contra-indicated in patients with hypertension or cardiac disease. Intermittent therapy with courses of not more than 4 weeks duration is preferred as it is as effective as continuous therapy, cheaper, and presumably less likely to cause addiction.

Although chemically related to amphetamine fenfluramine does not produce C.N.S. stimulation and indeed often causes sedation. If injected intra-arterially it has a fat mobilising effect and produces metabolic changes comparable to exercise. Its mode of action when given orally remains uncertain. It may be more valuable administered continuously but its weight reducing effect though sometimes dramatic is very variable and a significant proportion of patients either fail to respond or develop troublesome side-effects. The Diguanydes are another group of drugs which may be of value. In obese diabetics they abolish Glycosuria without producing the weight gain which occurs with Sulphonylureas. In some non-diabetics they cause weight loss, though whether this is secondary to an anorectic effect or alteration in body metabolism is debatable.

#### DRASTIC METHODS OF WEIGHT REDUCTION.

In spite of these various methods some patients will fail to lose weight or stop losing weight while still grossly obese. Previous experience has shown that once "Refractory Obesity" develops it is extremely difficult to achieve further sustained weight loss without

resorting to drastic measures.

(a) *Total Starvation.* Although practised since biblical times starvation has only recently become medically acceptable. It is not without risk and the complications include sudden death, cardiac arrhythmias, electrolyte and in particular Potassium imbalance, muscle catabolism, various nutritional deficiencies, hyperuricaemia, menstrual irregularities, alopecia, alteration in libido and psychological stress. With a appropriate selection and replacement therapy the major complications can be avoided. There is, however, no evidence to suggest that the long-term results of starvation are superior to more conventional therapy when undertaken for relatively short periods (i.e. up to 3 weeks) in patients without Refractory Obesity and in those with Refractory Obesity the weight lost during such treatment is almost invariably regained following discharge. However, a proportion of patients with gross refractory obesity starved to within 20% of their ideal will not regain weight but at best it seems probable that starvation is only indicated in a small proportion of grossly obese patients with Refractory Obesity.

(b) *Surgery.* Enthusiasm for bypass surgery has been rekindled by the reported successes of the 14/4 operation in which 14 inches of proximal jejunum are anastomosed to the

terminal 4" of ileum. The operation produces severe steatorrhoea and may lead to hepatic insufficiency. Experience in Edinburgh is limited and disappointing but further evaluation is probably justified. Surgery is also indicated to remove unsightly skin folds that sometimes develop following massive weight loss.

#### TREATMENT POLICY

It is important to try and prevent the development of Refractory Obesity by altering therapy whenever a patients stops losing weight. Although each patient must be treated individually one possible treatment scheme is summarised below, each step being continued until weight loss stops.

1. Establish rapport, assess motivation, give the patient realistic objectives, and prescribe low carbohydrate diet.
2. Add in either fenfluramine or intermittent therapy with an amphetamine derivative, (e.g. Phentermine) or accept the status quo.
3. Try one or more of the following in turn.
  - (a) Special dietary regimes
  - (b) Diguanydes
  - (c) High Dosage fenfluramine or accept the status quo.
4. Consider total starvation or bypass surgery or repeat anorectic Therapy or accept the status quo.

### OFFICE BEARERS FOR THE 236TH SESSION

Senior President .....	N. J. DOUGLAS
1st Junior President .....	P. D. ANGUS
2nd Junior President .....	Miss UNA M. MacFADYEN
3rd Junior President .....	W. A. BRANFORD
Senior Secretary .....	R. M. SIMPSON
Junior Secretary .....	A. M. NORTON
Convener of the Business Committee .....	Miss UNA M. MacFADYEN
"    "    "    Publicity Committee .....	Mrs. BEVERLEY NORTON
"    "    "    House Committee .....	J. B. SHERMILT
"    "    "    Library Committee .....	M. K. PALMER
"    "    "    Museum Committee .....	M. A. R. KNOCK
"    "    "    Dinner Committee .....	J. R. ANDERSON

# SUDDEN INFANT DEATH SYNDROME

Wm. A. Branford

Sudden infant death has only recently begun to attract widespread publicity and medical investigation. It is still largely ignored by the standard paediatrics textbooks and the only description of it given in formal lectures to Edinburgh medical students comes in the Forensic Medicine course. This perhaps helps to serve one useful purpose in that it may be an indication as to why the sudden infant death syndrome is still almost completely unexplained. These unfortunate children are usually found dead and, if they reach hospital at all, it is only as far as the accident and emergency department on their way to the mortuary. Most go directly to a coroner's or procurator fiscal's post-mortem, and are thus relatively insulated from the research-orientated clinician's attention and interest.

This syndrome is certainly no new phenomenon. Up to the end of the last century doctors were satisfied that these deaths were due to "teething", a wide category that included many deaths for which an adequate explanation would now be found. The 1839 Registrar-General's returns showed a total of 5,016 infant deaths due to this "cause". The fashion then changed towards attributing them to "status thymolympathicus", although exactly why seems to be unknown.

Gradually, however, the position is becoming somewhat clearer. Werne and Garrow in the United States produced some of the first significant papers during and immediately after the second world war. In Britain Professor Sir Samuel Bedson and Dr. F. E. Camps (1951) submitted a memorandum to the Ministry of Health, estimating an annual toll of 200 children, and suggesting an official investigation which finally bore fruit under Professor Banks in the 1965 Report.

The 1st International Conference held in Seattle, Washington, in 1963 firmly put the sudden infant death syndrome on the medical map, and although some still persist in ascribing all these deaths to accidental suffocation, overwhelming infection or infanticide, most will now accept the existence of this syndrome as a distinct entity.

If any worthwhile comparable epidemiological studies are to be made on this topic, then a definition of the syndrome must be universally accepted. There is considerable lack of agreement even on what to call it: cot or crib death, sudden unexpected death, sudden unexplained death in infancy, undiagnosed death in infancy and many others have all been suggested and used, but the 2nd International Conference on Causes of Sudden Deaths in Infants (1969), after much discussion, finally settled for Sudden Infant Death Syndrome (SIDS) as the term causing least confusion. Pathologists have yet to come up with any consistent finding and using this as a basis Dr. J. B. Beckwith produced the following definition at this Conference: "the sudden death of any infant or young child which is unexpected by history and in which a thorough post-mortem examination fails to demonstrate an adequate cause for death".

It was also proposed that the minimal acceptable investigation into one of these deaths should include:

1. Adequate history
2. Gross examination including thorax, abdomen, brain, entire larynx ( $\pm$  spinal cord)
3. Blood culture
4. Histological examination including brain, liver, lungs, heart, kidneys and other organs as indicated by 1 and 2.

5. Ancillary studies (toxicological, chemical, special cultures, virological studies and so forth) as indicated by results of above.
6. Counselling of family.

Most of the recently-reported epidemiological investigations have used, as far as was possible, these criteria.

The first thing to determine is its actual incidence in the community. How common is it? Is this a real problem or is it just another weird and wonderful syndrome that makes good reading in the journals, but which few are ever likely to see? One major difficulty has been that something "appropriate" was usually recorded on death certificates and SIDS or its equivalent was not used, making figures impossible to obtain from this source. The Registrar-General does now allow the use of the term "cot death syndrome". The 1965 M. o. H. report estimated that 83% of deaths certified by coroners as due to "respiratory causes" were in fact "cot deaths".

Most of the papers written on SIDS have come from Western cultures and they show a remarkable uniformity (Table 1).

Very similar figures were reported by Houstek (1970) from Czechoslovakia, who thought that the incidence was about the same in Yugoslavia, Poland and the U.S.S.R., although little data has come from these nations. Doctors working in underdeveloped countries have stated that sudden death in infancy is common, but no post-mortem is

usually available and any comparison of figures is therefore impossible.

In reviewing the statistics in Table 1, Dr. Marie Valdés-Dapena remarked at the 1969 Conference that it would appear that the incidence of SIDS increased with urbanisation and that the lowest figures seem to occur in rural areas.

The survey showing the highest incidence was that conducted in Canada (Steel 1970), closer examination of the figures revealing that it increased the further north one went. This would suggest some relationship with the temperature or weather conditions in general.

Froggart et al (1971) have conducted a major investigation in the U.K. by studying SIDS in Northern Ireland. Their results agree with most others by showing an excess of deaths during the winter months. This peaking was greater during the first year of study (August 1965 to July 1966) and it is interesting to note that epidemics of Influenza type B and type A2 occurred in this area during January to February 1966 and February to March 1966 respectively. In the Southern Hemisphere Australian figures show a peak in June, again during the colder season. This winter increase is not due to the relatively unimportant monthly fluctuations in live births.

There is some disagreement as to whether the incidence varies with the day of the week. Some state SIDS to be commoner over the weekend, suggesting an element of neglect, but other studies do not confirm this.

TABLE 1 (Valdes-Dapena 1970)  
(re-arranged)

Author	Year	Place	No. / 1000 Live Births	Subjects
Ministry of Health (GB)	1965	England & Wales	1.4	All infants
Carpenter	1965	England & Wales	2.2	
Peterson	1966	Seattle	(2.87 (2.71 4.66)	Caucasians Non-whites (Negro and American Indians)
Steel et al.	1967	Canada	3.00	All infants
Froggart et al	1968	Northern Ireland	2.3	All infants
Valdés Dapena et al	1969	Philadelphia	2.55 (1.41 4.32)	Caucasians Non-whites (negro)
Fitzgibbons et al	1969	Olmstead Co., Minn.	1.2	All infants

Virtually all are, however, agreed that the majority of sudden infant deaths occur during the normal household sleeping hours, which is quite different from the other causes of death in infancy. Most are discovered dead when the infant is looked at for the first time in the morning.

What then is found when individual cases are looked at? The Northern Ireland study showed a slight predominance of males, but this followed a similar pattern for infant deaths generally. Results of other investigations have ranged from a male excess through equality to a female excess, so there is probably no marked difference.

The most striking epidemiological finding is undoubtedly encountered when the age of the infants is considered. It is a constant result that there is a genuinely low prevalence in the first two weeks of life, followed by a rise to a peak incidence at around three months and then a rapid decline. The very low incidence in the first two weeks of life contrasts markedly with the other causes of infant mortality. Only rare cases occur after one year of life.

Templeman (1892) was the first to record that SIDS is commoner in families of lower social class. He, however, considered that all these deaths were due to overlaying by their

mothers as a result of carelessness, drunkenness or a desire to collect small sums of insurance money (up to 45/-). He was probably right in a number of cases, but it is thought that his description tallies closely enough with the modern concept of SIDS to include it here. Most recent studies have given at least an impression of the social conditions of families in which a sudden infant death occurs (Table II).

Froggart et al (1971) put their findings on a more scientific basis, and demonstrated a clear excess of deaths in Social Class V and a deficit in Classes I and II.

Illegitimate children and those of mothers under 20 have a higher incidence. Health visitors tend to have visited the homes of victims more often than those of unaffected children and their mothers have made fewer visits to child welfare clinics. It must be remembered, however, that this reflects only a general pattern, and there are marked deviations from it: SIDS can strike at any family.

As can be seen from Table I, American workers have found a higher incidence in negroes: this holds good even when correction is applied for the higher over-all negro infant mortality. The discrepancy between Caucasians and negroes is highest amongst the poor,

TABLE II  
(Valdés-Dapena 1970)

Authors	Year	Place	Total No. of Cases	Impression
Cooke & Welch	1964	W. Hartlepool	91	Preponderance of deaths in lower social groups
Carpenter & Shaddick	1965	England & Wales	110	Lower social class of wage earner. Poorer home and mothering
Sutton & Emery	1966	Sheffield	10	The squalor of the families is outstanding
Marek et al	1966	Poland	200	Living conditions bad in a considerable percentage
Hildebrand	1967	Hamburg	216	Many poor with deficient housing, disorderly, unclean and crowded.
Steele et al	1967	Canada	66	Poor general care of the infant Mother at all levels of educational attainment
Melton et al	1968	Richmond, Va.	199	Poor economic level, poor care of infants. Disproportionate number of negroes (50% vs 20% in population)
Fitzgibbons et al	1969	Minneapolis, Minnesota	46	Lowest reported incidence among farming people and middle-class urban dwellers

leading to the conclusion that there is an "increased incidence among negroes no matter what the socioeconomic level and an increased rate among the socioeconomically disadvantaged, independent of race". (Valdés-Dapena 1970).

As I have already pointed out, most of these children are found dead; few, if any, are seen to die. Some mothers claim to have found their babies alive but blue and have attempted to resuscitate them. This, however, is probably a natural reaction and the baby may well have been dead for some time. Another characteristic feature is that these children can die in the same bed as other children or in the same room as their parents and nothing is heard by them — the deaths are apparently silent. SIDS has been reported to have occurred in children in hospital without the staff detecting anything amiss (Gray 1971).

Retrospective studies have been carried out on the previous health of these infants and that of their mothers, especially during pregnancy. Prematurity may slightly increase the risk, but there is no evidence of an increase in "failure to thrive" in cases of SIDS. It appears to be commoner amongst twins but this may be a reflection of their lower birthweight, which is an important factor in infant death generally. The Northern Ireland group found no evidence of heredity playing any part, and the mothers of these children had no increase in foetal loss in their obstetric history.

Health of the children immediately prior to death is difficult to assess due to recall bias on the part of the parents and the problems of obtaining suitable control groups. Some results suggest an increase in minor illness (coryzal or digestive symptoms) in the week, and more especially the 24 hours, prior to death, but others disagree with this. There is certainly no history of major illness or anything to suggest that the child would shortly die.

This then is very briefly what epidemiology has contributed in attempting to unravel this problem. It is doubtful that it will produce much more of significance. Dr. A. B. Bergman, at the 1969 Conference, said "Epidemiology has only so much to offer — we mustn't try to squeeze out more juice than the whole orange possesses. I would make the rather dogmatic statement that except for the testing of specific hypotheses, large-scale epidemiological field investigations are apt to be repetitive and are not likely to produce new useful information."

With this in mind, combined with the pathological findings, what then can we say

about the present theories as to the cause of SIDS? No significant "at risk" factors can be isolated and this, combined with the characteristic age-range, mediates against the view that these infants have any as yet undiscovered 'disease'. Froggart et al (1971) suggests that as the infant is "passing through a developmental stage of physiological vulnerability some critical combination of extrinsic and intrinsic factors occurs which proves lethal." Just what is the "final common pathway" of these deaths causes the mystery and here epidemiology can be of little help.

I do not intend to attempt to discuss in any detail the enormous number of suggested causes, some of which are presented in Table III (an extension of one by Valdés-Dapena (1970)).

This is, of course, by no means a complete review of the literature. Evidence now exists, if not to refute, at least to throw doubt upon several of these hypotheses. That supporting others is, to put it mildly, tenuous. Some of them, although extremely ingenious, do not help at all to explain the epidemiological data. Perhaps the strongest support is given to viral infection being the major "extrinsic factor", although there is good evidence to back milk allergy. James' (1970) work on the development of the conduction pathways in the heart may well provide a clue as to the "physiological vulnerability" although a recent study has failed to confirm his findings (Valdés-Dapena 1971).

Froggart has called SIDS a "disease of theories", and until much more detailed scientific investigation is undertaken, particularly in trying to define whether the deaths are primarily pulmonary or cardiac, then little clarification can be expected.

No recipe is available for the prevention of these tragic deaths. It is thought probable by some that breast feeding is the most effective prophylaxis if continued for months, but even this by no means provides complete immunity, as 2 out of the 162 in the Northern Ireland series were entirely breast fed. The Ministry of Health report recommended that infants should sleep without pillows as their figures indicated that these were used in a significantly higher percentage of SIDS cases than in controls.

If a figure of 2.5/1,000 live births is accepted as the incidence in the United Kingdom, then this accounts for 10% of all infant mortality and 30% of post-neonatal mortality (28 days — 1 year of life) (Froggart et al 1971).



Table III  
SUGGESTED CAUSES OF SIDS

1. Accidental suffocation .....	Bedson, S. (1951) see Appendix I to Reference No. 13
2. Atlanto-axial occipito-atlantoid dislocation ...	Englander, O. (1971) B.M.J., 4, 625
3. Cardiac arrythmia .....	James, T. N. (1970) in Bergman et al pp. 118-120
4. Cortisol insufficiency .....	Finlayson, N. B. (1964)*
5. Electrolyte imbalance .....	Maresch, W. (1964): McGaffey, H. (1968)*
6. Epidural haemorrhage .....	Towbin, A. (1968)*
7. Gamma globulin defect .....	Leading article (1971) B.M.J. 4, 250
8. Hyperactive dive reflex .....	Wolf, S. (1964, 1965, 1966, 1968)*
9. Hypersensitivity to cow's milk .....	Parish, W. E. (1960), Lancet, 2, 1106
10. Immune complex disease .....	Urquhart, G. E. D. et al (1972), Lancet, 1, 210
11. Infanticide .....	Asch (1968)*
12. Infection (a) viral .....	Johnstone, J. M. (1966)* etc.
(b) bacterial .....	
13. Inhalation of vomited milk .....	M.o.H. (1965) Report on Public Health and Medical Subjects No. 113
14. Nasal obstruction .....	Shaw, E. B. (1968)*
15. Parathyroid insufficiency .....	Geertinger, P. (1967)*
16. Precapillary bypass .....	Jaykka, S. (1971), Lancet, 2, 1315
17. Saliva .....	Campbell, K. (1971), Lancet, 2, 1314
18. Stress .....	Bohrod, M. G. (1963)*
19. Vitamin E and Selenium .....	Money, D. F. L. (1971), B.M.J. 4, 559

\* See original list in Bergman et al (1970) p.12.

The 1965 Ministry of Health "Enquiry into Sudden Death in Infancy" put the problem succinctly into perspective by stating that "it would appear that the risk of unexplained sudden death before reaching the age of two is about twice that of being killed on the roads before leaving school".

This is not an inconsiderable proportion of early childhood mortality. The vast majority of the victims of SIDS have appeared perfectly healthy up to their sudden demise. It is an obvious cause of great distress and needless feelings of guilt in the parents, whose acquaintances may "show or feel cruel primitive rejection" (B.M.J. Leading Article, 1971). At least the recognition of this entity by the medical profession has led to kinder treatment of the parents by the press in their reporting of inquests.

A great deal of money and research effort is at present directed towards prolonging, in an often unsatisfactory manner, the lives of congenitally deformed children, whereas little is spent in the U.K. on trying to establish the cause of this perplexing problem.

These children, some 2,000 a year in Great Britain alone, are mysteriously deprived of their

chance to become normal, intelligent, active members of the community with apparently little concerted effort to determine why.

Valuable work in publicising SIDS and raising the money for research into it is undertaken in the United States by lay organisations, e.g. The National Foundation for Sudden Infant Death (New York). Recently similar bodies have been formed in the U.K.: The British Guild for Sudden Infant Death Study and The Foundation for the Study of Infant Deaths. The latter organisation is attempting to raise £500,000 to support research.

They also provide much-needed help and comfort to families in which one of these tragedies occurs, help that is all the more valuable as many of the members of these organisations have experienced SIDS within their own family group, and are thus uniquely qualified to be of assistance to those in need of it.

Even if the cause of SIDS is never found and it can never be prevented, there is much that can be done even now towards reducing its effects on the other members of the family. Unfortunately the necessary police investigations to exclude unnatural death, the post-

mortem and inquest, if held, all add to the parents' grief.

Someone must take the time to explain to them what is known about SIDS, in particular that no blame can be attached to anyone and that there is no indication that it is likely to recur in subsequent offspring.

Emery (1972) suggests that the best person to take on the role of counsellor would be the local paediatrician. The General Practitioner would seem to be the obvious choice but approximately one-third of affected families have been found to change their G.P. after a "cot death" has occurred. The health visitor is similarly placed in a difficult position.

There is a heavy psychiatric toll, particularly amongst the mothers of these children (Bergman et al 1969). The symptoms of grief should be discussed with the parents, medication may be temporarily required for insomnia or anxiety and if possible a close watch should be kept for marital problems and symptoms of emotional disturbance in siblings.

Future children born to the couple may well be in danger from the effects of over-protection, excessive care and surveillance leading to almost inevitable consequences on their future emotional development.

The sudden infant death syndrome therefore embraces more problems than its aetiology alone and should not, to my mind, be confined to the realms of forensic medicine.

## REFERENCES

- BERGMAN, A. B. (1970): Sudden Infant Death Syndrome in King County, Washington: Epidemiological Aspects: In Bergman et al, (1970), pp. 47-54.
- BERGMAN, A. B., BECKWITH, J. B. and RAY, C. G. (Eds.) (1970): Sudden Infant Death Syndrome: Proceedings of the 2nd International Conference on Causes of Sudden Death in Infants: University of Washington Press, Seattle.
- BERGMAN, A. B., POMEROY, M. A., BECKWITH, J. B. (1969): The Psychiatric Toll of the Sudden Infant Death Syndrome: G.P., xl No. 6, 99-105.
- CAMPBELL, K. (1971): Possible Role of Saliva in SIDS: *Lancet*, 2, 1314.
- CAMPS, F. E., CARPENTER, R. G. (Eds.) (1972): Sudden and Unexpected Deaths in Infancy (Cot Deaths): Report of the Proceedings of the Sir Samuel Bedson Symposium held at Addenbrooke's Hospital, Cambridge on 17 and 18 April, 1970. John Wright & Sons Ltd., Bristol.
- EMERY, J. L. (1972): Welfare of Families of Children found Unexpectedly Dead ("Cot Deaths"): *British Medical Journal*, 1, 612-615.
- FROGGART, P., LYNAS, M. A. and MACKENZIE, G. (1971): Epidemiology of Sudden Unexpected Death in Infants ("Cot Death") in Northern Ireland: *British Journal of Preventive and Social Medicine*, 25, 119.
- GRAY, O. P. (1971): Sudden Deaths in Infants. *Lancet*, 2, 1315.
- HOUSTEK, J. (1970): Sudden Infant Death Syndrome in Czechoslovakia: Epidemiological Aspects: In Bergman et al (1970), pp. 64-72.
- JAMES, T. N. (1970): New Observations in the Heart: In Bergman et al, (1970): pp. 118-120.
- LEADING ARTICLE (1971): Cot Deaths: *British Medical Journal*, 4, 250.
- MINISTRY OF HEALTH (1965): Enquiry into Sudden Death in Infancy: Reports on Public Health and Medical Subjects No. 113: H.M.S.O. London: Includes BENSON, S., CAMPS, F. G. (1951). Memorandum to M.o.H. as Appendix I.
- STEELE, R. (1970): Sudden Infant Death Syndrome in Ontario, Canada: Epidemiological Aspects: In Bergman et al (1970): pp. 64-72.
- TEMPLEMAN, C. (1892): Two-hundred and fifty-eight cases of Suffocation of Infants: *Edinburgh Medical Journal*, 38, 322.
- VALDES-DAPENA, M. A. (1970): Progress in Sudden Infant Death Research 1963-69: In Bergman et al (1970), pp. 3-13.
- VALDES-DAPENA, M. A. (1971): National Foundation for Sudden Infant Death: News Letter IX, p.5

# THE AUSTRALIA (Hepatitis-associated) ANTIGEN

Christopher Smith, M.B., Ch.B., M.R.C.P.E.

---

"Most great discoveries are accidental"  
*Sir Henry Dale*

"Discovery comes to the mind prepared"  
*Louis Pasteur*

## INTRODUCTION

The accidental discovery of the Australia antigen (HAA) in 1963 by Blumberg and his coworkers is a classical example of biomedical serendipity. Its recognition not only stimulated a substantial amount of research into viral hepatitis, thereby adding considerably to our knowledge about this important disease, but has had far-reaching implications in the understanding of several other hepatic and systemic diseases. Indeed, its discovery may well be seen in the future to have been the all-important breakthrough in attempts at culture of the virus as a means of vaccine preparation for future prophylactic usage.

## THE DISCOVERY OF THE ANTIGEN

Blumberg, a cytogeneticist working in the Philadelphia Institute for Cancer Research, was conducting a systematic study of the serum of patients who had received multiple blood transfusions. Using a technique of double diffusion in agar gel (Ouchterlony method) he searched for precipitating antibodies to inherited donor serum protein antigens, particularly those to low density beta-lipoproteins. A precipitating antibody was identified in the serum of a haemophilic patient which reacted with an antigen which was quite dissimilar from lipoprotein antigens. This antigen was present in the serum of an Australian aborigine, and the term Australia antigen was coined (1, 2, 3). This antigen is almost certainly identical to Prince's SH-antigen (4), and to avoid confusion the term hepatitis-associated antigen (HAA) is now considered by many to be more appropriate (5).

It was some time before it became apparent that the Australia antigen was closely related to, or might be located directly on, a hepatitis virus. It was found in the serum of some 30 per cent of patients institutionalised because of Down's syndrome (mongolism) but did not occur in out-patients with this disease or in hospitalised patients with other causes of mental retardation. In these institutionalised mongoloid patients antigenaemia was associated with chronic anicteric hepatitis, as shown by liver biopsy and elevated S.G.P.T. levels (6). The antigen was isolated by density-gradient sedimentation, and under the electron microscope was found to be a particle of 200 A° in diameter with knob-like subunits of some 30 A° on the surface. Agglutination of these particles occurred on addition of anti-HAA antiserum. The liver cells of patients whose blood contained the antigen was then shown to have striking fluorescent granules in or on their nuclei when fluorescent anti-HAA was added (7), a phenomenon not seen in patients without hepatitis or in those who do not have the antigen in their blood (3). Clinical hepatitis was then found to result in a high proportion of patients given transfusions of blood from donors who carried the Australia antigen (8). Epidemiological studies suggested that the antigen was an infective agent, and supported the conclusion that the Australia antigen was intimately associated with a virus that caused hepatitis. Subsequent studies suggested that the antigen was associated with clinically different forms of hepatitis, the form depending on the immune status of the patient (9). The antigen was also shown

to be rare in North America and the United Kingdom (0.1 per cent) but was common in people living in the tropics (2-20 per cent). Indeed, these patients seemed to be susceptible to chronic infection with the hepatitis virus (10), a point that will be more fully discussed later.

#### THE NATURE OF THE ANTIGEN

The Australia antigen was shown by the Blumberg group to differ from the serum beta-lipoproteins which they had previously described in that it had a high molecular weight and contained proteins with minimal amounts of lipid. Electron microscopic studies of antigen-positive serum, prepared by sucrose density-gradient centrifugation and a negative-staining technique, revealed virus-like particles (11). These particles were about 200 Å in diameter with knob-like subunits of 30 Å diameter. Some had a central core while others were elongated their length varying from 500 Å to 2 300 Å. The addition of specific rabbit antiserum to the Australia antigen resulted in agglutination of the particles. The unusual degree of pleomorphism was the only feature incompatible with firm acceptance of the particles as viral in origin. Nucleic acid has, however, never been demonstrated in these particles, another somewhat unusual feature, although it is possible that the virus contains little or no nucleic acid. A wide variety of particles can be detected in serum by electron microscopy and Australia antigen particles are so pleomorphic that they may only be recognised with confidence in the presence of tubular forms (12). Small antigen particles seem to predominate in acute hepatitis, while large particles and tubular forms occur in carriers of the antigen, or in patients with chronic hepatitis. Certainly, there is now wide acceptance that the presence of particles of 22 Å, with little substructure, and those of 42 Å with a dense outer coat about 7 Å across and an inner body of 28 Å in association with HAA, is indicative of infection with the S.H. virus. Since the specific antibody to Australia antigen will cause clumping of both these particles (12) it is now generally agreed that the particles themselves constitute the antigen (14).

Almeida and Waterson (13) have described three patterns of Australia antigen as observed under the electron microscope in relation to clinical features.

1. In the patient with fulminant hepatitis, large amounts of antibody separate the aggregated particles;

2. in the patient with chronic active hepatitis, unattached particles of antigen were seen, together with immune complexes with antigen excess; and
3. in symptom-free carriers of the antigen, randomly distributed spherical and tubular particles were observed without clumping by antibody.

It has been suggested by Dane et al (1970) that the large (42 Å) particles are the virus itself, while the tubular forms are surplus virus coated material (15). These particles have some features in common with arboviruses. It has, however, been variously suggested that the virus associated with the Australia antigen may be a variant of the cowpea-chlorotic virus, or a member of the picornavirus family, which cause duck hepatitis. Almeida and her colleagues have further suggested that the Australia antigen may be an aggregation of protein subunits derived from the virus (13, 16). Zuckerman, working with Almeida, has described particles found on electron microscopy that displayed the characteristics of the coronavirus group, and suggested that the particles appeared to consist of antigen-antibody complexes (17, 18). More recently this group have reported the apparently successful propagation of the serum hepatitis virus in human embryo liver organ cultures, confirming their results on electron microscopy. Material harvested from these cultures has been successfully passaged, so that the full elucidation of the virus, and the antigen with which it is inextricably associated, may now not be far away (19).

#### DETECTION OF THE ANTIGEN

The earlier workers elicited the presence of the Australia antigen in serum using the micro-Ouchterlony immunodiffusion technique (1). Since then, more sensitive methods have become available. These include complement fixation, immunoelectrophoresis, haemagglutination, haemagglutination-inhibition, radio-immunoassay, and electron microscopy using negative-staining techniques.

The immunodiffusion method is simple to perform, but not sensitive. Complement fixation is more sensitive, and is amenable to quantitative determination of both Australia antigen and specific antibody. Immunoelectrophoresis is a rapid and comparatively simple technique which is less sensitive than complement fixation but more sensitive than immunodiffusion. Haemagglutination and haemagglutination-inhibition are highly sensitive for detection of antibody to the antigen.

Electron microscopy is relatively elaborate to perform, but is a rapid and sensitive method. Radio-immunoassay is about twice as sensitive as complement fixation, while immunofluorescence is amenable to detection of the Australia antigen in liver biopsy specimens. The more recently developed methods include an immune adherence-haemagglutination technique which lends itself to automation, and a latex agglutination test which is simple and rapid to perform, and sensitive.

The Australia antigen has been found, not only in serum, but in liver biopsy specimens, where it can sometimes be demonstrated by immunofluorescence when negative in the serum. Faecal antigens have also been described (20, 28) while urinary spread seems probable, for chronic carriers of the antigen may excrete it in their urine. The antigen has also been detected in bile, and may well be present in saliva, as serum hepatitis can apparently be spread by kissing.

#### PREVALENCE OF THE ANTIGEN

Community studies have shown that the Australia antigen is rarely found in the healthy population of North America and the United Kingdom (0.1 per cent), and is exceptionally rare here in those under 18. In tropical countries, however, the antigen is found far more frequently in the serum of apparently normal people. Figures quoted by Blumberg (3) for these countries include Costa Ricans (2 per cent), Australian aborigines (2.1 per cent), Brazilians (2.5 per cent), Melanians (3.6 per cent) Filipinos (4.8 per cent), Vietnamese (6.3 per cent), Micronesians (7.2 per cent), Ghanaians (9.5 per cent), Taiwanese 13 per cent and Peruvian Indians (20.2 per cent).

The Australia antigen is also commonly found in patients with acute myeloid leukaemia, and with acute and chronic lymphatic leukaemia. Most of these patients, however, have received blood transfusions as part of their therapy, and few have hepatitis. Patients with lepromatous leprosy also have an unusually high incidence of positivity in relation to their fellows in the community and in comparison with patients with tuberculoid leprosy. This is presumably due to their altered cellular immunity, favouring persistence of the antigen, together with a high incidence of antigen in the community.

Patients on chronic renal haemodialysis programmes, who also have altered cellular immunity due to their uraemia, and may in addition be receiving immunosuppressive therapy, have an usually high incidence of positivity in

comparison with other hospitalised patients often becoming chronic carriers of the antigen after incidents of mild hepatitis.

High carriage rates have more recently been reported in "mainline" drug addicts in Europe and America, figures of 7 per cent being recorded in one recent survey.

The association between Australia antigen and Down's syndrome has long been recognised, and was reported by Blumberg in 1967 (2). Figures of between 27.7 and 35.1 per cent have been variously reported for mongoloid patients in institutions. Many of these patients have histological evidence of chronic active hepatitis, and their age at time of exposure seems to determine long-term antigen carriage. The chronic carriage of the antigen in these patients may also be related to immunological deficiency.

#### SERUM HEPATITIS

Although Blumberg and his workers recognised that the Australia antigen was intimately associated with a virus causing hepatitis and that its presence in serum was indicative of the presence of that virus (21), it was not until later that the clear association of the Australia antigen with serum or long incubation period hepatitis was shown.

Prince (22) reported a close association between the Australia antigen and long incubation period hepatitis in 1968, calling the antigen SH or serum hepatitis antigen. It is identical with the Australia antigen and is absent from the serum of patients with common source outbreaks of short incubation period infectious hepatitis. The finding of Australia antigen in patients with presumed infectious hepatitis has been reported and is due to the diagnosis being made solely on clinical grounds.

Unequivocal evidence for the presence of two quite distinctive clinical, epidemiological and immunological types of viral hepatitis was provided by the work of the Krugman group at Willowbrook State School for mentally retarded children in Staten Island, New York (23, 24). Whatever one may think of the ethics of these experiments — and they certainly provoked a world-wide outcry — the work was brilliantly conceived and methodically and scientifically done. By infecting these children shortly after their admission to the school with serum from patients known to have infective and serum hepatitis and taken from these patients shortly before the development of jaundice a considerable amount of clinical, bio-

chemical, and other data was collected. The trials were done with the acquiescence of the children's parents and were defended on the grounds that viral hepatitis was endemic at the school and that the children, most of whom could not be toilet trained, would almost inevitably acquire hepatitis by the faecal-oral route while resident in the school. The experiments were condoned and sanctioned by committees set up in America to monitor human experimentation, and were conducted in accordance with the World Medical Association's Draft Code of Ethics on human experimentation.

The data accumulated during those studies has contributed immeasurably to the understanding of viral hepatitis. Two quite distinctive varieties of infectious hepatitis were shown to exist. One type resembled classical infectious hepatitis (IH) and was characterised by an incubation period of 30-38 days, a relatively short period of liver function test abnormality and a high degree of infectivity. The second type resembled serum hepatitis (SH) and was characterised by a longer incubation period, a more protracted and severe clinical and biochemical upset and moderate infectivity. On re-infecting the patients it was shown that one attack of IH protected the child from a second and that patients who had been given the SH type were in no way immune from the IH variety of infection. They subsequently showed also that IH could be transmitted parenterally as well as orally and SH orally as well as parenterally. The mode of transmission did not affect the incubation period of the IH virus, this being essentially the same after oral and parenteral exposure. The incubation period for the SH virus however was longer following oral than parenteral inoculation. They also showed that gammaglobulin protected against the infectivity of IH but not against that of SH serum.

The discovery of the Australia antigen allowed the Krugman group then to extend their studies using the antigen as a marker. They tested 25,000 specimens of serum, collected and stored during their earlier experiments on 700 patients with viral hepatitis, for the Australia antigen. This showed that the Australia antigen was consistently present in sera from patients with long incubation period hepatitis (SH) but was not present in sera from patients with short incubation period infective hepatitis (IH). Moreover, the antigen was detected earlier after a parenteral exposure to SH than to oral exposure, appearing

jaundice. The antigen was also found to be transient in 65 per cent of the children given SH, lasting a mean of 49 days. It persisted for many months, however, in the remaining 35 per cent of children. In addition, the children given SH infection were immune following re-exposure to SH virus one year later. Their observations amply confirmed that the Australia (hepatitis-associated) antigen was specifically related to serum and not to infective hepatitis and showed that if the antigen was present for more than four months it was likely to persist indefinitely. They also showed that serum containing the antigen and obtained from patients who had never had overt hepatitis, was capable of causing serum hepatitis. Indeed, it has been shown that less than 0.001 ml. of serum containing the Australia antigen was infectious (25) although the severity of the resultant hepatitis may depend on the dose of the antigen (26). Barker et al (1970) have also shown that the transmission of serum hepatitis was associated with the administration of an HAA-positive plasma pool containing virus-like particles of approximately 20 A° diameter. This is further strong support for the hypothesis that the Australia antigen (HAA) is an integral part of the serum hepatitis virus.

## SEQUELAE OF INFECTION WITH THE AUSTRALIA ANTIGEN

### (1) Sources of Infection

In the clinical context, the classical mode of infection with the serum hepatitis virus and the Australia antigen is parenteral by the transfusion of infected blood or blood products, or by contaminated equipment, especially needles or syringes.

The increasing incidence of drug abuse exposes larger numbers of the community to potential syringe-borne infection. Close contacts with addicts, who are not themselves "mainliners" can contract the disease. It is probable on clinical grounds that kissing and sexual contact with antigen positive persons may well lead to serum hepatitis. It has, of course, also been shown in the Willowbrook experiments that the disease may be spread by the faecal-oral route. Hospital-acquired disease, for example in uraemic patients on chronic renal dialysis, may also spread to the community. Indeed, cases have been reported where the spouse of such a patient has contracted a fatal serum hepatitis illness.

The importance of mass screening of blood donors is apparent in this context, especially

as the antigen has been shown to have persisted over 20 years in a patient who has remained apparently entirely well over that period (27). Shaving brushes, razors, toothbrushes, hairdressing implements, dental instruments and tattoo needles contaminated with Australia antigen positive blood have all been implicated in the spread of serum hepatitis.

The identification of the Australia antigen in faeces (28) and urine and bile creates further obvious possible sources for the spread of the disease.

## (2) Consequences of Infection

This depends on the dose, virulence of the strain, previous exposure to the antigen, and the immune status of the individual. In the latter context, depression of cellular immunity is particularly important. The patient who is immunocompetent is likely to get severe or fulminant hepatitis, while the patient who is immunodepressed by disease or iatrogenically is likely to have mild hepatitis. The former patient, should he survive, will probably rid himself of the antigen within two months of the onset of clinical hepatitis. The immunodepressed or immunosuppressed patient is, however, likely to have mild hepatitis with persistence of the antigen, often indefinitely. Some of this group will become apparently healthy long-term carriers of the antigen, while others may develop chronic active hepatitis that may proceed to cirrhosis. This has been well shown in the outbreaks of serum hepatitis in renal dialysis units. The patients tended to have mild hepatitis followed by persistent antigenaemia while the staff had severe and often fatal hepatitis, but cleared the antigen from their blood.

The Australia antigen is detectable in the serum some weeks before there is subjective or clinical illness and may be detected in the liver after recovery, when the patient is seronegative. Some antigen positive patients show allergic manifestations during the late incubation period or early in the phase of active hepatitis with urticaria, arthralgia, angioneurotic oedema and sometimes migrainous headaches. This may be related to the presence of circulating immune complexes of Australia antigen, its antibody, and complement.

The antigen becomes demonstrable in the hepatic parenchymal cells at this stage and can be nicely shown by immunofluorescent or EM studies. The patient may then develop the fulminant picture, which is similar to that of acute massive necrosis from any cause. There is a high mortality rate in this group despite

intensive therapy with colomycin, lactulose, parenteral glucose, heparin and fresh frozen plasma followed by extracorporeal perfusion, or exchange transfusion. More often, however, the patient will recover after a protracted period of cholestatic jaundice. Most who are immunocompetent will clear the antigen within four to ten weeks (29). These are the group, however, who run the risk of destroying their liver, presumably by a vigorous antigen-antibody reaction. Many will have no lasting hepatic damage, although some may proceed to chronic hepatitis of the mild persistent or even the severe aggressive varieties. Chronic persistent hepatitis often follows the classical attack of acute serum hepatitis, leading to cirrhosis, while chronic aggressive hepatitis tends to follow a mild or subclinical attack of antigen positive hepatitis. Certainly, cirrhosis is most likely when the Australia antigen remains positive after the acute attack. This persistent antigenaemia is probably related to immunological defects, especially in cellular immunity. It is at least possible that the SH virus may gain in virulence by passage through patients who are immunosuppressed either by uraemia or by azathioprine (Imuran), cyclophosphamide and antilymphocytic serum, causing very severe or fulminant hepatitis when an immunocompetent person is infected. This is, however, conjectural.

## CELLULAR IMMUNITY AND THE ANTIGEN

It has been postulated that a complex series of interactions between the Australia antigen, the serum hepatitis virus and the immune response of the host, both cellular and humoral, follows infection with HAA positive material although immune complexes of HAA and antibody are important in producing some of the varied clinical manifestations that follow infection, the cellular immune response seems to determine the severity and persistence of the associated liver damage. It has recently been suggested that the competence of the cell mediated (T-lymphocyte dependent) immune system determines whether the infection is self-limiting or persists with varying degrees of damage (30).

Krugman and his associates showed that inoculation of children with standard preparations of Australia antigen-positive serum resulted in a spectrum of clinical outcomes (24). Although transient antigenaemia was usual in association with either overt or anicteric hepatitis, persistence of the antigen could occur in association with either chronic active hepatitis,

or without apparent disease. The actual course is dependent on many variables, including genetic predisposition, immune competence, the virulence of the virus, its dose and mode of transmission. The host response to the infective agent is, however, probably all-important in determining the clinical course followed by the individual patient.

Although the presence of immune complexes of HAA correlate poorly with the degree of liver damage, deposition of these complexes may play a role in the development of the serum-sickness like syndromes which sometimes precede serum hepatitis. Only a mild persistent hepatitis is seen in the presence of complexes in HAA positive polyarteritis, while acute and chronic hepatitis can occur in patients with agammaglobulinaemia. Immune complex disease is therefore unlikely to be the mechanism by which HAA causes liver cell damage (30).

It is more probable that liver cell necrosis is related to the cellular immune response of the host to the infecting agent. This response is controlled by thymus-dependent lymphocytes, and variations in their function could determine the clinical course that follows infection with HAA positive material. This is supported by the fact that impairment of the cellular immune systems results in mild hepatitis after HAA infection — with persistence of the antigen. People who are immunologically normal, however, develop severe hepatitis when infected, but clear the antigen rapidly from their blood (3). Persistence of the antigen is seen frequently in patients with impaired T-lymphocyte function (lymphoproliferative disorders, lepromatous leprosy, chronic lymphatic leukaemia). The high frequency of antigen in mongolism may also be related to an abnormal immunological status.

Dudley et al (30) have suggested that the Australia antigen, on gaining entry to the body, comes into contact with susceptible cells — possibly in the liver. Here it proliferates, producing further infective particles plus excess virus coat material, both of which are released from the cell without having caused cell necrosis. During transit out of the liver cell antigens specific for the infective agent become incorporated in the surface membrane of infected cells. The circulating foreign antigens are then recognised by immunocompetent T-lymphocytes which proliferate to produce a number of sensitised lymphocytes. These then recognise and react with the antigens

on the surface of infected liver cells. This lymphocyte/antigen interaction results in destruction of the infective agent, and necrosis of the liver cell. Modification of this pattern can explain the various forms of Australia antigen positive liver disease.

It follows from this postulate that, in the presence of a normal immune response the patient's T-lymphocytes will react in this manner, leading to extensive liver cell necrosis — but with clearance of the antigen, i.e. the infective agent. If all the liver cells are involved, severe fulminant hepatitis will result. If few cells are infected, the hepatitis will be mild or subclinical. The outcome, therefore, with normal immunological status, is fulminant hepatitis, or complete recovery. Conversely, if the patient's immunological state is abnormal, little or no liver damage will result, but the patient would become a healthy antigen-positive carrier. An intermediate course would likewise result in a mild hepatitis followed by continuing liver cell damage and persistent antigenaemia (30). It follows from this hypothesis that immunosuppression will modify the clinical course after HAA infection the therapeutic possibilities of this being offset by the likelihood of producing persistent antigenaemia.

#### CYTOPLASMIC LOCALISATION OF THE ANTIGEN IN LIVER

Recently, immunofluorescent and electron microscopic techniques have demonstrated the Australia antigen in the cytoplasm of hepatocytes. Haynes et al (14) have shown hepatocytes in antigen sero-positive patients to contain characteristic particles with membrane bound cytoplasmic vesicles. The appearances of these particles was similar to that of the Australia antigen particles found in the serum. Two sizes of cytoplasmic particles were observed, with average diameters of 26 and 46 Å. Particles of both sizes often had a membrane-like outer component and a moderately electron-dense inner component. They differed in both size and structure from the mainly intranuclear particles described by previous authors (31).

Although a direct connection between the two types of particles described above has yet to be established, it seems likely that the different types are related to stages in the development of the serum hepatitis virus. It has been shown that there is apparent intranuclear replication of 22 Å particles, with considerable disruption of the nucleus, and sometimes also with cytoplasmic replication, occasionally



with the production of rather different particles within the cytoplasmic vesicles. Others have shown no intranuclear replication or damage, but enormous production of particles, within the cytoplasm, of the larger type — and a much greater degree of cytoplasmic damage. Whether these two situations represent different stages of a single process or are alternative manifestations of infection by the SH virus is uncertain. It seems unlikely, however, that either one could lead directly to the other. Another possibility is that they are related to the immunological status of the patient (14).

#### CHRONIC ACTIVE HEPATITIS (C.A.H.) AND CIRRHOSIS

The remarkable specificity of the Australia antigen for serum hepatitis and chronic active hepatitis in comparison to other forms of liver disease suggests that persistence of the antigen may be aetiologically important in some cases of chronic active hepatitis and cirrhosis.

Chronic active hepatitis is characterised by a prolonged course and the presence of persistent hepatic inflammation with fibrosis which may progress to cirrhosis. Some patients develop polyserositis, hypergammaglobulinaemia, and positive serum antinuclear factors and even L.E. cells. The pathogenesis of C.A.H. is unknown, but may have its origins in infection with the Australia antigen, superimposed upon primary, or complicated by secondary immunological factors (32). Although one study showed an association with HAA, several others have failed to do so. Some reports have shown progression, on liver biopsy, from serum hepatitis to cirrhosis, so that the HAA may have a role to play in some cases of C.A.H. and cirrhosis. In these cases it seemed probable that persistent viral infection, as judged by antigenaemia, has contributed to the pathogenesis of C.A.H. Other cases may conceivably be initiated by infection with the SH virus and HAA, but be perpetuated by some other mechanism, possibly auto-immune, after clearance of the antigen (12). Zuckerman et al (33) have, however, detected particles by electron microscopy (EM) in the serum of a patient with C.A.H. and cirrhosis whose serum was HAA negative, so that the apparent anomaly may lie in the sensitivity of present methods of testing for antigen.

#### PRIMARY BILIARY CIRRHOSIS AND THE ANTIGEN

Particles identical to those associated with the Australia antigen were found by EM in the sera of 11 out of 12 patients with primary biliary cirrhosis. Antigen and/or antibody to

Australia-antigen was also found in 9 out of 10 of these cases by sensitive immunological methods (34). These findings remain to be confirmed, but the suggestion is that the liver damage in primary biliary cirrhosis may be due to either continuing replication of the SH virus, or be the result of the patient's immune response to persistent antigenaemia. The presence of autoimmune (smooth muscle and anti-mitochondrial) antibodies in this condition has led to the suggestion that the condition is either due to or associated with abnormal immune reactivity, the trigger possibly being the SH virus.

#### HEPATOCELLULAR CARCINOMA AND THE ANTIGEN

There has recently been worldwide interest in the possible oncogenic properties of the SH virus and HAA. In Uganda (35) 40 per cent of patients with hepatocellular carcinoma had the Australia antigen in their blood. These workers found a tendency for HAA positive individuals with hepatocellular carcinoma to be alpha-fetoprotein positive, and to have underlying cirrhosis of the posthepatic type. Young patients tended to be HAA positive more frequently than the older ones who were often alpha-fetoprotein negative. This data certainly suggests an association between persistent antigenaemia and the pathogenesis of hepatoma — at least in Uganda, especially as only 4 of their 122 controls (3 per cent) were antigen positive. It is certainly tempting to speculate that antecedent viral hepatitis plays a causative role in the neoplastic transformation of the liver cell. Other workers have lent support to this theory, with marked differences across the globe. In Singapore 3 per cent of 114 patients with hepatomas were HAA positive, in Japan 5 per cent of 10, in India 63 per cent of 11, and in Taiwan 80 per cent of 55. Alternatively, in South Vietnam HAA was not found in any of the patients with hepatoma that were studied, but was present in 4 per cent of their controls! (36)

#### POLYARTERITIS AND AUSTRALIA ANTIGEN

A most interesting survey of 11 patients with biopsy-proven polyarteritis nodosa showed 4 of them to be Australia antigen positive. The four HAA positive patients exhibited a typical polyarteritis syndrome, but differed from the antigen negative patients in having evidence of mild hepatic damage. The presence of circulating immune complexes in the sera of 3 of the 4 anti-

gen positive patients was demonstrated by serological, ultracentrifugal, and EM studies, and were shown to be composed of Australia antigen and immunoglobulin. Immunofluorescent studies of tissue from one of the patients revealed deposition of Australia antigen, IgM and B<sub>1</sub>C in blood vessel walls. The suggestion is that the syndrome of diffuse vascular damage observed in these patients was due to deposition of immune complexes in the blood vessel walls, and that these deposits were composed of Australia antigen, homologous IgM antibody, and complement components (37).

#### THE HUMAN BEING AND HIS AUSTRALIA ANTIGEN

During the recent tragic Edinburgh serum hepatitis outbreak, the community in general and hospital staff in particular came face to face with a new and terrifying reality — inoculation with antigen positive material might herald a fatal illness. This transformed people, and created problems that are only now being fully realised as the immediate danger seems past. Patients were sometimes treated as were lepers, and many are still aware of being ostracised, despite a long-standing negative Australia antigen. Many doctors and nurses did, however, behave in a manner that does them very great credit, and signs are around that medicine, in its broadest sense, is coming to terms with the discovery that Blumberg fell upon quite by accident almost a decade ago. He could hardly have realised then that he had, in truth, perhaps created more problems in his discovery than have been solved by the now clear cut delineation of the two forms of viral hepatitis by the recognition of the Australia antigen.

---

“Knowledge comes, but wisdom lingers.”  
Lord Tennyson

#### REFERENCES

1. Blumberg, B.S., Alter, H. J. and Visnich, S. A “new” antigen in leukaemic sera. *J.A.M.A.* (1965), 191: 7: 541-6.
2. Blumberg, B.S., Gerstley, B.J.S., Hungerford, D.A., London, W.T., and Sutnick, A. I. A serum antigen (Australia antigen) in Down's syndrome, leukaemia, and hepatitis. *Annals of Int. Medicine* (1967) 66: 5: 924-31.
3. Blumberg, B.S., Sutnick, A.I., and London, W.T. Australia antigen as a hepatitis virus. *Amer. J. of Med.* (1970), 48: 1: 1-8.
4. Prince, A.M. Relation of Australia and SH-Antigens. *Lancet* (1968) 2: 462.
5. McCollum, R.W. Serum antigens in viral hepatitis. *J. Infect. Dis.* (1969), 120: 641.
6. Sutnick, A. I., London, W. T., and Blumberg, B.S. Australia antigen, Down's syndrome and hepatitis. *J. Clin. Invest.* (1967), 46: 1122.
7. Millman, I., Zavatore, V., Gerstley, B.J.S., and Blumberg, B.S. Australia antigen in the nuclei of liver cells of patients with viral hepatitis detected by the fluorescent antibody technique. *Nature* (1969) 48: 9a.
8. Blumberg, B.S., Gerstley, B.J.S., London, W.T., Millman, I., Sutnick, A.I. and Zavatore, V. Hepatitis virus and Australia antigen. *J. Clin. Invest.* (1969) 48: 9a.
9. London, W.T., Di Figlia, M., Sutnick, A. I. Ziegenfuss, J. and Blumberg, B.S. Australia antigen associated hepatitis epidemic in a haemodialysis unit. *Clin. Research* (1968), 16: 567.
10. Blumberg, B.S., Friedlaender, J.S., Woodside, A., Sutnick, A.I. and London, W.T. Hepatitis and Australia antigen: autosomal recessive inheritance of susceptibility to infection in humans. *Proc. Nat. Acad. Sci. (USA)* (1969) 62: 1108.
11. Bayer, M.E., Blumberg, B.S. and Werner, B. Particles associated with Australia antigen in the sera of patients with leukaemia, Down's syndrome and hepatitis. *Nature (Lond.)*, (1968) 218: 1057.
12. Wright, R. The Australia (hepatitis) antigen. *Brit. J. of Hosp. Med* (1970), 4: 1: 75-82.
13. Almeida, J.D. and Waterson, A.P. Immune complexes in hepatitis. *Lancet* (1969), ii: 983-6.
14. Haynes, M.E., Haynes, D.W.G. and Williams, R. Cytoplasmic particles in hepatocytes of patients with Australia antigen positive liver disease. *J. Clin. Path* (1972) 25: 403-9.
15. Dane, D.S., Cameron, C.H. and Briggs, M. Virus-like particles in serum of patients with Australia antigen associated hepatitis. *Lancet* (1970) i: 695-8.
16. Leading Article. More about the Australia antigen and hepatitis. *Lancet* (1970) 2: 347-9.
17. Zuckerman, A. J. Viral hepatitis and Australia (SH) antigen. *Nature* (1969) 223: 569-72.
18. Almeida, J.D., Zuckerman, A.I., Taylor, P.E. and Waterson, A. P. Immune electron microscopy of the Australia SH antigen. *Microbios* (1969) 1: 117-23.
19. Zuckerman, A. J., Baines, P.M. and Almeida J.D. The Australia antigen as a marker of propagation of the serum hepatitis virus in liver cultures. *Nature (Lond)* (1972) 236: 78-81.
20. Grob, P.J. and Jemelka, H. Faecal Australia-antigen hepatitis. *Lancet* (1971) 1: 206-8.
21. London, W.T., Sutnick, A.I. and Blumberg, B.S. Australia antigen and acute viral hepatitis. *Annals of Int. Med.* (1969) 70: 1: 55-59.
22. Prince, A.M. An antigen detected in the blood during the incubation period of serum hepatitis. *Proc. Nat. Acad. Sci. (USA)* (1968) 60: 814.
23. Krugman, S., Giles, J.P. and Hammond, J. Infectious hepatitis. Evidence for two distinctive clinical, epidemiological, and immunological types of infection. *J.A.M.A.* (1967) 200: 5: 365-73.

24. Krugman, S. and Giles, J.P. Viral hepatitis. New light on an old disease. J.A.M.A. (1970) 212: 6: 1019-1029.
25. Barker, L.F., Shulman, N.R. and Murray, R. Transmission of serum hepatitis. J.A.M.A. (1970) 211: 1509-12.
26. Shulman, N.R., Hirschman, R.J. and Barker, L.F. Virus hepatitis. Annals. of Int. Med. (1970) 72: 2: 257-69.
27. Zuckerman, A.J. and Taylor, P.E. Persistence of the serum hepatitis (Australia) antigen for many years. Nature (1969) 223: 81-2.
28. Ferris, A.A., Kaldor, J. and Gust, I.D. Faecal antigen in viral hepatitis. Lancet (1970) 2: 243-4.
29. Iwarson, S. and Hermodsson, S. Hepatitis-Associated Antigen (HAA) in acute viral hepatitis. Scand. J. Infect. Dis. (1971) 3: 93-101.
30. Dudley, F.J., Fox, R.A. and Sherlock, S. Cellular immunity and hepatitis associated (Australia) antigen liver disease. A hypothesis. Lancet (1972) 1: 723-6.
31. Nowoslawski, A., Brzosko, W.J., Madalikski, K. and Krawczycki, K. Cellular localisation of Australia antigen in the liver of patients with lymphoproliferative disorders. Lancet (1970) 1: 494-8.
32. Buckley, B.H., Goldfinger, S.E., Heizer, W.D. and Isselbacher, K.J. Distinctions in chronic active hepatitis based on circulating hepatitis-associated antigen. Lancet (1970) 2: 1323-6.
33. Zuckerman, A.J., Taylor, P.E. and Almeida J.D. Presence of particles other than the Australia-SH antigen in a case of chronic active hepatitis with cirrhosis. Brit. med. J. (1970) 1: 262-4.
34. Krohn, K., Finlayson, N.D.C., Jokelainen, P.T., Anderson, K.E. and Prince, A.M. Electron microscopical and immunological observations on the serum hepatitis (SH) antigen in primary biliary cirrhosis. Lancet (1970) 2: 379-83.
35. Vogel, C.L., Anthony, P.P., Mody, N. and Barker, L.F. Hepatitis-associated antigen in Ugandan patients with hepatocellular carcinoma. Lancet (1970) 2: 621-4.
36. Welsh, J. D. et al. Hepatitis-associated antigen in hepatitis in South Vietnam. Lancet (1972) 1: 592.
37. Gocke, D.J., Hsu, K., Morgan, C., Bombardieri, S., Lockshin, M. and Christian, C.L. Association between polyarteritis and Australia antigen. Lancet (1970) 2, 1149-53.

## Indispensable Books for the Student

**THE 'SYNOPSIS' SERIES — ideal for revision purposes**

<b>Anaesthesia</b> Lee and Atkinson Seventh Edition about £5.50	<b>Gastro-enterology</b> Chandler £1.60	<b>Ophthalmology</b> Martin-Doyle Fourth Edition £2.50	<b>Renal Diseases and Urology</b> Miller, Slade and Leather £2.00
<b>Biology</b> Crow Second Edition £3.00	<b>Infections and Tropical Diseases</b> Woodruff and Bell £2.60	<b>Otolaryngology</b> Simpson, Robin, Ballantyne and Groves Second Edition £3.00	<b>Respiratory Diseases</b> Smart £1.30
<b>Blood Diseases</b> Kok £2.50	<b>Medicine</b> Tidy Tenth Edition £2.00	<b>Physiology (Rendle-Short)</b> Vass Fifth Edition £1.70	<b>Rheumatic Diseases</b> Golding £1.70
<b>Cancer: Genesis and Biology</b> Kark £2.40	<b>Neurology</b> Tatlow, Ardis and Bickford £1.70	<b>Public Health and Social Medicine</b> Essex-Cater Second Edition £4.20	<b>Skin Diseases</b> Solomons £1.70
<b>Cardiology</b> Weitzman £1.70	<b>Obstetrics and Gynaecology</b> Bourne and Holmes Thirteenth Edition £3.00		<b>Surgery (Hey Groves)</b> Cotton Sixteenth Edition £3.00
<b>Children's Diseases</b> Rendle-Short and Gray Fourth Edition £3.00			<b>Surgical Anatomy</b> McGregor and Du Plessis Eleventh Edition <b>Surgical Pathology</b> Kark £2.60
<b>Endocrinology and Metabolism</b> Ferriman and Gilliland £1.70			

### John Wright

JOHN WRIGHT & SONS LTD.  
BRISTOL, ENGLAND BS8 1EX

# OLD MEDICAL BOOKS IN EDINBURGH LIBRARIES:

## THE ROYAL MEDICAL SOCIETY CATALOGUE PROJECT

Geoffrey D. Hargreaves

In the justifiable belief that the older collections of printed medical books in Edinburgh libraries are exceptional in both quality and quantity, the Royal Medical Society has initiated a scheme for the production and, it is hoped, the publication of a composite catalogue of these collections for the benefit of scholars in the history of medicine and science. The institutions currently involved in the scheme are the National Library of Scotland, the Royal Botanic Garden, the Royal College of Physicians, the Royal College of Surgeons, the Royal Observatory, the Royal Society and the University of Edinburgh (including New College). In addition, the Royal Medical Society is, of course, contributing its own residual but important collection of older books withheld from the 1969 sale as being unrepresented in any other Edinburgh collection.

The quality of the Edinburgh collections as a whole may perhaps be adequately suggested by noting their strength in the classics of medical literature. There are, for example, three copies of the *De medicina* (1478) of A. Cornelius Celsus, one of the first general medical treatises to be printed; four copies of the herbal *De historia stirpium* (1542) of Leonhard Fuchs, and three of the anatomical work *De humani corporis fabrica* (1543) of Andreas Vesalius — books which, particularly through the quality of their illustrations, did much to release the study of medical botany and anatomy from traditional inaccuracies; one of

the three known copies of the *Christianissimi restitutio* (1553) of Michael Servetus, which includes the first Western account of the lesser circulation; no fewer than five copies (including one with the errata) of what is generally regarded as the most important work in the history of medicine, William Harvey's account of the circulation in his *De motu cordis* (1628); five copies (including a presentation copy with the plate in colour) of another classic of medical botany, William Withering's *An account of the foxglove* (1785); and a copy of Sir Charles Bell's exceptionally rare *Idea of a new anatomy of the brain* (1811), a pamphlet even more unprepossessing in appearance than the *De motu cordis*, yet — in the context of the nervous system — of comparable importance as an advance in medical knowledge.

Such classics are supported by the more ephemeral (and thus often more elusive) tracts in particular pharmacological, obstetrical and dietary instruction at a more popular level. Among the earlier works in this category to be found in Edinburgh may be mentioned the *Liber servitoris de praeparatione medicinarum simplicium* (1471) attributed to Albucasis and perhaps the first printed book of a wholly medical character; Aldobrandino's *Le livre pour garder la sante du corps* (c. 1481); the *Secreta mulierum* (two editions, c. 1495-1500) attributed to Albertus Magnus; the *Regimen sanitatis* (1486 and c. 1500) of Magninus; the pseudo-Aristotelian *Problemata de partibus*

*corporis humani* (c. 1500); the *Tractato mirabile contra peste* (1503) of Baverius de Baveriis; and the *Enneas muliebris* (c. 1505) dedicated by the author, Ludovicus Bonaciolus, to Lucretia Borgia. Earlier Scottish medical printing also falls into this category, and is of course well represented with such rarities as Gilbert Skene's *Anc breve descriptionn of the pest* (1568) and Patrick Anderson's *Grana angelica* (1635).

In addition to the texts, the provenance and associations of the books often reflect the history of Scottish medicine and science in general and of the Edinburgh Medical School in particular. Some examples are the works on medical botany acquired during a continental tour in 1670 by Patrick Murray, Laird of Livingstone, whose collection of plants formed the basis of the Royal Botanic Garden; the extensive collection of medical books of the sixteenth to nineteenth centuries formed by Sir James Young Simpson; and the copy of the *De motu cordis* (1628) owned by Professor Alexander Monro tertius. But the owners of medical books have not always been medical or scientific men, especially in the period before 1700. The Edinburgh copy of the 1527 edition of Avicenna's monumental exposition of Arabic medicine, the *Canon medicinae*, is preserved in a fine binding for John Hamilton, Archbishop of St. Andrews from 1546; the Drummond Collection features several rare ephemeral medical tracts owned by the poet William Drummond of Hawthornden (1585-1649); while John Gray, minister at Aberlady in 1690, was another owner of an Edinburgh "Harvey". In these ways the Royal Medical Society's project is likely to establish links with a wide range of Scottish studies.

If the quality of the collections is inviting, their quantity is daunting. The involvement of two major general libraries makes it essential that the project should work to a fairly strict definition of a "medical" book, especially for the period after 1700. Even so, sampling techniques suggest that there are at least 50,000 "medical" books in Edinburgh printed before 1851, the terminal date set by the Royal Medical Society for an "older" book. It should therefore be clear that the project must ultimately be of a long-term or multi-staffed nature, possibly both. The initial three-year term sponsored by the Society must be essentially an exploratory effort, aimed at covering no more than a suitable early portion of the scheme in addition to assessing its long-term

requirements. At first it was hoped that this early portion might extend as far as 1600 or even 1640, but experience in attempting to co-ordinate a project spread over eight separate libraries, where the appropriate books have first to be isolated from the general stocks, has now shown that this is impracticable. The Wellcome Historical Medical Library and the United States National Library of Medicine both commenced publication of their catalogues with a separate account of their fifteenth century books. It is doubtful if Edinburgh's "medical" (as opposed to "scientific") incunabula are sufficient in number to justify separate treatment, but the addition of the "post-incunabula" (books printed up to about 1540 or perhaps 1550) would seem to offer a unit of sufficient scope for publication and of considerable subject and bibliographical integrity, covering a period when both medical knowledge and the printed book remained to a large extent at "the cradle stage". Accordingly it is here that the efforts of the project are currently concentrated.

A particularly important feature of the project is the presence in Edinburgh of more than one copy of many of the books involved. The ravages of time and readers, coupled with the technical vagaries of book production, have ensured that many copies of early books (and indeed some copies of more recent books) are physically incomplete and textually imperfect. In these circumstances, the complete copy often has to be reconstructed from an examination of several individual copies, all of which may be more or less defective in themselves. In many cases, therefore, the Edinburgh collections can make a significant contribution to this process, and if the reconstruction is recorded in sufficient detail to indicate the full contents and make-up of a complete copy, the resultant description can be of great value to scholars, who may, for instance, find themselves reading what appears to be an incomplete copy and wish to ascertain how much is missing.

There are other ways in which a fairly detailed description can be of value to scholars in the history of medicine and science. They may be interested in the popularity and success of a work, and a useful indication of these is available in the number of separate editions called for, as the printer would not normally go to the trouble and expense of resetting type for a new edition unless the work was in demand. Distinct identification of editions is

therefore required, and since they may be very closely related and (especially in the case of popular works) undated or of the same date, detailed description is often necessary if the distinguishing features are to emerge. Again, scholars may be particularly interested in printers who specialised in medical and scientific printing, such as Bonetus Locatellus (fl. 1500) of Venice and Heinrich Sybold (fl. 1530) of Strassburg, who was himself a Doctor of Medicine as well as a printer. But Locatellus and Sybold, like many other printers before and after them, did not always indicate clearly in their books that they were the printers of them, and indeed many books carry no clear indication of printer, place or date. In these cases, it is desirable to present the identifying evidence of such features as printers' types and devices (this can also assist in the identification of editions), or at least to provide references to existing treatment of the problem.

For the incunabula and post-incunabula at least, some attempt has been made to take these considerations into account in the descriptive method currently being employed for the main entries, of which an outline is appended to this survey. These entries inevitably represent a compromise between the fullest standards of bibliographical description, as reached for instance by Allan Stevenson in the Hunt Catalogue of eighteenth-century botanical books, and simplified listing, which denies to historians and bibliographers alike so much of the information that they require. Even so, eventual publication may have to be confined to a selection of the information compiled, but it seems important to base this information as widely as possible in the first instance so that the largest possible number of options are kept open for the final product. This procedure also allows for the provision of full added entries and indexes of subsidiary works and persons, including editors, translators, illustrators, dedicatees, printers and publishers.

Finally, it is appropriate to emphasize that progress of any kind could hardly be made without the existing resources and organization of the participating libraries for the exploitation and retrieval of their collections in general. Many of these are covered by invaluable printed catalogues, and all individual items are readily available to the visiting student. It is also fitting to acknowledge the kind co-operation of the authorities of the participating libraries, and a pleasure to thank their libra-

rians and the many members of staff who, despite the pressure of their normal duties, are generously providing both practical help and technical advice.<sup>1</sup>

1. Thanks are also due for their help and support to Dr. Gweneth Whitteridge, Professor William Beattie, Dr. H. A. Feisenberger and Mr. R. O. MacKenna.

## APPENDIX

### *Descriptive formula (main-entry) for incunabula and post-incunabula.*

*Heading.* Normally includes author, short title, imprint (translated and given an arabic numeral date where necessary) and format. The form of the author's name follows the usage of the Edinburgh libraries themselves as far as possible; "established" usage is taken as the co-ordinating feature.

*Transcription.* In this period the information normally found on the title-pages of later books tends to be scattered throughout the book on title-pages (if provided) and in headings, incipits, explicits and colophons. Information is accordingly transcribed from these features, retaining the spelling, capitalization, punctuation and line-endings of the originals. The amount of information transcribed varies with each book in a flexible formula, the aim being, as far as possible, to let the book speak for itself in support of the heading and in respect of the subject interest. Title-pages and colophons are generally transcribed in full, but no attempt is being made to rival (or duplicate) the intensive descriptions already available for certain incunabula.

*Collation.* Given by signatures (Greg-Bowers formula) to indicate the precise make-up of a complete copy and provide reliable reference notation in preference to foliation or pagination, which are either not present or are very erratic at this period. If present, foliation or pagination is noted after the signatures in a simplified formula. (In a few very early books, as in many modern books, the gatherings are unsigned; this does not prevent the construction of a formula to indicate make-up, but reference in these cases is by supplied true foliation.)

*Typography, illustration, contents.* The number of lines to a (typical) page and the standard of measurement of twenty lines of type on that page are given to aid identification (of the edition and/or printer) and also visualization. There follows a note of any illustrative features (including printers' devices); "full-page" woodcuts are distinguished from those "in text". A simplified contents note seeks in particular to bring out any features (works, persons) of a complete copy not clarified in the transcription. Brief discussion of authorship and attribution problems may be added if necessary.

*References.* Normally given to sources which either treat the book in a bibliographically useful way, locate other copies of it, or provide notes on its subject matter and importance. Followed as required by notes on bibliographical problems.

*Copy or Copies.* Location of the Edinburgh copy or copies examined, with notes on individual variation, defect, or special features (association, annotation, binding, etc.) as appropriate. (It is important to emphasize that apparent "duplicates" in Edinburgh are rarely proving, on close examination, to duplicate each other in every significant feature).

# THE ROYAL MEDICAL SOCIETY TRAVEL FUND

---

October 1972

All Medical Under-Graduates and Pre-Registration Doctors are eligible to receive awards from the R.M.S. Travel Fund, to finance travel for the furtherance of their medical education. The amount of each individual award will vary with the merit of the project to be undertaken, but will not exceed £150 in any one instance.

Applications should be made by 10th February 1973, for projects up to 1st October 1973; and 10th September 1973, for projects up to 1st March 1974. They should be sent in writing, giving details of the proposed visits to :

THE SECRETARY,  
ROYAL MEDICAL SOCIETY TRAVEL FUND,  
3 HILL SQUARE, EDINBURGH EH8 9DR

# THE DOCTOR AND THE ELDERLY

James Williamson

Specialism in medicine is an inevitable accompaniment of modern life and one of the newest specialties is Geriatrics (or as many of us would prefer — geriatric medicine). Medical specialties develop for a variety of reasons, for example, the specialty of renal diseases has grown out of the great technical advances in diagnosis and treatment in recent years. Other specialties have developed more gradually as the total body of relevant knowledge has accumulated, for example, cardiology and neurology. Other specialties have appeared as a direct response to the needs of the community, and this is the category into which we place geriatric medicine. With the increasing numbers of old people consequent upon the much higher proportion who survive to old age, the needs of the elderly have escalated and it has been necessary to attempt to deal with this crisis in different and sometimes novel ways. At the same time as numbers of old people have been increasing, many of the traditional family patterns of care have been eroded by social alterations, and so we have both demographic and social reasons for a “geriatric crisis”. It is commonplace now to find that the married daughter (in her 30’s or 40’s) is unable to afford her mother more than token support because she herself is in paid employment and only available for housewifely and family tasks in the evening.

The result has been a great increase in declared demand for services for the elderly. Nor is this the whole story, because many studies in different areas of the Western World have shown that old people’s needs are often unknown until a crisis occurs (a fall, an acute infection, a stroke, or sometimes simply illness of a custodial relative or neighbour) and the situation is then found to be very advanced and perhaps irreversible and preventable sequelae have occurred.

It is against this background of increasing demand, unreported need and limited facilities that we have to try to take stock and assess the present situation. It is often suggested that these are “mainly social” problems and therefore the doctor’s role is a limited one. But the profession cannot wriggle off this particular hook so easily because when we examine these “social” difficulties many turn out to have an important (even exclusively) medical basis. Thus we find the old lady who lives in the top flat in a tenement and is becoming more and more disabled, unable to climb stairs and thus shopping becomes perfunctory, her social life and diet suffer, and she is in danger of loneliness, isolation and all the sequelae of such a state. A social problem? But what if the reasons for her increasing disability is dyspnoea associated with congestive cardiac failure and this is due to a rapid, unknown, uncontrolled



atrial fibrillation. This now becomes a medical problem, readily amenable to standard therapeutic measures with excellent results (and at almost negligible cost to the N.H.S. and the community). I have an old patient who presented in such a state eleven years ago and who still lives on in her old family flat and has done since the diagnosis and appropriate therapy was instituted. Unfortunately not all the problems of elderly patients are quite so simple and straightforward and the majority present with multiple pathology. In many cases traditional medical measures are only partially successful, and we are left with significant residual disability. In these cases modern rehabilitation techniques must be employed — assessing the functional deficit in stroke cases, for example, and using physiotherapy and occupational therapy to restore lost function. Where irrecoverable disability remains, then the patient's environment must be altered to suit. Thus family or neighbourly support can be mobilised. Home helps can take over household management tasks and the house itself can be adapted to suit the old person's disabled condition (provision of ramps, handrails, wider doors in W.C., etc.). In this way the doctor's skill is employed to establish an accurate clinical diagnosis; traditional therapies are then employed to cure or alleviate those disease entities which are uncovered, and the doctor then has to invoke the rehabilitation team to restore lost function or to make up for the loss when full restoration is not feasible.

Geriatric medicine can claim great success in many fields over the last twenty years or so. More and more it is realised that early diagnosis and appropriate treatment will enable an increasing number of old people to remain longer in their own homes, thus staying out of institutions and costing the rest of us less in terms of money and trained staff to look after them. Despite increased efficiency however, the increasing numbers of old people in many areas continue to swamp the community services, and in this case the old people "overflow" into hospital beds where they often are seriously misplaced. Thus every general ward in Edinburgh has its quota of "blocked beds" occupied by old ladies who should be somewhere else, for example, in sheltered housing (which virtually is non-existent in Edinburgh) or in old folk's homes (which themselves are occupied to a large degree by old people who could be in sheltered housing if this existed). These old ladies are often pathetic creatures —

disabled, frequently demented, bereft of family support and guiltily aware that they are not very welcome in the "acute" ward situation in which they find themselves. They are led to understand that they are "blocking a bed", that the bed "is needed for a more urgent case", etc. This leads to frustrations among the medical and nursing staff, and if there are students in the ward, this is communicated to them. The student thus gains the idea that somehow old people are "uninteresting", that they are "therapeutically unrewarding" and that their care scarcely impinges upon the activities of doctors (or at any rate "proper" doctors). It is not all surprising therefore that the young graduate often emerges from the Edinburgh Medical School with a highly negative view of his role in relation to elderly patients. This may last him a lifetime, although fortunately many manage to achieve a more useful attitude towards their older patients as they go through their professional career. If anyone doubts the accuracy or truth of this statement, let him ask a representative sample of final phase students or young graduates what are their views of the care of elderly patients, or the function of the geriatric services. I know what the response will be because I have asked these questions.

Another sad fact is that the student's contact with old age in his curriculum is almost exclusively in the wards of the teaching hospital and he remains largely ignorant of the huge majority of other old people. The importance of this is emphasised by the fact that 94% of elderly persons are in private households and only 6% in any form of institution. Of this 6%, only a tiny fraction are to be found in teaching wards, most being in old folk's homes or old-fashioned psychiatric hospitals, well away from the medical student's accustomed territory.

The result of this is that many students manage to become doctors with only a very incomplete and highly distorted view of the ageing process and the common problems which beset the older members of the communities he will be attempting to serve. This is particularly serious in view of the fact that these doctors (if they are to practise in a developed country) will be spending an increasing proportion of their time with elderly patients.

## THE FUTURE OF GERIATRIC MEDICINE

It has been pointed out that the specialty of geriatric medicine arose initially out of the pressing community need. This declared need,

combined with a general lack of interest and unwillingness on the part of most doctors, led to the setting up of special departments and a small number of pioneers in the profession showed what could be done to help the apparently "hopeless cases" of old people with multiple and complex needs. Gradually geriatric hospital units have evolved and now there is some sort of service for each area of the Kingdom. It is, however, readily seen that there are stresses and strains, and it is necessary to review the present situation and to try to devise more satisfactory plans for the future. There is no denying that geriatric units are difficult to staff — there are well over thirty vacancies at consultant level in England and Wales alone, and it is commonplace for posts to be advertised over and over again with failure to obtain satisfactory applicants.

It is therefore proposed by many responsible geriatric physicians that the present arrangements should be reviewed and that the recent trend for separation of geriatric medicine from general medicine should be reversed. This idea has much to recommend it, and some of us who initially were not too enthusiastic are now prepared to change our stance on this issue. After all, Geriatrics is defined as "that branch of general medicine which deals with the clinical, social and psychological problems of elderly persons", so that it is an integral and increasingly important part of general medicine. Indeed, some would go further and claim that geriatric medicine is by far the largest part of general medicine since so much else of the general field has been pirated by narrower specialisms. It would thus seem likely that we should encourage geriatric units to come closer to general departments, to share junior staff (either in joint appointments or in rotational schemes), to share expensive diagnostic and treatment facilities. Thus we might envisage the medical division of the future having as one of its constituents the consultant in Geriatrics who would deal with the "purely geriatric" cases and at the same time be available for advice on other elderly patients who were receiving treatment from other specialisms in the division. This "loose specialism" could extend outside the hospital into general practice where some general practitioners would be encouraged to develop special interests in the diagnosis and management of elderly patients. These specially interested general practitioners would be those who had spent some time in the geriatric department and many of them would retain an

active role in the local geriatric unit. This general plan would reverse the recent drift towards establishing a separate service for older patients — in my view such a trend would result eventually in two standards of service — a good and a bad — and there is little doubt which would be which! There will, of course, always be a need for specialist geriatric units, especially in large centres where large numbers of very difficult cases will occur. In addition there is a great need to encourage the establishment of academic departments in geriatric medicine, and it is difficult to escape the conclusion that until a medical school has a Department of Geriatric Medicine it cannot be said to be matching up to the demands of the late twentieth century. Ten years ago it could with justification be argued that the total body of knowledge on ageing and geriatrics was so scanty and disorganised that it could not justify being described as an academic discipline. The great advances in recent years in the practice and theory of medicine in relation to old age and the success of research has changed this and there are now as good arguments for academic departments in this subject as there were twenty or thirty years ago for academic departments of Paediatrics (or more properly Child Health).

These things, of course, cannot be done at the stroke of a pen, and the first essential is to provide undergraduate instruction in geriatric medicine. The Edinburgh Medical School is well-known in Scotland for having been extremely slow to develop such instruction — indeed, it only started on any scale in January, 1972! Even now the time allocated — two sessions in fourth year and the same in final phase — is so brief that it is quite difficult to know where to start.

It is therefore suggested that the following should be aimed at:

## PRE-CLINICAL YEARS

The student should have an introduction to age topics immediately he enters medical school. The biology of ageing is a fascinating and rapidly growing subject and should be taught in the biology class.

Similarly, physiological changes of senescence should be taught in the physiology and anatomy courses. Visits to geriatric units should be arranged at this stage so students can see the significance of what they are being taught in a clinical setting, for example, the ageing of bone tissue could be demonstrated in a dra-

matic fashion by showing students the skeletal changes in elderly patients.

Psychological aspects of ageing require to be taught in the behavioural sciences class, and again visits to geriatric departments should be arranged in collaboration with consultants in geriatric medicine.

In the studies on community health, the place of the elderly and the stresses and strains they experience (and generate) would be included.

In this way the student would be made to realise from the start that senescence is something that will affect him personally and that a large part of his professional life will be spent on dealing with its effects. If we believe that "normal" life span is three score years and ten, then we must accept that senescence is a normal part of human development and hence justifies study just as much as embryology, infancy, childhood and adolescence. At present our attitude to human development tends to suggest that once we reach maturity, nothing changes until death!

## CLINICAL YEARS

In the teaching of clinical methods there are great advantages to be had in bringing students to the geriatric department: a) there is a wealth of "clinical material" — heart murmurs, palpable masses, skeletal changes, malignant disease, cataracts, etc.; b) it is important that the student should have an opportunity to realise the special problems of clinical examination and history taking in the elderly, c) the distinction between "normal" age changes and pathological ones. Here attempts should be made to show students healthy octo- and nonagenarians in order to counteract the dismal impression he obtains of old age in the wards.

For the teaching of geriatric practice we need a good deal of experiment with different methods. If lectures are an important part of the course, then there should be lectures on geriatrics. But it is not, I think, a subject which lends itself readily to treatment in large formal lectures. It is better to develop topic teaching by concentrating upon specially important aspects. A good example is Stroke, which should be taught by a multidisciplinary approach — pathology, epidemiology, diagnostic procedures, management of "acute" phase, and finally assessment of functional loss and planning of rehabilitation. The special contribution of the geriatric teacher would be in the last two items, but there is much to be said for involving other specialists in the same teaching session, (And, of course, physiotherapists, speech therapists and nurses as well). There is a great scope for developing better teaching methods in these fields.

Lastly, some students should be encouraged to do "in depth" studies of ageing problems. The field of gerontology and geriatrics bristles with opportunities for special enquiry, and a keen young student could even at undergraduate level readily provide new evidence on old problems which might lead to better ideas on management. Much of this could be done in the community, away from the restrictions of hospital-bound medicine, and this in itself would be valuable (and also in line with much current student aspiration).

In these ways I believe it is possible to bring geriatrics back into the mainstream of general medicine before the separation has gone too far. Psychiatry drifted away from general medicine in the 19th century, and it has been a slow and painful struggle to bring it back; let us not allow this to happen to geriatric medicine.

# THE CONTRIBUTORS

JOHN F. MUNRO, M.B., Ch.B., M.R.C.P.(E), is Consultant Physician at the Eastern General and Edenhall Hospitals. His work into obesity is well-known to Edinburgh students and he has given a talk to the Society on the subject.

\* \* \*

WILLIAM A. BRANFORD, B.Sc., is a final phase student and third Junior President of the Society.

\* \* \*

CHRISTOPHER SMITH, M.B., Ch.B., M.R.C.P.(E), is Senior Registrar on the Infectious Diseases Unit at the City Hospital, Edinburgh.

\* \* \*

GEOFFREY D. HARGREAVES is currently engaged by the Society in cataloguing all the older medical textbooks scattered throughout various libraries in the city. His work has already revealed little-known examples of valuable rare books.

JAMES WILLIAMSON, M.B., Ch.B., F.R.C.P.(E), is Consultant Geriatrician at the Eastern and Royal Victoria Hospitals. As a result of his efforts Edinburgh students are now given formal teaching in geriatrics although, as he points out in his article, this is still inadequate.

\* \* \*

JOHN PARKER, B.Sc., an honours graduate in biochemistry, travelled on an R.M.S. travel grant in 1971 to Nepal.

\* \* \*

PETER CALVERLY, a final phase student, is a past Secretary of the M.S.C. and Scottish Regional Chairman of the B.M.S.A. He travelled on an R.M.S. grant to India in 1971.

## R.M.S. TRAVEL FUND AWARDS 1971

Alec Workman	Nazareth	£50
Jon Parker	India	£75
Margaret Tatam	India	£50
Peter Calverley	India	£30
Brian Duerden	U.S.A.	£125
Alan Rees Watson	Malaysia	£120

## R.M.S. TRAVEL FUND AWARDS 1972

William Killin	Malaysia	£50
John Nixon	India	£50
Diana Girdwood	U.S.A.	£50
Hardial Singh	India	£70
Sandie Falconer	Uganda	£50
Gail Young	Rwanda	£50
Andrew Norton	India	£50
Beverley Arthur	India	£50

# SOCIETY NEWS

Perhaps the most encouraging feature of the 235th Session's busy programme was the revival of interest in the presentation of Dissertations by members.

In recent years relatively few have taken this opportunity to express their knowledge and views on a subject of their own choice and to defend them if necessary in the face of all comers.

One only needs to look at the list of topics chosen to appreciate that the interests of Society members are still as wide as they have ever been: "Lignocaine — its rational use in Clinical Practice", T. F. Benton: "There's nowt as queer as Folk", R. Bolas: "The Effects of Prolonged Spaceflight on Man", W. A. Branford: "Out Damned Clot", I. J. Cartledge: "Carcinoid Tumour", N. J. Douglas: "Consciousness Altering Drugs" — Man's Search for Unreality", A. R. W. Forrest: "Some Aspects of Cystic Fibrosis", Diana Girdwood: "Concerning the Function of 3' 5' Cyclic Adenosine Monophosphate in Brain", M. A. R. Knock.

Happily, at the time of writing, a sneak preview of the new syllabus reveals that the 236th Session also has its full quota of Dissertations.

This resurgence of Society talent did not, however, prevent the usual importation of guest speakers from outside the Edinburgh medical school. Dr A. M. Braverman of the Harlow Hospital Group spoke on "The Relationship between the Hospital and Community in the care of the Elderly". The importance of Geriatrics to the modern doctor is at last being recognised in Edinburgh and Dr. Williamson expresses his views elsewhere in this issue. Another fast expanding field was presented by Dr. S. Sevitt of the Birmingham Accident Hospital with his talk "Medicine in Trauma and the Role of the Physician". However, in view of the present interest in vasectomy perhaps the most topical of all was Professor J. P. Blandy's discussion of "Male Sterilisation — Snares and Delusions".

The rest of public business for the year was provided by past and present members of the Edinburgh staff. Professor McWhirter delivered the Inaugural Address and Professor A. P. M. Forrest, Dr J. S. K. Stevenson, Dr. J. H. Smith, Dr. W. H. Price and Dr J. W. Farquhar also accepted invitations to speak.

To balance the whole picture of the Session one must, however, present the other side of the coin and regret the poor attendance at some of the meetings. Public Business is, of

course, open to all, so why is it that some draw only a mere handful of students? It may be that they prefer to restrict their attendance at 'lectures' to their University timetable or perhaps our new technicolour advertisements are still not enough. Whatever the reason, one often wonders what happened to those among the 1,100 who flocked to hear Dr. Issels who have never returned.

A further downward trend has also occurred in the attendance at Private Business. This is however a different, a purely internal, matter for the Society. Surely no-one will deny that these meetings should be the core of the Society's existence, whatever fringe benefits it offers to its members. Yet year after year many pay their subscriptions and never even arrive to sign the Obligation (a convenient method of introducing a new member to his fellows), and to see for themselves what Private Business is about. It is to be hoped that the measures to be introduced for the new session will improve "communication" and result in more members contributing to these meetings.

Despite being delivered at times to a very "select" audience, these meetings went on regardless. Among them were the first reports from recipients of R.M.S. Travel Grants. Tales and pictures from such far-flung places as the Malaysian jungles, the mountains and valleys of Nepal and, on a less exotic plane, the metropolises of New England greatly impressed those present with the worth of this venture.

Although many applicants have unfortunately had to be disappointed, the second groups of recipients have already in some cases started on their travels, and as a result of their predecessors' efforts we await their reports with anticipation.

Controversy at the A.G.M. mainly centered around the Revision of the Laws, in particular the annual subscription. In this era of Society affluence it was argued that it could be reduced to a nominal level to encourage an increase in membership. In the end this view was narrowly defeated and members will pay £2.00 as from October.

On the 4th November, 1971 a short ceremony was held outside the new Midlothian County Council offices on George IV Bridge, when Fred Benton, the Senior President, unveiled a plaque commemorating the old premises at 7 Melbourne Place. With the start of demolition on the site of the new R.M.S. building all the plans are at last beginning to take on an air of reality — who knows, perhaps actual construction will have started before the next issue of "Res Medica".

# NEPAL

John Parker

Living in a valley, lush and green, 4600-ft. above sea level, filled with paddy fields, surrounded by terraced hill slopes and dominated by an inspiring backcloth of snow-capped Himalayas is a personal experience to be cherished. Working among a people, poor and poorer yet happy and cheerful, seeing textbook cases of gross, unattended pathology, tropical and "Western", is a medical experience never to be forgotten.

The valley of Kathmandu in Nepal provides the setting. Until 1951, when the King regained control from the Rama family, a hereditary line of Prime Ministers since since 1846, all Nepalese borders were closed to foreign travellers. The country maintained a mediaeval lifestyle with a mediaeval medical care.

Since 1951 the influx of foreign influence has been accelerating. Now Kathmandu is a popular tourist stop. The people, however, are caught between the wealth and status of the visitors and their ancestral customs and poverty. Owing to the very rough terrain of the region and the poor communications within the country, most areas outside the city still retain their original life-style and value system.

In 1953, the United Mission to Nepal began a hospital which still exists as the Shanta Bhawan Hospital (Palace of Peace). It caters mainly for the Nepali inhabitants of the area but also extends its foreign residents and tourists. Charity or credit concessions are available to the poor whilst the rich and foreign patients are over-charged.

The hospital runs between the Western and Eastern styles of medicine. The staff is international, providing wide scope in medical background and practice. The nurses are mainly Nepali, efficient and elegant in their uniforms of white sarces. The most important factor in the running of the hospital is finance — or lack of it. Emphasis is always on out-patient care and admissions tend to be acute conditions: there is little scope for the care of the chronically ill. The in-patients are cared for by their families who also help the nurses in their duties. Diagnostic investigations are limited so that clinical diagnosis is all the more vital.

Being a medical student in such conditions

allowed ample opportunity for performing ward procedures, assisting in the operating-room and even tackling minor operations oneself unsupervised. Nevertheless, what I found to be the most stimulating was the necessity to improvise, compromise or reject. So many factors concerning the patient, the language, patient care and treatment available that are assumed in Britain, are altered so that it becomes essential to reassess their relevance. To take a case history through two interpreters, auscultate the heart of a young girl who believes that baring her breasts before marriage brings ill-luck in child-bearing, or a patient refusing to have his haemoglobin estimated because it is too expensive and he is too proud to accept charity, may seem ridiculous in Britain but are commonly encountered in Nepal. To refuse admission to a person whose prognosis is hopeless may appear unethical, but to brand a hospital with too many deaths would cause unlimited harm.

The obvious advantage of "going East" to work the clerkship is to see gross pathology. Tuberculosis, leprosy, malaria, elephantiasis, dysentery, tetanus and diphtheria, rarely seen in Edinburgh, were frequent in Nepal. Furthermore, "Western" diseases such as heart disease, carcinomas, pneumonia, osteomyelitis, peptic ulcers and neurological disorders will usually present in advanced states whereas early diagnosis is stressed in Britain.

The most valuable lesson which I gained during my visit to Nepal was to appreciate the problems and methods of setting up a medical care system among a people totally ignorant in basic hygiene, in a nation where the medical budget is 1/200 per member of population of Britain's. The hospital becomes a capital expenditure that cannot be afforded when preventive medicine is imperative; the doctor is seen as an over-trained, easily frustrated and expensive member of staff when emphasis is placed on economy.

No-matter-what, the opportunity of visiting a country where the priorities of medical care are different, the relevance of every practice must be revised, the cost of every drug prescribed must be justified and the satisfaction of treating diseases not previously encountered is so immense, should be missed by no-one.

# INDIA—AGAIN!

P. M. A. Calverley

It is with some reluctance that I start another article, for another magazine, about my elective in India just over a year ago. Not that I feel any ingratitude to the R.M.S. who partly financed my trip but merely because I seem to have been talking about India, its culture, its medicine, its people and problems, from the day I set foot again on the chalky soil of Kent and I'm beginning to feel a bit of a fraud, rather like the American who spends two weeks in Europe and then starts to profess intimate knowledge of its every nuance the moment he returns home. At the end of two months in India I had seen enough to realise that I'd seen nothing yet.

Many impressions remain, of course, and I'd like to focus on just two of them. One of the happiest is that of the wonderful hospitality that we received. The Indian people were, on the whole, astonishingly friendly and helpful despite the linguistic difficulties. The latter were not as great as might be imagined as English is widely spoken among educated Indians, partly because it's a useful international language, partly as a hang-over of 'our Indian Empire'. Certainly at the New Civil Hospital in Ahmedabad, a post-war concrete structure with all the architectural grace of the S.M.M.P., the medical students were taught all their medicine in English (despite their previous education in the Gujerati medium), and much of the professional practice was carried on in that tongue. Whilst less than satisfactory for the budding Gujerati anatomist, it was ideal for use as it opened all the necessary professional and social laws.

We stayed in the student hostels which were also sited on the same enormous campus and were quickly befriended by the other students who were anxious to meet us and show us their city. Hospitality is part of the Indian social tradition and at some points, one almost felt a surfeit of it. Certainly two different sets of people showed us some of the same sites on two different occasions! The junior staff

on our ward were also at pains to see us settled and one of the medical internees soon became a good friend who took us to meet his family and to participate in the Indian festival of Rakshavon (pitiful phonetic spelling) when brothers and sisters exchange gifts. The registrars were equally friendly and when we started our journey home it was especially touching that three of our special friends among them, along with my room-mate and friends, should turn up at the station at 11 o'clock at night to see us off.

As you might imagine with such a wide range of social contacts, some of whom were non-medical, we were often invited out for meals. However, I made the sad discovery quite early on that the only connection between Vesta packed curry and Indian curry was the spelling of the word curry. The people of Gujerat like hot foods and hence I once bit greedily into something I thought might be a Cornish pasty, only to discover it was a large green chilli pepper with a flaky pastry coat! Needless to say, it was not deemed polite to deprecate the spiciness (or unpalatability) of one's hosts' food and so bearing a fixed, slightly maniacal grin I ate what I could. It was only when someone showed signs of offering me more from their own plate that I drew the line! The other thing about Gujerati food is that it's all vegetarian and so to break the monotony we were taken out by some of our Indian friends to one of the city's posher meal-serving restaurants where we were treated to something that sounded, tasted and looked like "Chicken Crucified". After that we kept to scrambled egg for a while and certain less spicy Indian delicacies we grew.

It was quite an experience to live in an almost entirely Indian environment for 6 weeks and to see all sides of their city life from the cotton workers' 12-ft. x 4-ft. single room home for five people to the Governor's select Independence Day celebrations. At times it was

frustrating, often amusing and always challenging, but it was also manageable because I had someone from my culture-society with me and because of all the kindness and consideration our hosts showed us. Looking at the polyglot wards of the Royal Infirmary with their willing post-graduates and exchange students, many of whom must feel the same "culture-shock" in reverse, I wonder if we are anywhere near so friendly and what impressions of Edinburgh they will take back to their homelands with them?

Of course, one noticed many things which might be criticised as well as many to praise. In the latter must come Indian medical education (partly because it's one of the few things I feel able to criticise). Much has been written about immigrant doctors in this country, one of the best general reviews being found in *Synapse*, Vol. 21, No. 1. Having seen many such doctors in training their actions in Britain can be viewed a little more sympathetically.

Medical education in India resides in the 200+ medical schools, some of which receive G.M.C. recognition (but only a few), which are financed partly by the Government, partly by the fees their students pay. Inevitably few of the population have sufficient funds to educate their offspring to the intermediate science level required before entry to medical school and, as with other aspects of Indian life, stories of corruption are rife regarding people who 'fixed' their admission. Once admitted the ordeal has hardly begun for the student must now stop thinking and working in his native language and start to use English instead. He must begin his 1½ years of pre-clinical work, with the emphasis on physiology and anatomy and a little biochemistry. The standard anatomy book was Gray's and there were people who seemed to have read it cover to cover! (Wee Cunningham's was used as a sort of hors d'oeuvre for the "real Anatomy" books). Once this chastening experience was passed successfully came the three clinical years timetable much as any British medical school might have 20 years ago with large blocks of medicine, surgery, social medicine et al. The students are taught by lectures and in groups of about 14 on the wards with little in the way of small group tuition. The emphasis is on rote learning and the contrast between the 6-year olds in the little school opposite, learning to read by chanting the words to their teacher, and the medical students doing much the same across the way, was not a great one. The

knowledge imparted was subject to regurgitation in chunks and the emphasis was on knowing facts rather than knowing what to do with them. Despite these several difficulties many of the students and teachers were very capable and managed to escape the confines this rigid system imposed. Resits in at least some subjects were assured for all but the best, each one involving 6 months repetition of the particular course. The school record-holder for resits left just before we arrived, having taken 10 years to complete his 4½ year course!

The newly-qualified graduate, having completed his internship of 4 attachments in medicine, surgery, obstetrics and in the peripheral village hospitals each for 3 months, has then a career choice to make. He can set up in practice in the city which is very competitive; he can practise in the villages where he is needed but has few of the comforts and distractions of the city where he trained; or he can continue in hospital and gain some higher qualification in India and/or abroad. The first two choices pose the same problem many of us face but more acutely so for the Indian villages need doctors more than our "peripheral" towns and have fewer at present. The last choice will lead the graduate to a training totally unsuited for the medicine he will practise should he return home. His hard-won skill in pneumoencephalography will atrophy through disuse and his intimate knowledge of fibrin degradation products will not be exploited to the full. Alternatively he may never return and instead choose to practise Western medicine in the West, with little prospect of the promotion he probably deserves, because as we all know "90% of registrars are Caucasian males". His is not an enviable choice.

This is a deliberately harsh view of Indian medical education but, I repeat, not of Indian doctors who despite all practise a remarkably high standard of medicine considering. Perhaps the difference between student and doctor lies in the intern year when he is more than a student but still with less responsibility than a houseman. The benefits that accrue from this form of learning should be recognised by the Indian Government and the traditional scheme revised. We too should (although B.M.S.A. are) agitate that the criteria for admission of overseas graduates be changed and that more realistic training be provided for them. But perhaps we should attack our own beams before other peoples' motes, but then again that's another article. . . . .



## BOOKS

*Principles and Practice of Medical Computing*: £3.  
Churchill Livingstone, L. G. Whitby and W. Lutz.

For anyone with doubts as to the applicability of computer science to medicine, this book gives a comprehensive view of its present uses and the hopes for the future. Following a basic introduction to computer theory with a lengthy glossary of terms, each chapter in the book describes the use of computers in a specific field of medicine: research, clinical work, administration and teaching. The twenty-two authors involved have been chosen to provide specialised information and have a wealth of personal experience in the different areas. In this way the problems besetting the use of computers in medicine generally and any one speciality in particular can be dealt with authoritatively.

Because or perhaps in spite of the fact that the majority of the contributors are Edinburgh-based, like the major authors, the book as a whole is well-co-ordinated and theory section may seem rather daunting but this is a fault of all computer language and not of the present volume.

The division of the book into five sections with titles ranging from "Specific application of digital computers affecting groups and populations" to "Some examples of the use of computers in research and medical education" emphasises the underlying theme of practical application of computers which makes the book relevant to all medical personnel from general practitioners to hospital administrators. It is pleasant to find a general introductory text of this kind with extensive references allowing the interested reader to follow through any specific topic with ease. At £3.00 the book may be a

rather expensive extra for undergraduates but can be recommended to anyone entering modern medical research, general practice or administration.

U. M. Mac F.

*Textbook of Medical Treatment*, 12th Edition, £4.25.  
Longman Group Ltd., 1971. Edited Alstead, MacGregor and Girdwood.

To review as old a favourite as "T.B. of Medical Treatment" is a difficult task. Everything worth saying has been said by wiser reviewers in the thirty-two years since it was first published.

The first striking change is on the spine. The name Dunlop is missing for the first time — it has been replaced by Girdwood. The shape and layout have changed too. The use of various type-faces to indicate sections and subsections is eye-catching and together with the comprehensive index makes the text a pleasure to use for quick reference, while the clear style of most of the authors is likely to detain one browsing.

The thirty-two contributors, with the exception of Her Majesty's Senior Medical Inspector of Factories, all work or have worked until recently in Scottish Medical Schools, seventeen of them in Edinburgh. The thirty chapters, ranging from "Psychiatry in General Practice" to "Disturbances in Water and Electrolyte Balance and in Acid Base Equilibrium", from "Common Disorders of Infancy and Childhood" to "The Care of Old People", have been revised or rewritten. They discuss treatment in its widest sense emphasising both the practical details and the dangers.

Thus, it is a useful book for the clinical student, the houseman and the G.P. to keep at

hand. However, in a text devoting two pages to the treatment of barbiturate overdose, in an age of drug abuse, it seems incongruous that the section on hypnotics should recommend barbiturates, merely listing nitrazepam along with glutethamide and methaqualone as other hypnotics not proven superior to barbiturates. M.H.

*An Introduction to Clinical Research.* £1.50  
Churchill Livingstone. W. P. Small and Urban  
Krause.

A book attempting to introduce controlled scientific method to clinical research fulfils a much needed purpose. However, if the "basic scientists" should glance through this volume their worst suspicions regarding the intellectual capacity of their clinical counterparts may be strengthened. The inclusion of a prominent and detailed diagram of several box files (closed) and a filing cabinet (open) and such subtitles as "This is fact -- is the patient alive or not?" may encourage cynicism, although the emphasis placed upon follow-up clinics as primarily for collection of research data and only secondarily for the after-care of patients would seem to be in the best "scientific" traditions.

Despite the over-simplification in parts, there is much useful information in this text and such chapters as "The Presentation of Results" with its dire warnings of the effects on an audience of overlong lectures, emphasis on rehearsal for the spoken presentation of results and advice on how to write an article for publication are a rare and valuable find. In its 121 pages the book is claimed to do no more than to serve as an introduction to this topical field and much space is devoted to very practical

problems such as the essential equipment required for follow-up surveys, the tracing of lost patients and the interpretation of results. Examples are taken mainly from post-gastrectomy follow-up in Scandinavia and in Britain, reflecting the interests and places of work of the joint authors. No doubt interested researchers in other clinical fields could extrapolate to their own situations.

New recruits to clinical research will find here, in an easily readable form, details of how to avoid many of the pitfalls encountered by their predecessors. Veterans in this field should find the book useful but might feel that the theories expounded apply to a rather ideal situation and not to the uncertainty of reality. However, the authors must be congratulated on making this preliminary attempt to raise the scientific status of the much-maligned clinical research.

Y. F. L. T.

*The Significance of Physical Signs in Medicine* (1st Edition). £2.25. H. K. Lewis and Co., Ltd., Peter Mills.

This is a book that every medical student should read during his clinical training and will probably want to read more than once. The material is set out in a precise and compact way and deals with the conditions commonly seen in medical practice. It is short, being a mere 93 pages crammed with valuable information, and yet it is also easily readable in a few hours. This combination of qualities should appeal to the majority of medical students, especially just prior to examinations.

Being short there are many omissions and the author has not yet included diseases which do not produce physical signs. Investigations

are not discussed but are occasionally mentioned in relevant places.

It is unfortunate that this book is priced so highly for its size, a feature which will deter many students from purchasing their own copy. Nevertheless, an extremely informative little book.

J. R. A.

*Jameson's Illustrations of Regional Anatomy, Section VII, Lower Limb:* Livingstone: £1. Revised by Robert Warmlesley and T. R. Murphy, 9th Edition.

In effect the new edition of these famous illustrations represents an attempt to bring them into line with the reduced amount of detailed anatomy required to be learned by the medical student of the seventies.

The content remains essentially unchanged. All the plates of the previous edition have been included except one, that of the Relations of the Transverse Tarsal Joint, an omission that will not be missed by many students! Two useful new plates have been added, a radiograph of the foot and one illustrating the dermatomes of the lower limb. Unfortunately, the illustrations themselves have been printed on a poorer quality of paper than that of previous editions and they lack the same definition, clarity and pleasing colours of their predecessors.

There has been a reduction in the amount of legend, which has the advantage of highlighting the more important structures, but the revisers have fallen into the prevalent medical trap of excessive abbreviation. Thus, to take a florid example, the Infrapatellar branch of the Saphenous Nerve is slaughtered to Infrapat. br. saphen.n: this presentation of legend is irritat-

ing and constitutes the main criticism of the new edition.

While undoubtedly these plates will continue to be widely pondered over by medical students it is a pity to note changes in its revision that cannot be said to be improvements.

J.S.H.R.

*Statistics in Small Doses.* £2. A Livingstone Medical Text. Churchill Livingstone. W. M. Castle.

This book attempts with little success to combine programmed learning with an informal approach. Like all programmed learning texts the question and answer ritual is frequently repetitive and frustrating. Answers are printed in the right hand column and a card is provided to cover the correct answer while the reader attempts the questions.

The basic concept of building up the details of the subject gradually, commencing with a description of numbers and measurement, is laudable but explanations of the theory behind the formulae used are seldom given. As described in the introduction, this allows the author to keep the length of the text to a minimum but brevity seems to have been achieved at the expense of clarity. Brief summaries at the end of each chapter are helpful but might usefully have been extended.

The worked examples and numerous tests of progress are useful and are medically-orientated. Formulae are repeated several times and the notation is clear. On the whole this book provides a good guide to some practical uses of statistics in medicine but would require to be supplemented with theoretical background for any basic understanding of the elements of the subject.

N. de P.

# "We take the worry out of money"

... at Bank of Scotland, with a wide range of services specially tailored for personal and business needs. It's probably the most comprehensive range of services you'll find, all designed to take the worry out of money.

For example—

**SCOTLOAN** — helps you get the things you want, without waiting.

**SCOTBUDGET** — provides the easiest way to meet your bills.

**SCOTCASH** — dispenses money 24 hours a day.

Call in at any of our Branches and start to live.



## **BANK OF SCOTLAND**

**TAKES THE WORRY OUT OF MONEY**

---

---

# THE SCOTTISH DEFENCE UNION

offers

## WORLDWIDE PROFESSIONAL COVER

(excluding U.S.A. and Canada)

AT COMPETITIVE RATES

MEMBERSHIP IS OPEN TO GRADUATES OF SCOTTISH MEDICAL AND DENTAL SCHOOLS AND TO OVERSEAS GRADUATES WHO TAKE UP THEIR FIRST U.K. POST IN SCOTLAND.

### 1973 RATES

NEW GRADUATES . . . .	£5 for first year
MEDICAL MEMBERS . . . .	£18 per annum
DENTAL MEMBERS . . . .	£12 " "

THREE MONTHS DEFERRED PAYMENT SCHEME AVAILABLE TO NEW GRADUATES

THE  
**MEDICAL & DENTAL DEFENCE UNION**  
OF  
**SCOTLAND**

105 ST. VINCENT ST., GLASGOW G2 5EQ

041-221 8381

Apply to Joint Secretary for further information.