RES MEDICA
Journal of the Royal Medical Society

Contents

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, New Series No.2, 1980
doi:10.2218/resmedica.v0i2.1980

SECOND ISSUE

Res Medica

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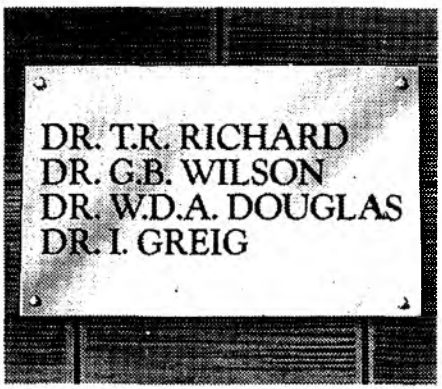

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Journal of the Royal Medical Society

Second Issue — 1980 ISSN 0482—3206

Published by the Royal Medical Society
Students Centre
Bristo Street, Edinburgh

Tel: 031-667-7969

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EDITORIAL

Despite the many exciting discoveries in scientific medicine which fill the medical journals at present, one senses that patients are growing disillusioned with their doctors ability to help them. The staggering increase in litigation cases against doctors and the slowly advancing growth of "alternative medicine" are symptoms of a growing rejection by some of the public of the standard type of medical care they are presently offered. Interestingly, these alternatives are invariably less scientific than the medicine we practice; if we are to recover the trust of our patients and restore a healthy relationship with them we must relearn to value our patients as people and not as objects for our scientific appraisal.

It is hoped that this second issue of "Res Medica" will stimulate its readers to think harder on medical issues, and encourage a more balanced practice of medicine. Hamish Maclaren opens with his criticism of the new Edinburgh medical curriculum and makes observations on medical education generally. Dr. Jack Cormack gives his views on the role of obstetrics in general practice, and to conclude the "Comments and Reflections" section of the magazine Dr. Edward Duvall (Olim Praeses) contributes a fascinating account of the control of infectious diseases.

Under the "Principles and Practice" section Dr. Howard Davies relates the development of the management of haemophilia, a fascinating account of the scientific investigation of a human disease. Since many doctors may be called to give evidence in a court of law, Prof. J.K. Mason gives wise and witty advice "On being a Professional Medical Witness". There follows an account by Dr. Jeanne Bell of her original work in the new field of Fetal Pathology. Finally, Charles Clark has adapted a dissertation he read before the society on "The Diving Casualty".

And last of all, "Medical Maracas" has flowed from the witty and ubiquitous pen of Dr. James Owen Drife.

THE NEW MEDICAL CURRICULUM: A RESTORATION OF THE *STATUS QUO*

by
Hamish Maclaren M.A. (Hon.); B.Sc.
Medical Student: University of Edinburgh

"Where is the knowledge we have lost in information?"
T.S. Eliot – Choruses from 'The Rock', 1934

I HAVE TO WARN YOU: this is a piece of adverse criticism, but perhaps not quite in the way that you might expect.

Last June, *Res Medica* asked me to write an article on the New Medical Curriculum, which has recently been inaugurated in this University. The original idea was that two essays would be written on the subject, one by a Professor of Medicine, and one by a medical undergraduate.[1] In fact, so far as I know, the Professor of Medicine declined the offer for the very good reason that he considered that the new curriculum ought to be given a chance before it is evaluated. I, however, having nothing to lose, accepted the commission, because, as a matter of fact, I have a point of view. But of course there are various reasons as to why my overview of the subject must be even more blinkered than that of a Professor of Medicine. When Faculty switched curricula, she (I always think of her as a young girl) also chose that moment to convert me from a pre-clinical to a clinical student. Now there are various reasons why this should confound me as a critic of the New Order, they are wearisome to relate and surely self-evident to the attuned. I want to get round them by stating that this piece of adverse criticism is not really directed specifically against the New Medical Curriculum, which may well turn out to be much better than the old. I don't want to talk about all the current curricular hot potatoes – the extra time devoted to clinical chemistry, the curtailment of time spent on the wards in Phase II, the question of whether Phase III Year 1 should have to compete against Phase III year 2 in the same subjects, and so on. I have been trying to ask myself what I think is really wrong with the way we are taught. I think that there *is* something wrong, that, as my title implies, the existence of a new curriculum has done nothing to improve the situation, and that, really, the new curriculum represents a series of quite superficial changes in the Faculty's approach to medical teaching, beneath which things are going on exactly as before.

Much of what I have to say here concerns the earlier part of the medical course. Despite the fact that one of the aims of the revising of the curriculum was to wean the student, to some extent at least, off lecture courses, lectures still remain the main educational tool of the Faculty for the first three years of the new course. Thereafter, Phase III represents an attempt to encourage the practice of self-education combined with the gaining, on the wards, of practical experience.

*1 I was to be the prof.

One can say therefore that Faculty puts its concerted teaching effort, quite reasonably, into the early parts of the course.

I will therefore begin by considering "The Typical Medical Lecture", then try to say what is *wrong* with such a lecture, and conclude by reviewing certain implications arising. I may say here, parenthetically, that I have found this article very difficult to write [2], partly because of problems of style, and, related to this, because I have been continually losing my temper with the subject-matter. Though it may be a poor reflection on me, it will probably be more entertaining to you if I *do* lose the place a bit, therefore I'd like to insert in here a kind of blanket apology to any of those who consider any of this to be what I believe Socrates or one of his pupils called an "argument *ad hominem*". Nothing personal.

"The Typical Medical Lecture"

At 9 o'clock on a Monday morning 150 very talkative but somewhat bored and definitely unexpectant students gather in a lecture theatre. The lecturer, an harrassed individual, enters with the hand-outs at about four minutes past nine. There is a certain trafficking to and fro within the body of the assembly as these handouts get disseminated. During this time the lecturer consults with the servitor, arranges his slides, puts his flimsies on the overhead projector, and writes a few things on the board. The background level of noise does not decrease. The lecturer leans for a few minutes on the lectern with a look of mock appeal on his face; the noise does not abate and he begins; the noise abates very slowly.

Over the next fifty minutes, the lecturer employs a variety of techniques to fulfil one task — he spills the contents of a book in the general direction of his audience. The audience participates by transcribing these contents piecemeal into 150 loose leaf folders, to the incessant accompaniment of the low, monotonous background conversation emanating from the rear stalls.

Although this process is undoubtedly tedious, it can also be quite exhausting. It is not uncommon for a lecturer to display a slide on the screen, a flimsy on the overhead projector, and a battery of formulae on the board simultaneous with his own high speed verbal delivery. The conscientious female student sitting in the front row doesn't know where to turn but soon learns that, if she doesn't think about any of the information coming her way, she can usually get most of it into her loose leaf folder. [3]

It is easy to criticise this sort of lecture. But it is worth pausing to consider why people lecture in this way. It seems to me that what appears at first to be a rather low-key, nondescript event lasting for an hour, is in fact a quite highly ritualised conspiracy between lecturer and audience, largely designed to soothe the nerves of the lecturer. If you look very closely at *most* (not all) medical lecturers you will see that they are actually quite tense. Who can blame them? But they keep a firm control of their nerves by employing various tactics designed to elevate the background count of boredom. Crucial to this process is the practice of the delayed start to the lecture, the elaborate setting up of the indispensable lecturer's crutch, the visual aid, designed to distract the attention of the audience away from the central figure of the lecturer, to dull the audience with the narcotic of an array of factual information, to get the heads of the audience into the loose leaf folders, with the blind concentration of the post-menopausal bingo player.

*2 It is becoming virtually impossible these days to write an article of this nature — medically orientated without being "scientific" or, in other words, a discursive article, without falling foul of a certain obnoxious error of style. We are forced by sheer weight of tradition to write after a certain manner. I will call this manner, for want of a better term, "Medical Baroque". Medical Baroque is characterised by a kind of smug, complacent undergraduate ribaldry. The exponent of Medical Baroque always laughs at his own jokes. These jokes are invariably full of pus, and sex. In our introductory lecture of welcome to the medical school we were all told, I believe, that there exists some strange subtle bond between doctors and writers (the names Conon Doyle, and Maugham, are invoked). This seems to me to a kind of distortion of logic: it is possible to show that a lot of good writers were interested in medicine, but it is much easier to show that a lot of good doctors are very bad writers. This has absolutely no relevance at all to the New Medical Curriculum, except in that, in my attempts to avoid writing Medical Baroque, I find myself continually slipping into the style of Ivan Illich, the author of *Medical Nemesis*. This to my mind is disastrous. I entertain no ambitions to blow up Edinburgh Royal Infirmary.

*3 If this sounds chauvinistic, it is not meant to be — but it just happens to be the picture conjured up in my mind by the idea of a medical lecture. I suffer the same dilemma, and occasionally used to sit in the front row.

What is wrong with "the typical medical lecture"? Well, we must sympathise with our harrassed lecturers, but at the same time there is something far wrong with the whole dreaded formula of the boring lecture. Most people take a kind of pragmatic, fatalistic view of medical lectures. They may attend the lecture or they may not; probably the majority *do* attend in case they miss something of importance. It is widely recognised that there are good lecturers and bad lecturers, that no amount of tinkering with curricula is going to alter this fact, that it is up to the student to sift knowledge from books and from lecturers largely as he pleases. Bad lecturers are a kind of thorn in the flesh.

And yet there is a terrible consistency in the way in which our bad lectures are bad which makes me hopeful that in fact they could be improved. I am now approaching the main point of this essay. It seems to me that our medical teachers concern themselves very largely in the trafficking of information. This pursuit has a kind of doctrinal, dogmatic basis in a particular notion which finds almost universal acceptance both within medical circles and with the lay public, and which seems to me to be fundamentally invalid: this is the notion that medicine, unlike, say, mathematics or physics, is "conceptually" easy. It is the notion that all that the study of medicine requires is a good memory or, if you haven't got one, plenty of stamina and black coffee. It is the notion that the study of medicine is a matter of memorising lists — the longer the list, the better your knowledge. The process of medical education arising from such notions, equals the process of confronting the medical student with an array of facts. These are the facts that appear on blackboards, slides, overhead flimsies, and books; the facts which are vocalised in lectures, and copied into loose leaf folders. I think this mindless regurgitation of text book information is the great disaster area of medical teaching.

I have discussed, in general terms, the way in which a medical lecture is a mass of factual information, but I really ought to give a concrete example of this. Every medical student's first exposure to undergraduate medicine is his first 9 o'clock Monday morning anatomy lecture, the first horrified glimpse of the cadaver to the accompaniment of the smell of formalin, impinging on the consciousness. I think most people are rather bad at anatomy, probably because a good visual imagination is relatively rare. Anatomy is conceptually difficult; I wonder how many students at the end of Phase 1 Year I have a good mental picture of, for example, the convolutions of the peritoneum?

The first anatomy lecture I ever went to concerned, among other things, the position of a certain neurovascular bundle passing through the axilla and drawn in transverse section for our benefit at the level of T4. I didn't know what a neurovascular bundle was, nor a transverse section, nor T4. Come to think of it, I didn't know where the liver was, and I don't think I'd ever heard of a spleen. Did that lecture therefore do me any good? I suppose it might have had a value as a kind of shock treatment designed to make me open a book. I realize now that that was its purpose. But this effect might equally well have been achieved by the use of some non-specific shock tactic, perhaps by sneaking up behind me, unawares, and firing blanks from a pistol three feet from my ear.

I think anatomy lectures could be of great value,[4] but as they stand at present, (and I'm talking of regional anatomy) I think they are unsuccessful. But I do not wish to make an isolated attack on the anatomy department. What I say here could equally well apply to a host of other departments. Lecturers seem to have this pathological desire to vocalise all the examinable facts at least once, as if such a recital absolves them of all responsibility for the student's progress (a responsibility which they never held anyway), and puts the onus of making good headway squarely on the student's shoulders (it was there in the first place). It seems to me to be self-evident that the lecturer is there to help the students (he certainly isn't there for his own benefit, so what other reason can there be?). But the presentation

*4 Would it not be more sensible to supply an idiot's guide to the human body at the first anatomy lecture; It might even be a good idea to get an idiot to give the lecture, somebody who had not forgotten the extent of the ignorance of his audience. It would be helpful to find out, on that occasion, where the really big bits reside.

of a "factual package" is a quite futile undertaking because such a package exists in the text book anyway.

What then should a lecturer do? To my mind, while he is preparing his lecture, he should ask himself the following questions:

(1) What are the crucial, guiding principles of today's subject (assuming there are any)?

(2) How can I present them in such a way as to keep my train of thought as simple as possible without becoming incomprehensible to an audience which has little insight into the background of my subject?

(3) How can I present a readily digestable package of knowledge upon which the student can subsequently build up for himself the quantity of information that he undoubtedly needs to know in order to function as a doctor?

If I were asked to say in one sentence what is wrong with the way I have been taught in this university, I would say that hardly any of our teachers have offered an "approach" to the subject. We have been swamped in a great welter of undigested, undigestable information. We cannot see the wood for the trees. I could count on the fingers of one hand the number of lecturers who have stood up and said, "This is the way I hold a given body of knowledge in my head; this is my approach to the subject."

Why not? Why do they not do this? Let us consider the possible answers to this question. They are:

(1) Our lecturers have no more "approach" to their subject than we do. They too, have learned it by rote, and continue to hold it in their minds as a task of memory.

(2) They have an approach, but they fail to draw attention to it because of (a) indifference (b) embarrassment (c) belief that it is not relevant.

(3) They have an approach but they conceal it because the medical profession must be entered by a kind of masonic, personal ordeal-by-rote-learning.

(4) They have an approach but they conceal it to cut down the competition for top-grade medical posts. [5]

If I were to be woken up in the middle of the night and asked which single one of these possible reasons was most *a propos,* I would settle for reason No. 2c. For some unknown reason, we have this desire to depersonalise our knowledge in favour of some impossible god's-eye view of the subject.

Implications

This apparent refusal of medical teachers to place ideas and facts in some kind of hierarchical order, to point to the facts that are crucial in that other facts may be deduced from them, to pass over the dead-end facts, has meant that the medical student gradually forgets how to use his brain, if the mental exercise involved is not merely the act of memorising. This is true despite the following quotations:

> "My first point is therefore this, that in any branch of university education, including medical education, we should aim at using the methods of education rather than instruction. We must teach the student how to collect the facts, to verify them, to assign a value to them, and how to draw conclusions from them and test those conclusions; in short, how to form a judgment. As Karl Pearson said, 'the true aim of the teacher should be to impart an appreciation of method rather than a knowledge of facts. . .' "?
>
> Sir George Pickering
> Medicine's Challenge to the Educator.
> BMJ, 1958 Vol 2 p. 1117
> (Quoted on the frontispiece of Macleod's Clinical Examination).

*5 One is reminded of an occasion when Andre Previn asked the one-time Principal Horn of the LSO if he found any advantage to his playing in having a beard. The man replied, "Only insofar as it conceals my *embouchure* from my students."

"If there is a fault in us bred of familiarity it is, I believe, the old fault of omitting to probe sufficiently deeply into causes; the fault of accepting the fact of common symptoms without trying to explain them."

John A. Ryle (1948)
The Natural History of Disease.
(Quoted at the head of Ch. 3, Macleod's Clinical Examination – The Analysis of Symptoms and Signs).

Despite all this, we are still left struggling with the welter of uncategorised facts. A lucky few seem to respond very well to this educational system, and shine. Most people learn, pragmatically, to ride the system, and to put up with varying degrees of mild neurosis. One or two have "nervous breakdowns" (whatever they are) and retire, temporarily or permanently, from the field. The identity of these unfortunates is usually utterly astonishing to everybody else. Very often they are remarkably bright, perhaps somewhat unwordly individuals who have failed to realize that if you want to learn Pathology, you learn it out of a textbook of medicine; if you want to learn Bacteriology, you learn it out of a textbook of Pathology (a good method of getting to grips with the real "basics") – simple, obvious little tricks like that, to be gleaned from the inter-student exam tips Black Market, certainly not from the staff.

But most people seem to acquire the MB ChB – and then apparently settle down to learn some medicine. The undergraduate course seems to be a kind of ordeal by tedium and strain; but you ride the system, don't buck the system, and treat it as a bit of a game, even a joke.

This, at any rate, is the prevalent attitude of the survivors. It just seems a pity that undergraduate medicine is so universally envisaged as a hurdle to be crossed rather than as a preparation for subsequent medical life. And it's a pity that the people who help you over the hurdle are not the lecturers, but very often last year's students; thus you acquire an approach to the subject, stealthily from the commonweil with the audacity with which, twenty years ago, you might have visited a back-street abortionist.

I have heard people say that the "Sink-or-Swim" predicament of the medical student is character-building, that it prepares him for the "one long oral examination" that is a medical career, that it allows him to become accustomed to stress. I don't think this is so. I think most people respond to encouragement rather well.

Besides, the stress in doctors' lives is almost all iatrogenic. We doctors and medics build stress around us; we accept it every time we acknowledge as normal the behaviour of the obsessive surgeon who throws scalpels and retractors at the poor wee nurse who can't find the swabs. ●●

THE G.P. OBSTETRICIAN — GONE FOR EVER?

by
J.J.C. Cormack MD FRGGP DObst RCOG
General Practitioner, Edinburgh

I never accept proffered cups of tea during the course of duty — well, hardly ever. The one invariable exception used to be the inevitable cup offered by the proud new father: his one constructive act at the time of the delivery at a home confinement. What could be more happy, more natural, more satisfying that the birth of a new baby in the environment of its parents' home, in the midst of the family to which it was the newest addition? When all went well — and that was undoubtedly the norm — the pleasure, indeed the unequivocal joy, of all concerned, parents, midwives, grannies, doctors — was one of the great abiding satisfactions of general practice.

There was, though, an obverse side to the coin. The prolonged labour with exhaustion starting to supervene, the occasional limp, apnoeic babe, the mother bleeding steadily with placenta stuck, these could cause concern and anxiety and sometimes downright alarm never fully compensated for even by the existence of the most efficient flying squad.

The pattern of change

Fifteen years ago when I started in pactice we would have two or three home confinements a month — now we have none. What has happened, and why?

Somebody has said that the best place for a mother to have her baby was her own home and the best place for a baby to be born was in a hospital. There is much truth in that, if for 'in a hospital' one substitutes 'in easy reach of anaesthetists and paediatricians'. Obstetricians too, perhaps, but not quite so urgently, for the skills of resuscitation, both paediatric and maternal, are required, if they are needed, within minutes, while obstetric skills can generally be deployed with slightly more leisure.

Childbirth remains (thank God) a physiological process, but neonatal mortality rates considered acceptable even a decade ago are now seen as an affront to proper care. Where human life is at stake risks have to be minimised. The recent advances in obstetrics have been largely in the field of monitoring and early intervention if indicated round the expected time of delivery. Technology has invaded the labour ward, and to be fair, so has humanity, in the sense that obstetric units now more than fifteen years ago, strive to be aware of and sensitive to the needs of mothers rather than laying down authoritarian and unquestionable regimes.

In the mid — 60's the g.p. obstetrician's first skill was in selection: the rigour of selection in many instances depended upon the availability of obstretric beds and the philosophy of local consultants. The grand multips and the women who had had a previous ante- or post-partum haemorrhage were obvious candidates for hospital confinement. But what about primiparity? The 'elderly primip' used to be 35, but the age at which consideration was given to this unlovely title steadily dropped.

What about previous instrumental delivery? With more and more primips being delivered in hospital and with an increase in the rate of intervention there were fewer para I's who had not had inductions and/or forceps deliveries, and so

a cycle of hospital confinement — instrumental intervention — future hospital confinement was begun. Then there was the birth rate itself; the obstetric beds planned to accommodate the results of the post-war population 'bulge' were under less pressure as the birth rate stabilised, but they still needed to be filled.

Thus the criteria for possible selection for home confinement became more stringent till the point was reached where the g.p. obstetrician, often with experience as an SHO in obstetrics, and with a Diploma in Obstetrics, was attending only three or four confinements in a year. At this stage his intranatal skills were in danger of dis-use atrophy and if he was wise he withdrew from the field and reorientated his thinking, admitting gracefully that changed times required changed practices.

The present position

The ideal solution, at least in urban areas, might seem to be the existence of g.p. maternity units, in close proximity to consultant units, with the facility of easy consultation and if necessary transfer of patients. To such a unit the patient, selected with proper consideration and given full antenatal care by her practitioner and midwife would be brought when in labour, delivered by the district midwife and the practitioner and, all being well, returned to her own home within four, twenty four, forty eight hours — whatever seemed appropriate in all the given circumstances.

Several such units exist south of the border and suggestions were raised that there might be similar units in Edinburgh, but these met with intractable opposition from the consultant obstetricians (for reasons not always very obvious) and from consultant paediatricians who had perhaps greater grounds for doubts about general practitioners' experience and skills in the more modern concepts of neonatal paediatrics.

Some general practitioners, especially in close geographical proximity, are able to deliver their own patients in consultant units (there is at present such a scheme in operation at the Simpson Memorial Maternity Pavilion in Edinburgh), and thus retain and practice their delivery skills. For the rest, does the virtual elimination of domiciliary confinements mean the demise of the g.p. obstetrician?

I think not — provided he sees his role as general practitioner first and obstetrician second. As a general practitioner his primary role lies in diagnosis and the establishment of relationships: diagnosis in the sense of the assessment of the physical, psychological and social well being of his patient rather than simply the attachment of pathological labels; relationships in the sense of building a pattern of continuity within which doubts and fears can be discussed and guidance given in as relaxed and informal a manner as can be established within any professional setting.

The pattern of care

The modern g.p. obstetrician has the responsibility of confirming pregnancy, of arranging for confinement, of organising and supervising antenatal care, of overseeing the puerperium and of providing postnatal advice. Above all he has the responsibility of providing continuity of care.

In these days of population mobility a pregnancy is often the practitioner's first contact with a new family; it provides the opportunity for collecting data about the family and for getting to know at least the new mother-to-be and in many cases her earlier children who may come along to the antenatal clinic toddling or in push chairs and see the doctor in a non-threatening situation. The mechanics of antenatal care are not essentially different whether provided by general practitioner or hospital clinic, but the setting in general practice is likely to be more relaxed, less antiseptically clinical and almost certainly more personal.

The high technology of obstetrics — amniocentesis, ultrasound, foetal monitoring and so on — belongs properly to the hospitals; the simple technology of stethescope and sphygmomanometer, trained hands and eyes and above all sensitivity to individual's needs belong wherever medicine and obstetrics are practiced well — in the community setting as much as in the institutional one.

The g.p. obstetrician must, as soon as pregnancy is confirmed, ensure that the benefits of high technology are made available to his patient; he must organise referral for booking for confinement and he must see that liaison is maintained between himself and the hospital so that the

fullest information is available should there be any sign that things are not progressing satisfactorily. Many of the base line measurements such as blood group, WR and MSU will be done at the hospital booking visit and there is little sense in duplicating these, but it is worthwhile for the general practitioner to take off blood for rubella antibodies at the earliest possible stage, as it is he who will have to sort our the situation if the mother comes in contact with a child with proven or suspected german measles.

Teamwork

The organisation of the general practitioner's own antenatal clinic will vary depending on his supporting staff and availability of space. If it is possible to obtain the assistance of the local district midwives this is valuable not only because of the specialist skills they bring to bear but equally importantly to introduce the concept of teamwork and to enable midwife and mother to get to know each other before delivery, as increasingly district midwives are involved in puerperal care. Like general practitioners, district midwives (unless directly attached to hospitals, as many of them now are) have decreasing experience in intranatal work. With earlier discharge from hospital maternity units, however, the midwives' role in the home during the puerperium is of increasing importance as highly trained nurses and as experts in teaching the elements of baby care.

The other member of the primary care team who should be involved is the Heath Visitor. Her role is an educational, preventive and supportive one, and she has a statutory duty to visit babies from the age of 10 days onwards. If she already knows and has established a relationship with the mother before the baby arrives on the scene then her effectiveness in her job at that crucial but emotional stage of the family's development is considerably enhanced.

The general practitioner in this as in many other situations acts as a co-ordinator of services, supplying some himself and enlisting the help of other members of the team as appropriate. The question of the 'leadership' of the team is one which is quite frequently debated, but in functional terms the debate tends to be unreal. The ultimate responsibility rests with the general practitioner and while he need not be authoritarian his responsibility can only be discharged if he remains effectively in control as co-ordinator.

The new family

Having provided at least the major part of the antenatal care the general practitioner will in present circumstances often (indeed in urban areas usually) hand over to the consultant obstetrician and his team the responsibility for care immediately before and after delivery and for the conduct of the delivery itself. However, obstetric care does not end with the delivery; it continues for some time thereafter before it merges imperceptibly into general medical care in the continuing process of advice on feeding, contraception, developmental assessment, immunisations and the management of illnesses in the family.

The visit or visits to the home of the mother and new baby after discharge from hospital provide an excellent opportunity to strengthen the link of the doctor-patient relationship which is the rock on which all good practice stands. Although this is the time when the midwife and the Health Visitor traditionally deploy their skills in giving detailed advice and help, the doctor continues to be the co-ordinator and it is he who must ultimately, with his colleagues, plan the policies which are to be followed with regard to follow-up and prevention (in particular developmental assessment and immunisation programmes).

The postnatal examination, traditionally at or about 6 weeks after delivery, provides an opportunity to demonstrate the role of the doctor and his team in preventive as well as curative medicine. Provision of, or guidance about contraception, advice on cervical smear programmes and perhaps breast self-examination, checking on rubella immunity status and emphasis on the importance of immunisations for the new baby are all important parts of this closing of a chapter which at the same time opens a new book.

Happy at his work

Our practice midwifery bag is black and battered: it is a leather Gladstone bag and it bears in faded gilt letters the initials of my grandfather: it

was in use at my own birth and at the birth of one of my own children. Perhaps I can be forgiven a nostalgic pang as I glance at it gathering dust in a cupboard, ready still for use in an emergency, but not in regular action over the past eight or nine years.

Some of the fun and some of the romance has certainly gone out of the g.p. obstetrician's life with the handing over of intranatal obstetrics to the hospitals — but so too has some of the anxiety and the occasional tragedy. The g.p. obstetrician of today has an important role — the emphasis has changed but the important core remains — that of establishing and maintaining a skilled caring relationship and providing stability and continuity. By remaining in the obstetric field (and there are provisions whereby he may opt out if he chooses to do so) the general practitioner can still lay claim to his other title of family doctor.

The march of time may have deprived me of those cups of tea, but it has not removed some of the deepest satisfactions of a challenging and satisfying discipline.

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LAZARRETOS

An Outline of the Measures taken Against Epidemic Disease from Biblical Times to the Ascent of Queen Victoria to the Throne (1837)

by
Dr. Edward Duvall, M.A., D. Phil., M.B., Ch.B.
Past Senior President of the Royal Medical Society from his Valedictory Address to the Society

Tonight I would like to talk about Lazarettos. An alternative title might be "a historico-economic study in depth of socially accepted means of community behaviour and preventative disease containment in the ongoing epidemic enzootic disease situation", or more shortly – "To flee or not to flee". I would like to discuss the various attitudes and preventative measures taken by populations when they are threatened by an outbreak of an epidemic infection. By the way, for those of you who are still wondering what a "Lazaretto" is and have not found out where the dictionary is in the library – it is derived from the Italian word "Lazaretto" and is a house for the reception of the diseased poor, especially lepers. Lazar houses were a particularly popular means of controlling epidemic disease in the Middle Ages – but more of that anon.

The measures taken by any group against a sudden outbreak of disease obviously depend upon the current theory of its causation. Probably the first hypothesis explaining the universe was a magical one where anthropomorphic supernatural forces decided the sequence of events. According to this view epidemic disease was caused by a righteous and avenging God, or fickle and feckless deities. Thus, the rational approach to an epidemic was to placate the offended party, or parties. The spread of the twelfth plague of Egypt where the first born of the Egyptians and their cattle were killed was combated, in an attempt to appease Jehova, by allowing the Children of Israel to depart. Similarly, when the Philistines captured the Ark of the Covenant, they were stricken by a lethal disease, characterised by an outbreak of "emerods in their secret parts". The "lords of the Philistines" and their "priests and diviners" sent back the Ark and compensation – or hush money of five golden emerods and five golden mice to the Israelites in an attempt to divert the wrath of God (with a singular lack of success). An emerod is said to be a haemorrhoid – the mind boggles at what a golden one looks like; other authorities claim that emerods are "buboes" and that the association with mice suggests the Philistines knew of the connection between plague and rats.

A similar belief in divine displeasure led to the rise of the Flagellant sect during the years 1348-50. The Flagellants were large bands of itinerant penitants who toured the countryside scourging themselves and performing other monstrous penances. Thus they took upon themselves the sins of the citizens of towns they passed that were threatened by the Black Death in exchange for board and lodging.

This belief that disease was a divine punishment persisted in a diluted form into the seventeenth century as the regulations issued during the outbreak of bubonic plague in London in 1629 had, along with quarantine instructions, ordinances to suppress such ungodly pastimes as stage plays, bear-baitings, tumbling, rope dancing, prize fencing, cock-fighting and bull baiting. The number of ale-houses was to be reduced and the rules preventing the sale and consumption of meat on fast days were strengthened.

In medieval times this belief in malignant

external influences was modified to throw the blame on unfavourable conjunctions of the planets. Guy de Chauliac thought that the plague of 1348 could be, in part, ascribed to the influence of Saturn, Jupiter and Mars in providing a favourable environment for the plague which, in cooperation with a patient's individual diathesis led to an outbreak of the disease.

The first attempts to postulate a physical cause for disease rather than a spiritual one were made by the Greeks. Hippocrates in "Airs, Waters, Places" expounded the doctrine that adverse winds and atmospheres might be responsible for disease. During the plague of Athens in 430 B.C. Thucydides describes the Athenians lighting fires to try and purify the air. They hedged their bets by supplicating their gods (in case Hippocrates was wrong). Empedocles and Acron (two physicians of the time) are credited by legend to have been able to control these bad winds. There is a possibility that they had the mountain passes through which the poisonous winds were blowing down onto Athens, blocked, as a public health measure, thus giving rise to this legend.

The theory that a miasma caused disease developed into the belief that bad smells would have the same effect. This idea was current in Anglo-Saxon England and was probably the basis for the numerous edicts issued during outbreaks of bubonic plague much later in London in 1349, 1629 and 1665 and in Edinburgh in 1585 to enforce the inhabitants of these cities to keep the streets free of the ubiquitous filth and to control trades such as fat rendering, tallow candle-making and fleshing which produced noisome smells. In London in 1665, for example, laystalls, i.e. middens, had to be made far from the city, unwholesome fish and flesh and musty corn was not to be sold and no hogs, dogs, cats, pigeons or ponies were to be kept in the city.

A last vestige of this belief in bad air was seen during the outbreak of yellow fever in Philadelphia in 1793 when the Town Major had to forbid the practice of lighting bonfires in the street to purify the air. He later had to ban the firing of guns which was also thought to have some effect upon the constitution of the atmosphere.

With these two theories of causation of disease — that either supernatural forces or climatic influences produced plagues there was little that a civil authority could do when the community was threatened by an epidemic except pray and hope for a change in the wind. However, with the advent of the Black Death in the 1340's came the realisation that epidemic diseases were contagious and that measures might be taken to prevent the spread of the disease both to the community and within it. Boccaccio mentions that in Florence in 1348 it was recognised that attendants of bubonic plague victims could catch the disease from their patients and also from their fomites.

Once this was realised policies of isolation were instituted, primarily by the Italians who in the succeeding centuries were always at the forefront of sanitary organisation. (However much they may have slipped back lately). They developed the concept of quarantine and of a cordon sanitaire as protection against epidemic disease.

In 1404 the Council of Health of Venice opened the first quarantine station and lazaretto on the island of Santa Maria de Nazareth — two miles from the city. Here all people and goods arriving from the Orient (from whence the plague was believed to emanate) were confined for forty days. This period was apparently chosen rather arbitrarily as both Moses and Christ spent forty days in the wilderness. This method was widely adopted. In London a quarantine station for ships and their cargoes was set up on the lower reaches of the Thames in 1663 to stop the plague spreading from Amsterdam and Hamburg. Typically, Pepys complained that the 30 day isolation period could not strictly be called a quarantine. In Scotland a quarantine period of 15 days was chosen. This approaches the actual incubation time of bubonic plague which is at most 14 days. From the inauguration of the use of quarantine it was realised that consigned goods as well as the sick themselves might spread the disease. Cargoes were often fumigated with sulphur or vinegar or simply exposed to the air and sunshine for a time before they were admitted into a town. In the seventeenth century the Scots burghs had a regulation that any man who wanted to claim goods that came from a place where there was plague had to live with them and handle them for a set period of time to show that they were non-infectious.

The Italians built up an organisation which united the many warring states of the "geograph-

ical expression" that was Italy in an attempt to stop the spread of plague – especially during the years 1618 to 1659 when war and disease were both endemic. They had an elaborate system of health passes – first introduced in 1485 by the Venetians – as well as a swift system of communication between the Health Boards of each major town allowing each to close its frontiers against travellers and goods from any district where plague had been discovered. The normal practice was to establish a cordon sanitaire round the town rather than to seal off the town itself.

This system was rarely effective in stopping the spread of disease both because of inefficiency in enforcing any ban on travellers from a particular area and also because there was resistance from the influential mercantile interests of the towns to any admission of the presence of plague as this seriously affected their trade. A third reason was that in the case of bubonic plague the regulations only affected the movements of relatively inefficient vectors of the disease (man and his fleas) and not those of the primary vectors – the rat and the rat flea.

Once the pestilences had gained entry into a community then the reactions of the civic authorities were remarkably similar in Italy and Britain from 1350 or so to the 1660's. Regulations that all suspicious deaths were to be reported were first issued in 1374 by Visconte Bernabo of Reggio in Calabria. He devolved this responsibility onto the clergy and many communities followed suit, for example, Venice, in 1485 and Vienna in 1562. The London regulations of 1663 required the master of the house to give notice within two hours of persons complaining of blotch, purple, swelling or any other symptoms not assignable to another disease. In case the master of the house was not to be trusted to report plague,"searchers" were often appointed to go round the community and detect victims. In London, from 1532, these searchers, who were elderly and supposedly wise matrons (not medically qualified), provided the data for the parish clerks to compile the Bills of Mortality. These Bills analysed the causes of all deaths within the parishes of London and were intended to warn the Court and the nobility that London was becoming too unhealthy and that it was time to remove to Windsor or Oxford.

Once the existence of cases of epidemic disease (usually bubonic plague) was known then they and their contacts were usually sequestrated. The first incidence of this was in Reggio in 1374 where regulations were issued that all those sick of the plague were to be taken out of the town into the fields to die or to recover. All those who had nursed the sick had to go into isolation for ten days before having contact with anyone afterwards. This harsh attitude towards the unfortunate sick persisted and is also seen in the proclamation of James IV in 1497 that those citizens of Edinburgh suffering from venereal disease (at that time an epidemic) should be exiled to the Inch (in the Firth of Forth) until cured. A more humane attitude eventually prevailed and two policies were adopted – sometimes both at once. The Italian solution (and that used in Edinburgh) was to provide pest houses or lazarettos outside the town to house the sick and their contacts whilst that adopted in London was to isolate both categories of people in their homes.

The Italian pest houses were sometimes custom built – often on a very large scale. That in Milan in 1630 held 15,000 patients and the Veronese one had 4,000 inmates. These lazarettos had separate divisions for the infected and their contacts. They also had attached convalescent homes as those recovering from the disease were recognised as infectious.The smaller towns requisitioned a building for a lazaretto when required. In Prato a monastery outside the town walls was taken over. Conditions in these pest houses must have been very unpleasant as in Prato there were five to a bed and shortages of food and other necessities. They did, however, have a staff of surgeons (physicians, being of a higher class, usually thought themselves too valuable to expose themselves to infection) and lay nurses – unlike the British lazar houses. In the smaller towns which could not afford large lazarettos the contacts of the sick were confined to their houses rather than also taken into the pest house and they were supplied with food by the municipal authorities.

In Edinburgh there was no permanent lazaretto established but "huts and ludges" were erected on various parts of the Burgh Muir when the need arose. This stretched from the present Royal Infirmary to Blackford Hill. In 1501 the town

council authorised the construction of St. Roch's chapel near the present Astley Ainslie Hospital and provided medical attendance there for any sick of the plague. In 1585 temporary hospitals were set up near the site of the Astley Ainslie Hospital and at the ruined convent of St. Catherine of Siena (to the south of the present Royal Hospital for Sick Children). "Cleansers and curers of the pest" were appointed by the town council to attend the sick at these hospitals. The pay was high for those who survived. Alex Fraynche was given a free house and a pension for life for being in charge of the staff. Later in the outbreak of 1585 the sick were segregated at Adam Purvis's Acre – to the west of the Astley Ainslie site and convalescent centres were set up at Morningside, Canaan and Greenhill.

A different approach was adopted in London. The first two pest houses were only set up in 1636, which was probably just as well as they had a mortality rate of 98%. The main measure to prevent the spread of infection was to seal up any house where there was plague with its occupants still inside. In 1583, 1606, 1629, 1663, 1665 and 1666 proclamations were made that plague stricken houses were to be shut up with their inmates for 40 days and a red cross and the inscription "Lord, have mercy upon us." painted on the door. Often guards were provided to see that no-one entered or left the house. This measure only helped to ensure the spread of plague throughout the inhabitants of the house. A classical example of the dangers of isolating a group of people together once one has contracted plague is that of the village of Eyam in Derbyshire. In 1665 the village was visited with the plague and the rector N. Mompesson persuaded the villagers not to flee. Their continued and maintained contact with the infected rats of Eyam led to the death of 75% of the human population.

With some of the populace immured in their homes and trade with the outside world stopped some form of poor relief was required to stop famine killing off more than the plague. Edinburgh in 1585 was the first city in Britain to institute this and an official was paid six pounds Scots a month to distribute bread and drink to the needy. The Italians and the burghers of London followed suit by providing a subsistence diet to those confined to their homes. The administration of Prato also gave subsidies to those whose livelihood was directly effected by the plague and the measures taken against it.

Further methods to prevent the spread of infection apart from segregation of victims were usually used. In Prato the bedding of those who had died of the plague was destroyed with most of their possessions that were hairy or woolly as it was thought that the contagion adhered more readily to these objects than to shiny metal surfaces. Anything too valuable to destroy was disinfected. All those leaving the convalescent home were supplied with a fresh suit of clothing. Houses where plague had broken out were fumigated with sulphur, lime or vinegar and often replastered. In Edinburgh it was prohibited to wash the clothing of plague victims in the South Loch which was the main source of the city's water supply. One of the main duties of the cleansers in attendance at the pest houses on the Burgh Muir was to boil the clothes of the sick in a large iron cauldron provided. The badge of their office was the long cleek provided for this job.

A very common procedure was to ban all public assemblies especially those which might be construed as ungodly. In London, public gatherings, particularily at stage plays and other entertainments were banned. This served the double purpose of preventing contagion and reducing any likelihood of Divine wrath as an aetiological factor. Foreigners were frequently expelled, as in London in 1580 – perhaps because they were "Godless" and might also have come from a plague stricken place. The Jews in particular were given a rough time; many of them were slaughtered on the pretext that they had been poisoning the wells to produce disease or that they were spreading the plague by distributing pus from bubos. The reasons for this antisemitism are complex. The very presence of the Jews was probably regarded as a reason for Divine displeasure whilst they themselves were the only usurers and source of capital in the medieval economy and were for that reason hated by those who had to make use of them as bankers and moneylenders. A third reason is that the authorities may have whipped up pogroms against an easily recogniseable subpopulation as scapegoats for the administration's own inability to counter

the plague.

Having outlined the administrative measures against epidemic disease taken in the fourteenth to the seventeenth centuries in Europe, let us examine the organisations which took them. The Italians, at the forefront of the fight against disease, had special bodies set up to deal with the problems. The first of these was in Venice in 1404 when a Council of Health, consisting of three noblemen was set up to decide upon policies to be used against bubonic plague. Venice was in the vanguard as it was one of the chief European ports trading with the Orient. Other Italian cities followed this example. I shall discuss the organisation in an Italian town by referring to the town of Prato in Tuscany (13 miles from Florence) mainly because some industrious Italian has written a book about it all. When Prato was threatened by plague the town council established a Health Board of four (later eight) laymen — usually gentlemen — to organise the fight against the disease. To advise the Board were the local college of physicians, the doctors of the district and health officers seconded from the capital, Florence. It is probable that expert advice was not often required as the measures taken to stop the movement of people and to run a pest house did not require any medical knowledge. To help supervise the Health Board employees — 25 for a population of about 17,000 — the Board chose a layman Provveditore della Sanita, one Christophano du Guilio Ceffini. Cristophano wrote that the duties of his post were to:

(1) trust in God, the Virgin and the Saints,
(2) disinfect with sulphur and perfumes, rooms and houses where there had been sickness and death,
(3) segregate the sick,
(4) burn fomites,
(5) shut up houses where infected people had been and to quarantine the inmates for 22 days,
(6) stop trade,

The city Health Board also thought that he should:

(7) administer the pest house,
(8) isolate contacts and pay subsidies to those who required them i.e. those who were unable to earn a living because of the effects of the anti-plague regulations,
(9) end quarantine on houses at the appropriate time as some people were keeping themselves shut up in their homes after the required time in order to collect the subsidy paid to the inhabitants of all closed houses,
(10) enforce all Health Board decisions,
(11) audit all the subsidy accounts,
(12) supervise the workings of the public health service and ensure that the patients were treated well and that the dead were buried.

A tall order for a man paid the same wage as the municipal grave digger!

Thus the Italians had an organisation exclusively to deal with the situation. The central government formulated the policy to be taken — prevention of movement into an uninfected area or out of an infected one, separate segregation of the sick, the convalescent and the contact, a disinfection programme, subsidies to be paid to those effected by the plague. In the towns there were physicians and surgeons employed by the municipal authorities (not exclusively during epidemics) to treat those who could not otherwise have afforded medical care. In the countryside the local justices were instructed to popularise the current remedies against the plague if the populace had no access to a physician. The local government set up and paid for its own apparatus to carry out these policies with the help (or hindrance) of a Commissioner from the capital who reported back on the state of affairs.

This organisation should be compared with the contemporary situation in Edinburgh. Let us take the epidemic of bubonic plague of 1585 as an example. There the town council assumed full responsibility for coping with the situation. This was hindered at first because James VI's placeman as Provost, the Earl of Arran, decided that Edinburgh was altogether too unhealthy for him when he discovered plague was present and the council could only function once a new, less pusillanimous Provost was chosen. They then directly issued ordinances of very much the same import as those of the Italians. Pest houses with separate

accommodation for the infected and their contacts were to be set up on the Burgh Muir, a town surgeon, attendants for the pest house and an official to pay subsidies to the destitute were to be appointed and all public assemblies were to be banned. It is difficult at this late stage to compare the efficiency of the two systems — the specialised and the ad hoc but it is interesting to notice how the two different approaches led to a common solution.

One interesting facet of the story is the problem of the execution of the arrangements made. One difficulty encountered in both Edinburgh and Prato was the lack of money. It was very expensive to set up a pest house and to distribute even the bare minimum of food to keep alive those confined to their homes or unemployed because of the plague. It was doubly difficult to find the money because the town had no income from customs duties in Italy or from the working of public land in Edinburgh. Both administrations also could count on a smaller income from taxes because of the death or destitution of so many ratepayers.

A second difficulty which had to be faced, particularly in Italy was pressure from merchants to conceal the outbreak of plague or prematurely to declare the epidemic over because of the effect on their trade. A very powerful aristocracy who complained to higher authority whenever a municipal decision interfered with their plans or their pleasure did not help matters either.

A third factor was the resistance of the populace to any public health measures. The penalties for disobedience of statutes and regulations on this subject were often capital. The regulations of Visconte Bernabo in 1374 stated that the clergy were to examine the sick. Any clergyman who did not report the presence of bubonic plague and anyone who ministered to the sick without permission was to be burnt at the stake and all their goods were forfeit. In Edinburgh every pest house had its own gallows and in 1585 two men were hanged for stealing infected clothes. Earlier in the century a number of women were judicially drowned for failing to report the presence of plague. In London, in 1636, health officers who failed to enforce the regulations were threatened with Newgate.

These draconian measures probably stemmed both from the generally severe sentencing policy for trivial crimes in those days and from a desire to make the consequences of disobedience of the regulations more unpleasant than the results of obedience. It must have been very difficult to persuade people to admit to the presence of plague in their houses when the sick person would be carted off to a pest house with few comforts, little attention and even less of a chance of survival and his contacts would be immolated in their houses for a period without any means of earning a livelihood and having to survive, if they were lucky, on a meagre ration provided by the town.

A change of attitudes began to occur in the eighteenth century. This was heralded by the report commissioned from Richard Mead by the Secretary of State when there was a threat of the bubonic plague spreading to Britain from Marseilles in 1720. "A short discourse concerning pestilential contagion and the methods to be used to prevent it" ran to seven editions in the first year of publication and contained several revolutionary ideas. Firstly, it advocated that as plague was a disease of the poor who lived under dreadful conditions, if they were re-housed at public expense and their overcrowding reduced then there would be a less suitable environment in which the plague could spread. This was the first suggestion that a continuous measure should be taken against epidemic disease rather than the previously intermittent steps whenever a plague was imminent. Mead also put forward the idea that people should be encouraged to report the presence of plague by the payment of fees and the appointment of diligent and understanding men as searchers instead of old women. He thirdly suggested the appointment of officers of state (civil and ecclesiastical), magistrates, physicians and other responsible members of society to central Councils of Health to watch over the nation's wellbeing.

This advise was largely ignored for the next hundred years or so except that legislation to enforce his ideas on quarantine to contain bubonic plague and the removal of the sick to pest houses was passed only to be repealed by a different government a year later to make a petty political point.

The occasion of the adoption of Mead's ideas

was the cholera outbreak of 1831 which started in Sunderland. The response to the threat of cholera spreading to the rest of the country was similar to that seen in the previous five hundred years with the imposition of quarantine regulations and the segregation of the sick and convalescents. However a consultative Board of Health was set up with two non-medical and nine medical members. This board advised upon the measures to be taken. However this was soon supplanted by a central Board of Health with a ratio of lay to medically qualified members of two to one. This balance or imbalance may have been responsible for the definite change in policy manifest when this new body took over. The element of coercion was removed from the regulations and those groups at risk from the disease were identified by official inspectors. Measures to improve the nutrition and clothing of the lower classes and their temperance, the cleanliness, ventilation and space in their homes, and the methods of sewage disposal were taken.

At a local level, short lived Local Health Boards were set up to administer dispensaries and to educate the people to recognise the first symptoms of the cholera so that they would seek early treatment.

This change of emphasis from particular measures against a disease to the general was soon followed by a spate of horrific reports on the state of the working classes by Engels (1840), Chadwick (1842) and Littlejohn (1865) amongst others and the great public health movement, led by such as Sir John Simon, the the latter half of the nineteenth century.

What brought about this change of attitude is not clear. Cynics and Marxists would say that manufacturers realised that a healthy working class worked better and was less likely to rise in rebellion than a semi-destitute one. Sir John Simon thought that this change was due to the rise of humanitarianism in the eighteenth century and the influence of the Wesleys, Whitfield, Jeremy Bentham and John Howard. The truth probably lies somewhere between the two views.

Thus the history of the administrative measures taken against pestilence is a potted history of medicine as it reflects the medical dogmata of the time. The actions of civil authorities were appropriate to the supposed causal factors, be they supernatural intervention, the atmosphere or an invisible contagion. Usually they were not sure of the exact aetiology of epidemic disease and also took additional precautions which reflected previous but not entirely discredited ideas. It is also a brief history of administration and the development of local and central government and of specialised agencies. Thirdly it shows the development of man's feelings towards his fellow man. The medieval picture is one of a few selfless men, usually the humbler surgeons who were capable of working with the poor during an outbreak of an epidemic disease but of a much larger group of the merchantile, professional and monied classes who beat an undignified retreat whenever there was any threat. Then, throughout the eighteenth century the idea rose to the surface that the powerful had a responsibility to the powerless although there may have been some self-interest in that if the sinks of disease were removed there would be less change of pestilence overflowing from the slums into the drawing rooms. It was only when it was realised that trying to prevent a contagion entering a community where it would flourish was less efficacious than removing the factors which allow it to flourish that effective preventative measures could be implemented. The conquest of epidemic disease is more a success for humanitarianism than for therapeutics. ●●

HAEMOPHILIA, PAST AND PRESENT

by
Dr. S.H. Davies
lately Consultant Haematologist; Royal Infirmary of Edinburgh

INTRODUCTION

The term haemophilia, meaning "lover of blood", was coined comparatively recently in the long history of bleeding disorders, having been first used in the early 19th century. Then it defined a bleeding disorder which was transmitted by certain unaffected females to some of their sons. Now, as a result of the enormous increase in scientific knowledge developed in the interim, haemophilia can be defined more precisely as a coagulation disorder transmitted in a sex-linked recessive manner and primarily expressed in males, in which the level of factor VIII clotting (or biological) activity in the blood is reduced below normal because some of the precursor molecules, (named immunological factor VIII), are functionally abnormal and cannot be converted to clotting factor VIII. The clinical grade of severity of the disorder breeds true and correlates well with the amount of circulating clotting factor VIII, severely affected haemophiliacs having less than 1%, moderately affected between 1 and 5% and mildly affected between 5 and 50%. The normal range is 50 – 200%.

HAEMOPHILIA IN HISTORY

The first recorded references to haemophilia in man are probably to be found in Jewish writings of the 2nd Century A.D. where families are mentioned in which more than one male child died of excessive bleeding following ritual circumcision. The disorder also occurs in animals and probably first began in placental mammals through genetic mutation about 50 million years ago.

The severely affected patient bleeds 'spontaneously' in response to the 'microtrauma of living' and this grave gene deficiency was usually lethal in olden times before the reproductive age was reached. We therefore believe that the severe grade of the disorder was maintained in man by spontaneous genetic mutation and the mutation rate has been estimated at about 2×10^{-5} (W.H.O., 1972).

The history of haemophilia makes fascinating reading and the reader interested in a more detailed study of the subject is advised to read the excellent article by Ingram (1976). Apart from the methodic early clinical studies on the subject and the more recent scientific investigations recorded by Ingram, it is interesting to speculate on the role the disorder has played in European and Russian political history. Queen Victoria is thought to have acquired a carrier state at conception through genetic mutation, which was first recognised with the birth of her eighth child Leopold who was a severe haemophiliac. More significantly she produced at least two carrier daughters in her large family. One of these, Beatrice, transmitted the carrier status to her daughter Victoria Eugenie who married Alfonso XIII, King of Spain, and at least two of their five sons were severe haemophiliacs. Through Alix, whose daughter Alice married Tsar Nicholas II of Russia, was born in 1904 the severely affected haemophiliac, and only son, Alexis. The inability of doctors to prevent, or even alleviate, the repetitive painful joint bleedings associated with this disorder in the boy, may

have contributed to the evil influence that the monk Rasputin acquired over the unhappy Royal parents for the Tsarina had great faith in his healing powers. Isolated from everyday affairs by their preoccupation with their son's disability, the Royal parents may well have contributed to the tragic downfall and death of the family in the Bolshevik revolution. The drama of haemophilia is vividly portrayed in two novels by R.K. Massie ("Nicholas and Alexandra", 1968) and Dorothy Sayers ("Have his Carcase", 1932).

PRESENTATION AND NATURE

Classical haemophilia (factor VIII deficiency) has to be distinguished from von Willebrand's disease, and particularly from Christmas disease (factor IX deficiency) with both of which it shares some common clinical and laboratory features. The distinction, made as recently as 1952 in the case of Christmas disease, is important not least because modern clotting factor replacement therapy is reasonably specific for each disorder and the products used are not interchangeable.

The hallmark of a bleeding disorder is the persistence rather than the rate of blood loss. The typical haemophiliac is therefore a male in whom the clinical symptoms correlate well with the degree of his clotting factor deficiency. The severely affected patient, with less than 1% factor VIII, bleeds 'spontaneously', that is without apparent injury, the moderately affected in response to minor injury, but not spontaneously and the mildly affected in response to minor injury, but not spontaneously and the mildly affected only to moderate injury. More than 90% of bleedings in the severely affected patient occur into joints, and the knees, ankles and elbows are most frequently affected, though the reason why these particular joints should be involved is not known. The unpredictability of such bleedings in the severe haemophiliac with apparent normality in between episodes had a profound psychological effect on him in the days before modern factor replacement therapy became available to abort the bleeds in an early stage. Such joint bleeds, if untreated, produce intense pain and immobility and will eventually, when repeated sufficiently often, result in joint damage, permanent limitation of movement and finally crippling haemarthritic deformity. Less commonly occurring bleeds into muscles and tissue compartments can likewise, by their pressure effects, cause muscle necrosis or nerve damage and hence loss of function. Haemorrhage into a closed cavity such as the skull can cause brain damage and often death. Intracranial haemorrhage is now one of the commonest causes of death in this group of patients.

Classical haemophilia is the commonest of the single clotting factor disorders in Great Britain and has an approximate incidence of 6 per 100,000 in the population. About half of these patients are severely affected and the remainder are about equally distributed in the moderately and mildly affected grades. Whilst there is reason to believe that even some severely affected haemophiliacs have not yet been diagnosed and recorded, there is even stronger evidence that more of the moderately and mildly affected patients have escaped the net. These latter groups do not manifest their disability so obviously as the severely affected patients, particularly as the accepted blood loss due to trauma in normal males varies widely.

Christmas disease (factor IX deficiency) is about one fifth as common in the population as haemophilia.

MANAGEMENT

The factor VIII molecule is unstable in solution; it and factor V, only rarely congenitally deficient, are known as the 'labile' clotting factors. It has a half-life at blood heat *in vitro* and *in vivo* of 8 – 12 hours, whereas that of factor IX is 18 – 24 hours. It is ofen not appreciated that primary normal wound healing which, depending on the size of the wound, the presence or absence of infection and the nutritional state of the patient, may take up to 14 days to complete requires adequate levels of factor VIII and the other clotting factors in the blood throughout. Failure to meet these needs will result in immediate or delayed wound bleeding according to the time in the healing process that it occurs. The quantity of factors needed is roughly proportional to the size of the wound. Less explicable is the fact that 'spontaneous' injury needs less clotting factors for healing than does overt trauma of apparently similar degree. The mildly affected haemophiliac

who has some endogenous production of factor VIII can therefore cope unaided with mild trauma as can the moderately affected patient with minimal trauma, but the severely affected haemophiliac will often bleed abnormally in response to the occult trauma 'of being'. The principle of treatment of injury in these patients is to raise the deficient clotting factor to a safe blood level by exogenous means and to so maintain it until healing has been achieved.

In the 'spontaneous' joint bleed this may mean obtaining a 'once only' peak of 20% whereas for more severe trauma, for example operative surgery, a pre-operative level of at least 60% will usually be required, and thereafter the trough level must not fall below 20% until late in healing. To achieve this goal repetitive infusions of factor VIII as often as six-hourly may be necessary for much of the healing period. The greater the threat of re-bleeding, for example in intracranial injury, the longer is it wise to sustain a haemostatic level of the clotting factor in the blood. Meticulous surgical haemostasis together with wound rest by splintage etc. minimises factor VIII requirements and is always advocated in the management of injury in haemophilia.

The improved management of severe haemophilia is the result of four main factors: (i) precise diagnosis, (ii) early treatment of bleeding episodes, (iii) a better understanding of factory VIII requirements as discussed above and (iv) the availability of stable, concentrated preparations of clotting factor VIII. Clearly it is impossible to raise and maintain the factor VIII level of a severe haemophiliac from less than 1% to at least 60% with whole blood or even plasma without causing circulatory overload and heart failure. The availability of such concentrated factor VIII products has therefore revolutionised the management of haemophilia.

The History of Replacement Therapy

The history of blood transfusion in man is much shorter than that of haemophilia. It began in the 17th century, initially with the use of animal as well as human blood, and usually in either case with disastrous results to the patient. It was mainly due to the discovery, first by Landsteiner of the ABO blood groups, and then by Weiner of the Rhesus (Rh) groups, in the last fifty years that blood transfusion has become safe and practicable.

The management of bleeding battle casualties in World War II soon brought an appreciation of the urgent need for early restoration of the intravascular deficit by an isosmotic, colloid containing fluid even without red cells, at least until the loss of these became very severe. The separation of plasma from whole blood by centrifugation followed by the preparation of a dried, and hence stable, product met this need. Such a product was also easy to reconstitute and administer in forward battle areas, and did not require the skilled technical compatibility matching which is always desirable, and often essential, when whole blood is used. By this time Macfarlane in Oxford and other workers elsewhere, had also realised that only intravenous factor replacement therapy would satisfactorily and safely control bleeding in the haemophiliac.

The knowledge gained during the War years in the preparation, handling and use of plasma stimulated further exploration of this product for its more specific component fractions. This had two main objects, namely (i) to produce more specific products of high potency which would make possible sustained, intensive replacement therapy in patients, and (ii) to make more economic use of blood which, in Great Britain, is obtained from voluntary donors who have to be allowed a minimum period of four months to make good the deficit produced by the giving of one whole blood donation.

Factor VIII requirements at that stage, because of the instability of the molecule *in vitro*, were best met by using fresh plasma (within no more than four hours of blood donation) which was inconvenient. However, the factor VIII activity in such plasma could be preserved for at least three months either by immediately deep freezing it to not less than -20° C or by freeze drying it.

Unfortunately neither of these measures solved the problem of the unacceptable volume it was necessary to transfuse to achieve and maintain an adequate haemostatic level of factor VIII in a severe haemophiliac for other than trivial bleeds.

The next logical sequence was plasma fractionation. After the pioneer work in this field of Cohn et al (1946), crude concentrates of human

and animal products were produced in various European and American centres in the early 1950's and used with increasing success in the management of the more serious and major bleeding episodes in haemophiliacs whether they were spontaneous or traumatic, accidental or elective in origin. The species specificity of the highly potent concentrates obtained from the pig and the cow limited their use in man because of the sensitisation and thence severe reactions they eventually induced in the recipient, but they fulfilled an important interim role where large dosage of factor VIII was needed until the human product could be adequately refined. This latter achievement dates from a chance observation made in America by Judith Pool and her colleagues (1965). She noticed that when human plasma was deep frozen at -20°C and then slowly thawed, the large plasma protein molecules, factor VIII (m. wt about 2×10^6) and to a lesser degree fibrinogen (factor I, m. wt 560,000), would at 4°C remain for a time as a sludge or cryoprecipitate whilst the other plasma proteins went into solution. By careful removal of most of the liquid supernatant the sludge could be harvested and then stored stable deep frozen at not less than -20°C in the little remaining plasma until required for use. It could then be dissolved as required in the retained plasma by controlled thawing at blood heat (37°C) and injected intravenously at once. Comparatively high doses of factor VIII could thus be obtained in an acceptably small plasma volume by pooling as many of these cryoprecipitate donations as were necessary.

The theoretical and practical disadvantages of such cryoprecipitate were (i) the time consuming skill needed in preparation of each individual pack, (ii) the variability of factor VIII levels in the normal donors and the manipulative loss in preparation, (iii) the need for controlled temperature thawing (37°C) to avoid factor VIII loss immediately prior to use and (iv) the need to enter several packs with its attendant infection risk to obtain a sufficient dose of factor VIII for therapeutic use.

On the other hand despite the technical, organisational and economic difficulties of harvesting large quantities of whole blood, separating and deep freezing the plasma immediately, storing and transporting it deep frozen to a central large scale fractionation unit, and the technical difficulties of large scale fractionation without loss of potency of the components, cryoprecipitate has been an important interim advance in the treatment of haemophilia and it still remains so today. Nevertheless, at various centres in Britain (Oxford, Elstree, Edinburgh) large scale plasma fractionation is a reality and the quality and quantity of the products available, not least of factor VIII, are increasing and in some cases (e.g. factor IX) do meet all the present day requirements. The freeze dried factor VIII so produced has many advantages over the stalwart cryoprecipitate. First, it is produced from a large, pooled, plasma batch and equal aliquots of the finished product are freeze dried in individual vials; thus the dose is constant in each vial from any batch. Secondly, by standard random sampling the vials can be checked for potency and sterility and duly labelled with this information. Thirdly, the product is stable for a considerable time if kept at 4°C, and it can be reconstituted in a small volume of sterile water immediately prior to use to give a known factor VIII dose which usually has a potency of at least twenty times that of an equal volume of fresh plasma.

The present aim is to produce dried products of ever greater potency with acceptable recovery loss in manufacture and in such quantities that they will meet *all* needs of haemophiliacs for factor VIII.

Management today

How has the management of haemophilia changed and kept pace with the blood product development? First, it has become more aggressive; that is treatment of spontaneous bleeding, commonly into joints, is given earlier and is more effective so that bleeding is arrested before distention and pain occur, and temporary disability, permanent damage and deformity are minimised or prevented. This results in less time lost from school, work or leisure activities. Secondly, major surgery, whether for disorders that may beset us all, such as the complications of peptic ulcer or extensive dental caries, or for those peculiar to the haemophiliac, such as joint deformities from past haemarthroses, has been made much safer and can now be undertaken electively and not only as a desperate life-saving

measure. For these aims to achieve fruition other associated developments have been necessary. Haemophilia Centres have been established and developed in major hospitals so that precise diagnoses can be made and patients counselled, advised and instructed. At these Centres specialised out-patient treatment is available on a 24-hour basis and the haemophiliac can report direct to the Centre using the Ambulance Service or his own 'car for the disabled' to get there. Joint bleeds etc. can thus be treated at a very early stage and the haemophiliac, if sufficiently competent, can be taught self-injection of the factor VIII made available at the Centre 'on demand'. When he is fully trained, and when adequate dried factor VIII supplies are available, he can change to the Home Treatment programme. He will then be issued with a limited supply of dried factor VIII with which he can treat joint bleeds – the bugbear of the severe haemophiliac's life – at home and thus save time and trouble and gain independence. Since 95% of all factor VIII used by a severe haemophiliac is for such bleeds, the advantages of home therapy are obvious. For bleeds other than into joints, out-patient management at the Centre, or occasionally in the Ward, is still advisable and the haemophiliac must not attempt such treatment at home.

The future

Whilst present progress has revolutionised the life of the severe haemophiliac, given him self-confidence and a much greater degree of independence, it has not cured him. At present we know of no means of achieving this. Perhaps as supplies of factor VIII improve still further, we will be able to offer him prophylaxis, but the short factor half-life would probably necessitate twice daily self-infusion of factor VIII rather like an insulin-dependent diabetic though using the intravenous route. However, about 10% of haemophiliacs develop a neutralising 'antibody' to factor VIII which is not dose related, and at present is causally not understood. In some it persists in the plasma indefinitely and in others it wanes if not provoked by further therapy, but in all it constitutes a bar to replacement therapy, either totally or save in urgent circumstances. Management of these patients is at present not very satisfactory even though blood products with 'factor VIII bypassing activity' have some beneficial use. We can, however, hope for a radically different approach to the problem of haemophilia. It is possible even now to sex the foetus *in utero* accurately in early pregnancy, and where the mother is a known or potential carrier of the disorder this may be advisable; however, the procedure is associated with a small abortion loss. Hopefully in the near future it will be possible to measure the factor VIII level in the foetus accurately and thus offer therapeutic abortion of an affected male foetus if desired.

The investigation of suspected female carriers of haemophilia is becoming more specific, based upon the dissociation of the levels of clotting and immunological factor VIII in the blood of those positive for the trait, but it is not yet wholly reliable. An entirely new approach to the management of haemophilia depends upon using an exogenous stimulus to induce a temporary increase in endogenous production of factor VIII. Deamino-D-arginine vasopressin (DDAVP) given intravenously or as snuff is used for this purpose but is effective only in the moderately or mildly affected patient (Mannucci et al, 1975). And so the search for better products, more effective and simpler treatment, prophylactic rather than therapeutic, goes on. Selective abortion as a preventative measure is never likely to overcome the perpetuation of the disorder through spontaneous genetic mutation and hence the goal must ultimately be to find a cure. This still seems to be a long way off, but progress towards it accelerates.

The Haemophilia Society

What have haemophiliacs done to help themselves? In the early 1940's in Britain they formed their own Haemophilia Society and this is now linked with other similar societies elsewhere in the world in the World Federation of Haemophilia. Through their Society, and as a minority group in the population, they have helped to dispel fear and ignorance of the disease and this has improved their opportunities for better medical care, schooling, employment and leisure. The Society has, through organised meetings and its literature, helped parents of newly diagnosed haemophiliacs and the effected children themselves, to adjust and adapt to the disability and to lead a fuller, more balanced life. It has provided funds for research and has agitated

and helped to make Governments understand the needs of all handicapped people, and of those with haemophilia in particular.

And so whilst haemophilia and its problems are so much better understood and managed than in those far off days of the Rabbinic references to exsanguination of ritual circumcision, there is still much to be done and the contributions made in this field in the future, as in the past, will add to as much as they derive from, the advances made in medicine in their widest sense. ●●

REFERENCES

Cohn E.J., Strong L.E., Hughes W.L., Mulford D.J., Ashworth J.N., Melin M. and Taylor H.L. (1946) A system for the separation into fractions of the protein and lipoprotein components of biological tissues and fluids. J. Amer. Chem. Soc., 68, 459.

Ingram G.I.C. (1976) The History of Haemophilia. J. Clin. Path. 29, 469-479.

Mannucci P.M., Aberg M., Nilsson I.M. and Robertson B. (1975) Mechanism of plasminogen activator and factor VIII increase after vasoactive drugs. Brit. J. Haemat., 30, 81-93.

Massie R.K. (1968) Nicholas and Alexandra, Gollancz. London.

Pool J.G. and Shannon A.E. (1965) Production of high potency concentrates of antihaemophilic globulin in a closed-bag system. New. Eng. J. Med., 273. 1443.

Sayers D.L. (1932) Have his Carcase. Gollancz. London.

World Health Organisation (1972) Inherited blood clotting disorders. Wld. Hlth. Org. Techn. Rep. Ser., No. 504, p.14.

ON BEING A PROFESSIONAL MEDICAL WITNESS

by
Professor J.K. Mason, CBE, MD, DCP, DMJ, FRC.Path, DTM & H
Professor of Forensic Medicine, University of Edinburgh

When asked by the Editor to contribute an article on this topic, I became conscious of the fact that as a result of my arthritic leg which has defied the best efforts of clinical colleagues to heal, my recent Court appearances have been few and far between. Perhaps this means that I can take a relatively detached view of the Court rather than one dominated by current attacks of hyper-adrenalinism.

I doubt if there are many people called to give professional medical evidence who approach the witness box without some quickening of the heartbeat. So if it happens to you, you can at least feel you are in good company. But it is the essence of a good witness that he is free of emotion, and this state depends firstly upon experience, and secondly on one's preparation of material before the trial.

This preparation begins with the reception of the patient. The great majority of cases in which the recently qualified doctor is likely to appear in Court involve trauma and most are due to accident, particularly road traffic accidents — in fact, just the sort of case in which there is least time for reflection and, indeed, most likely to be turmoil during the phase of admission and initial treatment. Nothing must be allowed to interfere with the doctor's true function — the saving of life and the reduction of morbidity — but, if there is time for consideration, it must be remembered that the original findings are going to be of primary evidential importance in any later Court proceedings. If the patient dies, there is bound to be a Fatal Accident Inquiry (or, in England, a Coroner's Inquest with jury) in the case of industrial accident, there may be very serious criminal charges following a vehicular accident, and in a case of apparent deliberate wounding, a man or woman may be on trial for his or her liberty for many years to come. In short, it is not just one's own position as a witness which is in jeopardy but the reputation and living of others. So the first step to a successful Court appearance is the making of accurate notes in all relevant cases. Unless a patient is bleeding to death, it is not a great problem to measure a wound or to make a simple diagram of an abrasion; it is not difficult to incorporate in the notes items which are not strictly relevant medically — e.g. was there any obvious paint around the wound? These notes can be backed up by the retention of items which may form valuable forensic scientific evidence. Thus, particularly in cases of criminal assult, it may be important to preserve any foreign bodies discovered within a wound. Similarly, the clothing should be retained in such cases, most especially if there is a sexual element to the assault; the fact that the clothes are torn, blood-stained and muddy may be an extra good reason for their preservation — not destruction.

There are two additional points about the notes which I would make here. Firstly, it may well be that when the case comes to Court, it is not the obvious medical features which are of main importance — the fact that a man had received a stab wound may be accepted by all parties — but were there bruises on the knuckles indicating that he had been fighting? In other words, so far as is possible, the notes must be comprehensive,

not limited to the primary centre of medical interest. It follows from this that there may be a lot to remember, and no matter what you feel at this time to the contrary, these are easily forgotten details. So the second point to be stressed is that the notes should be written up as soon as possible. One appropriate example concerned a very famous forensic pathologist who gave his opinion as to the time of death in a murder trial; he was asked what he measured the rectal temperature of the body to be and then had to admit that he had no note of it and could not remember! Your notes made at the time may not be very good and you may want to alter them in the clear light of day. However, it is very important from the point of view of a future witness that these are clearly annotated – e.g. "corrected on 1.2.79." or "added on 2.3.79.": significant alterations must never by made after the result is known.

The hospital notes on a patient may be of considerable length; all of them may be required in a later hearing, so it is important when writing them up, to think what they will look and sound like in a criminal or civil court, at a fatal accident inquiry or in a Coroner's court. Don't make judgements. In the incredibly unlikely event of such a thing happening, let the second operation note simply say "a pair of scissors was removed from the pelvic cavity" and not "a pair of scissors which had been negligently left there by Mr. X...." – somebody else will determine that. If a patient is being extremely difficult, don't describe her in the notes as "this dreadful woman" – it can only be an embarrassment when uttered publicly. By and large, chatty remarks seem rather out of place when read out in supercilious monotone. Back in the War (Second World – but I was pretty young at the time) I had a tented sick quarters and what appeared to be an intractable psychoneurotic patient (actually, you were allowed in those days to use the term N.Y.D.N. – which meant "not yet diagnosed – nerves"!). I arranged to evacuate him to a civilian base hospital and did so with a friendly valediction in his notes – "sorry to inflict this snag case on you". It sounded far less of a pleasantry when a coroner's inquiry some weeks later established that he had died following malarial treatment of neurosyphilis! Remember that the precise wording of your notes can rebound on you even though they are in no sense derogatory or inaccurate. More recently, I wrote a report on the examination of a girl in a potential case of rape and said something to the effect "the injuries are compatible with aggressive love-making". Counsel for the defence saw his chance; "We would, of course, bow to your experience in the field", he said with a wide grin on his face, "but perhaps you would give My Lord and Jury your definition of aggressive love-making".

Which prompts me to disabuse you of one popular misconception. Contrary to what you read in books, no Counsel is "out to get" an honest professional witness. In the great majority of cases, the Court wants no more than assistance and to hear a considered opinion from someone fully conversant with the facts. The fire and brimstone impression that persists stems, I think, from the famous days of the evolution of modern British forensic pathology. In the early part of the century, pre-meditated murder was at its height, but forensic medical experts were few and far between. They tended to be very partisan and to regard their evidence as something like the fruits of a visit to Mount Sinai. It was very seldom that an expert of similar calibre was available to oppose the main medical opinion, and as a result, Counsel were forced into forensic chicanery. These conditions do not apply today and the professional man is assured of a courteous hearing provided he does not destroy his own image. So, what are some of the things which have a bearing on this relationship between the witness and the Court?

First, don't be late. Everyone admits that a lot of time is wasted in the witness waiting room but you cannot depend on a delay. Most Courts do their best to clear the doctors early in the case, and if your time is limited the Fiscal will always try to help.

The second general point is equally obvious – do be dressed for the part. Most judges and jurors are fairly "square" and past the first flush of youth; they like their doctors to be of conventional appearance and will probably vaguely distrust the evidence of one who does not come up to expectations. First impressions *are* important.

It goes without saying that the case should have been properly prepared. This will depend on the

type of evidence you are being asked to give – and this will have become clear in precognition – but if you are to be asked about the causation or management of a surgical or medical condition, it is well to be aware of the alternative opinions to your own. There is a tendency to do a literature search for articles favourable to one's own opinion; but the medical advisers to other interested parties will be able to quote from the other side of the page and one must be ready for it. Lawyers love books so I would suggest having a look to see if there is anything relevant to your case in the more widely used text-books of forensic medicine – Glaister (1) is by far the most common reference in Scotland and Simpson (2) in England.

Don't get angry with Counsel – or at any rate, don't let your irritation cloud your judgement and show in public. It is worth reflecting, that if the lawyer is being obtuse, it may well be because your answers are not clear enough to a non-medical man; we are brought up on medical jargon and often find it difficult to appreciate how very little is understood by the lay-man. Above all, don't try to make the Advocate look silly. The temptation to attract a little "laughter in Court" must be resisted, except, perhaps, when it is to no-one's disadvantage. By and large, lawyers have a quicker and more acid wit than doctors who seldom come off best in such exchanges. A stupid mistake on the lawyer's part – and there are very few of them – can be pointed out with deference; a soft answer at least does not attract wrath.

Finally, there is the well known advice to all witnesses to confine oneself to answering the question and to avoid making a speech. By the same token, do not be cajoled into giving opinions on conditions outside your experience. There is a story of the great pathologist, Keith Simpson, being led into giving a clinical assessment of a case; Counsel casually asked, "Tell me, Doctor, when did you last see a live patient?" – the reply must have had the effect of a counter offensive, but it illustrates what I mean. On the other hand, don't be afraid to give a full answer, particularly if it involves several points of importance. You may well expect Counsel to follow up your line of thinking and then be surprised when he does not. Few things are more annoying than finishing the game with an unplayed ace in one's hand and it may be more than just annoying to oneself – justice may have been impeded.

So, dealing with a well prepared and well presented case, you will find that the time has passed quickly and you may be surprised when the Sheriff or Judge says "Thank you, you may be excused now"; looking back on it, the experience has not been nearly so bad as you anticipated. And when it's all over? Well, don't forget to claim your witness fee – it is unlikely that anyone will remind you.

(1) Rentoul, E, and Smith, H. (eds.) (1973) "Glaister's Medical Jurisprudence and Toxicology", 13th edn., Edinburgh and London: Churchill Livingstone.

(2) Simpson, K. (1979) "Forensic Medicine", 8th edn., London: Edward Arnold.

(1) Rentoul, E, and Smith, H. (eds.) (1973) "Glaister's Medical Jurisprudence and Toxicology", 13th edn., Edinburgh and London: Churchill Livingstone.

(2) Simpson, K. (1979) "Forensic Medicine", 8th edn., London: Edward Arnold.

FETAL PATHOLOGY IN SPONTANEOUS ABORTION

by
Jeanne E. Bell B.Sc., M.D.
Senior Registrar: Dept. of Pathology: Royal Hospital for Sick Children

Pregnancy is a truly remarkable process. While a single fertilised cell is developing into a complete individual, the mother usually tolerates this rapidly growing "transplant", her enlarging uterus and the accompanying dramatic hormonal changes with equanimity. Perhaps it is hardly surprising that such a complex process sometimes fails.

Changing times have influenced the degree of importance society attaches to fetal and perinatal loss. It is not very long since recurrent pregnancy, high infant mortality and stillbirth were accepted with resignation, as normal hazards of living. However with effective contraception, careful obstetric supervision and the improvements in nutrition, hygiene and health which have come to affluent western societies, the present expectation is of a chosen number of pregnancies, precisely timed and with perfect outcome. Yet about one in ten couples is infertile; nearly one in five recognised pregnancies ends in spontaneous abortion[1] and an even greater number of unrecognised early pregnancies are aborted[2]. Fetal loss is still a common problem.

A major breakthrough in the study of human reproductive wastage resulted from successful cytogenetic studies of aborted material. The normal diploid human chromosome number is 46 with an XX (female) and XY (male) mechanism determining sex. At least half of all *early* spontaneous abortions have detectable chromosome abnormalities incompatible with the normal development of the embryo[1]. Abnormalities commonly detected in these abortuses include trisomy (with a single extra chromosome), monosomy (in which one homologue of a pair is missing) and polyploidy, particularly triploidy, with one additional haploid set of chromosomes, and tetraploidy with two sets. Monosomy appears to be almost exclusively restricted to loss of an X chromosome whereas trisomy has been reported for every autosome. Mosaic individuals, with more than one cell population, can be found.

The classical descriptions of human abortuses were made without the benefit of this knowledge[2,3]. Even today, the way in which a chromosome abnormality exerts its phenotypic effect or contributes to abortion at a particular stage of pregnancy is not understood and remains a challenge jointly to cytogeneticists and pathologists.

The pathology of spontaneous abortion is also interesting. Complete "products of conception" comprise both fetal and maternal tissues, particularly decidua. Most of the tissue expelled in early spontaneous abortion is actually maternal in origin. In pregnancies ending at about 12 weeks of gestation, it is quite usual to find that fetal development has failed almost totally. In these cases the intact gestational sac is very much smaller than normal and contains only fluid or a small embryonic knob (Fig. 1). The general term,

"blighted ovum", is sometimes used to describe sacs which do not contain an organised embryo. These sacs are usually accompanied by a large quantity of decidua or by a complete decidual cast of the uterine cavity (Fig. 1). Unfortunately, many spontaneous abortions commence in the mother's own home and the tissue received for pathological examination is only the retained decidua which has been removed by curettage. This presents problems for the cytogeneticist since the accurate separation of fetal from maternal tissues is essential if chromosome analysis is to be of clinical value.

Apart from the small sacs just described, there are many other cases of spontaneous abortion in which the fetus is not of a size appropriate to the reputed length of gestation. These unduly small embryos and fetuses often show varying degrees of maceration, indicating that fetal death occurred some time before the onset of labour (Fig. 2). Maceration takes place at body temperature and usually in a sterile environment, so that body form is often retained sufficiently well to allow recognition of major structural abnormalities. The investigation of smaller (or particularly interesting) embryos is best achieved using a dissecting microscope and sometimes even serial sections.

Both fresh and macerated aborted fetuses include some individuals with no apparent structural abnormality, some with minor and some with major abnormality. Examples of clearly defined defects, compatible with post-natal life and identified in spontaneous abortuses, are hypospadias, cleft lip, Meckel's diverticulum, polydactyly, double ureter (Fig. 3) and bicornuate uterus. More severe abnormalities include facial cleft, diaphragmatic hernia, coarctation of the aorta (Fig. 4), cardiac septal defects, pulmonary hypoplasia (Fig. 4), polycystic kidneys, anal atresia, and central nervous system malformations of all kinds. Of special interest are the cases where patterns or associations of abnormalities occur. Less easy to classify are those abortions in which the fetus has an odd appearance but is apparently morphologically and chromosomally normal. These may have subtle anatomical and metabolic abnormalities that elude us at present.

Table 1 summarises the pathological classification of more than 1,300 spontaneous abortions examined in Edinburgh over a period of five years.

Table 1 Pathological Findings in 1300 cases of Spontaneous Abortion

	Percentage of Total
Twelve weeks of gestation and less (62% of total)	
Decidua and ruptured sacs	28
Intact empty sacs	9
Intact sac with embryonic knob	9
Fresh normal embryo	1
Fresh abnormal embryo	7
Macerated embryo	8
Greater than twelve weeks of gestation (35% of total)	
Fresh apparently normal fetus	6
Fresh abnormal fetus	17
Macerated apparently normal fetus	6
Macerated abnormal fetus	6
Others (e.g. Placenta only)	3
	100

Why do so many pregnancies end in spontaneous abortion? To suggest that this is simply nature's way of eliminating developmental errors discounts both the apparently normal aborted fetus and on the other hand, the many babies surviving to term with major defects. If a screening mechanism for fetal defects does exist, whereby the mother rejects abnormal pregnancies, then it is probably most efficient in detecting the chromosomal abnormalities which are present in so many early abortions

The study of central nervous system (CNS) malformations shows that if selective spontaneous abortion does occur, then it is far from perfect. Before the antenatal detection of CNS defects became possible, with the accompanying option of termination of affected pregnancies, the birth incidence of spina bifida and anencephaly in Britain was about 0.4%. These severe structural abnormalities had not triggered an abortion mechanism in spite of being present from an early stage of gestation. However, CNS defects are found with an incidence of 4% in spontaneous abortuses (personal observation) especially among the small embryos where they may escape detection on a cursory glance (Fig. 5). This is a tenfold increase over the incidence at birth prior to antenatal diagnosis. If comparable figures apply for other congenital defects, this would suggest that despite the number of such defects seen in babies at term, spontaneous abortion does serve

a useful purpose in eliminating a majority of the abnormal conceptions.

The identification of a CNS defect (Fig. 5) in a spontaneously aborted embryo gives rise to a problem. Is the mother of that embryo at the same increased risk of recurrent defect as the mother of a baby born at term with a CNS abnormality? In fact, we know remarkably little about the implications for the subsequent pregnancy, after a spontaneously aborted abnormal fetus, except where the abnormality originates from a cytogenetic error. We are involved at present in a prospective study of pregnancies subsequent to spontaneous abortion. Meanwhile, it seems unwise to regard a spontaneous abortion as a negligible obstetric event. However, the findings in aborted material may well prompt appropriate investigation or counselling or simply reassurance of the parents. For example, ultrasound examination and alphafetoprotein estimation should be recommended at present in any pregnancy subsequent to the abortion of a CNS defective embryo.

Apart from the possible benefits to the mother and her future children, is anything else to be gained from a study of spontaneous abortion? It does provide opportunities to investigate the pathogenesis of defects in early human development, when the original pathology is not obscured by infection, by attempts at regeneration or by surgical intervention. Unfortunately maceration sometimes interferes with histological assessment. Because the time interval is shorter than in a full term pregnancy, maternal recall may well be more accurate in respect of illness, drugs, X-irradiation and other factors operating early in pregnancy, making retrospective epidemiological enquiry more profitable.

How is progress to be made in the understanding and prevention of fetal disease? I have neglected many important factors which may cause problems for the developing fetus. Since the fetus is totally dependent on its mother, some aspects of fetal pathology are clearly a reflection of maternal pathology, (as in maternal diabetes) or of the interaction between mother and fetus (as in rhesus incompatibility). I have concentrated on the congenital malformations because we have made little headway in their understanding or prevention, while perinatal mortality from other causes has declined.

Animal experiments have contributed greatly to the study of abnormal fetal development. The most important conclusion, confirmed by numerous studies, is that it is the timing rather than the nature of the experimental insult which results in a particular defect. Totally unrelated agents administered separately at a given time in gestation may produce the same defect. Conversely, one given agent may produce different defects if administered at different stages of gestation. This may have important implications for the aetiology of human malformation but caution is needed when animal development is compared directly with that of man.

Undoubtedly the possible antenatal detection of inborn errors, chromosomal abnormality and morphological defects has revolutionised the care of high risk patients and enabled many couples to achieve a successful pregnancy after repeated failures, while the birth of individuals who would have been an almost intolerable burden on the family and society has been reduced.

However, these advances generate problems. The methods of antenatal diagnosis must be perfected to avoid elimination of normal fetuses. It is a drastic step to "kill the patient to cure his disease" as Warkany[4] has put it. If the relation between spontaneous abortion and fetal defect were better understood, we might be able to increase the efficiency of early spontaneous elimination thus avoiding active intervention later in pregnancy. Even if we accept that there may not be an early alternative to antenatal detection and selective abortion, let us continue to strive for understanding of congenital defects and to make the best use of the knowledge we have. While drugs, nicotine and excess alcohol are all viewed with suspicion, we still have difficulty in protecting pregnant women from those agents, such as rubella virus, which are known to be teratogenic.

We should also be alert for the less obvious forms of fetal pathology. Until recently, the fetal alcohol syndrome may have been missed precisely because the abnormality in the affected fetus is only slight (until mental deficiency becomes

obvious in post natal life). Intrauterine growth retardation, resulting in small-for-dates babies, may be another preventable handicap which impairs many individuals. Responsibility falls heavily on all those caring for the pregnant woman and her fetus and on those engaged in the related research fields.

It is encouraging that we now have signals, such as protein and enzyme levels in maternal serum, which indicate the possibility of fetal maldevelopment or distress even when the pregnancy appears to be progressing normally. I hope it is not unrealistic to believe that more of these will be discovered thus allowing active intervention to prevent further fetal deterioration in progressive disease, or to correct abnormality already present, while conserving the pregnancy.

●●

REFERENCES

1. BOUÉ, J. PHILIPPE, E. GIROUD, A. & BOUÉ, A. (1976)
Phenotypic expression of lethal chromosome anomalies in human abortuses.
Teratology 14: 3-20.

2. HERTIG, A.T. ROCK, J. ADAMS, E.C. & MENKIN, M.C. (1959)
Thirty-four fertilised human ova, good bad and indifferent, recovered from 210 women of known fertility.
Pediatrics 23: 202-211.

3. MALL, F.P. & MEYER, A.W. (1921)
Studies on abortuses: A survey of pathological ova in the Carnegie embryological collection.
Cont. Embryol. 12, No. 56.

4. WARKANY, J. (1979)
"Quo Vadis" Unpublished observations at the 7th European Teratology Society Meeting. Herzlia, Israel.

LEGENDS

Fig. 1 – Decidual cast with opened gestational sac containing opaque knob of embryonic tissue.

x1

Fig. 2 — Macerated fetus, C.R. length 7cm., attached to placenta. There is an amniotic connection between the cord and the left hand.
x1

Fig. 3 — Dissection of posterior abdominal wall shows left double ureter. Meconium is spilling from the cut end of the colon. Normal fetal lobulation of the kidneys is shown.
x2.3

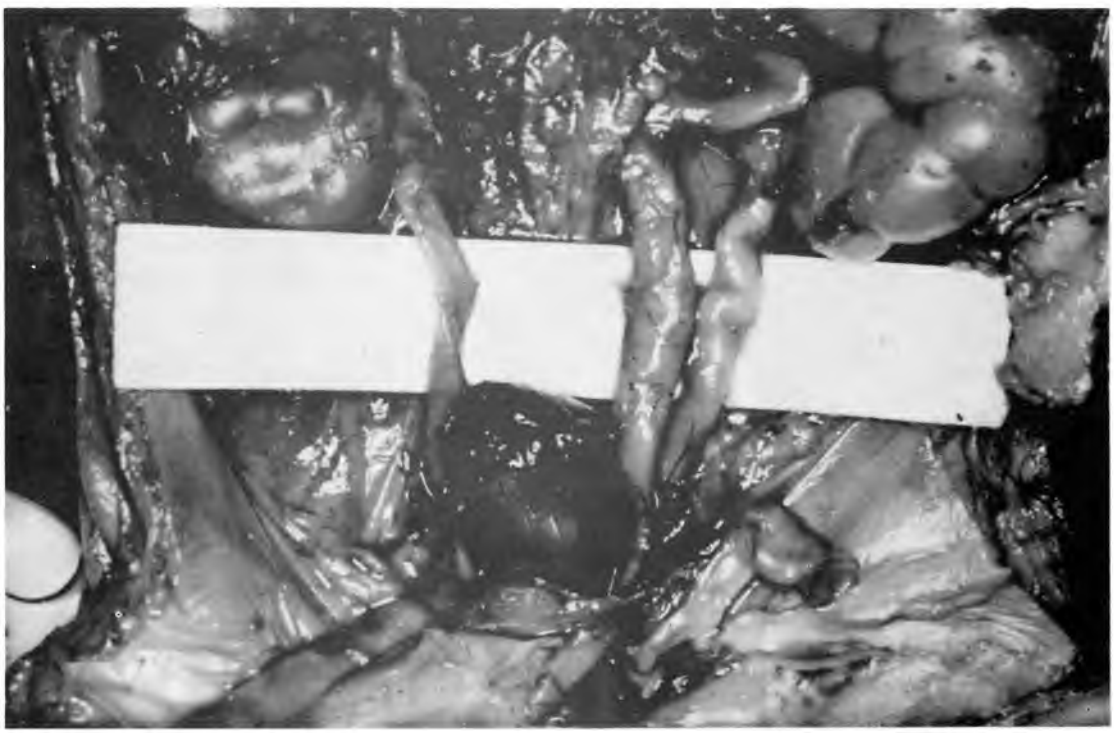


Fig. 4 — Dissection of thorax in a macerated fetus showing pulmonary hypoplasia (marked by pins) and coarctation of the transverse aorta.
x2

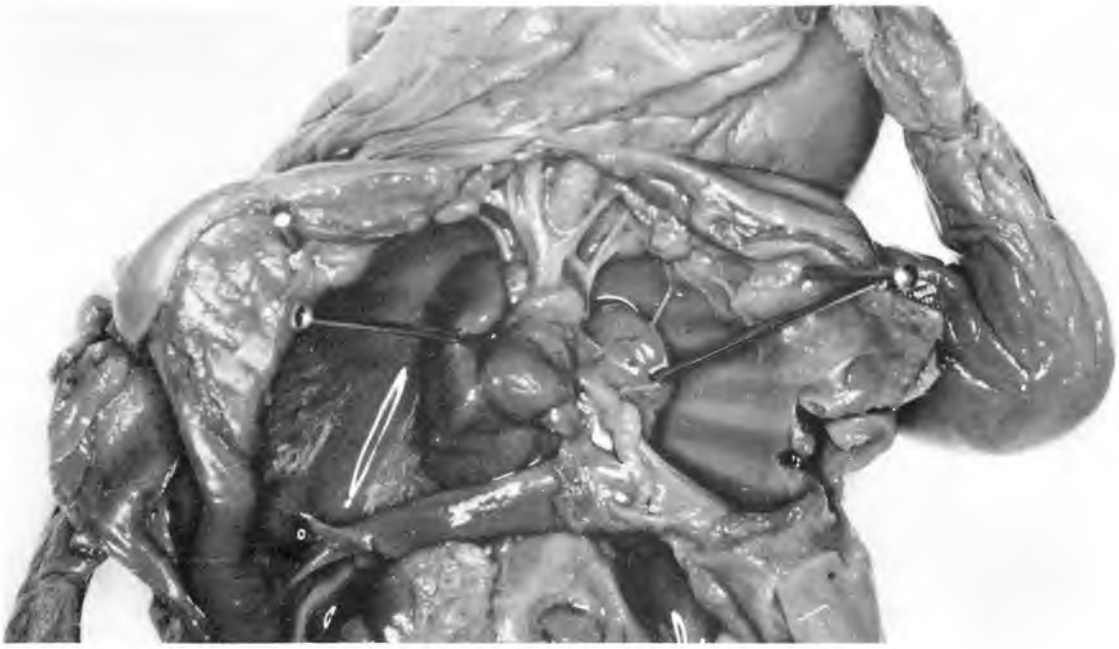
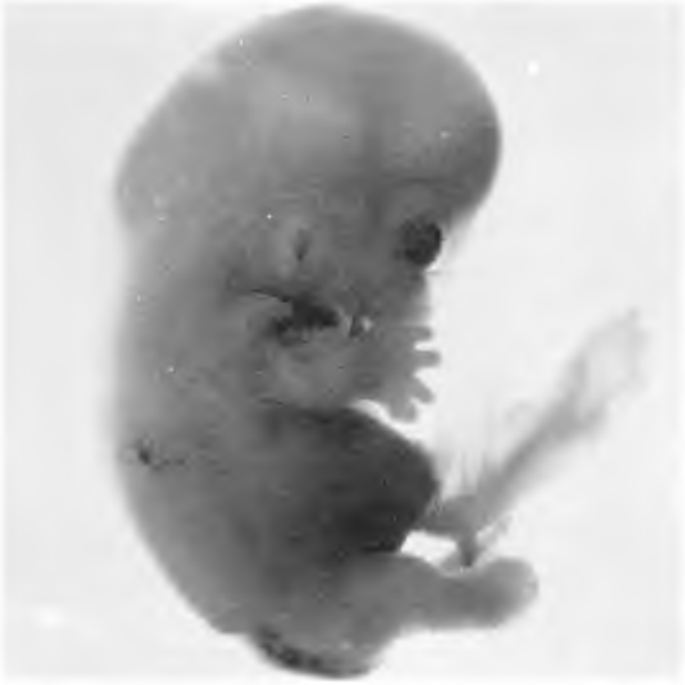


Fig. 5 — Slightly macerated embryo with lumbo-sacral spina fibida. The only other visible abnormality is polydactyly (extra digit attached to the ulnar side of the hand).
x4



THE DIVING CASUALTY, AETIOLOGY AND MANAGEMENT

by
Charles V. Clark, B.Sc.

Adapted and revised from a dissertation read before the Society
on
Wednesday 1st February 1978

The logarithmic increase in diving, both professional and amateur, is paralleled by an unprecedented rise in related morbidity and morality. Improved breathing apparatus and protective clothing are enabling man to remain in water longer and to dive deeper than ever before. Many diving accidents are untreatable owing to the circumstances in which they occur: in a modern saturation diving system it may be impossible to bring a diver back to atomspheric pressure in less than several days – whatever the medical emergency. However, an increasing number of fatalities are occurring due to lack of knowledge of the basic signs and symptoms which a diver, surfacing obviously with great difficulty, may present.

This dissertation is an attempt to review and collate current work and knowledge of the medical problems presented by the underwater environment.

1. HAZARDS OF THE UNDERWATER ENVIRONMENT

Compression Barotrauma: barotrauma may occur on descent when external pressure, in accordance with Boyle's Law, acts on the gas-filled cavities of the body to compress their contained gas. Thus compensation is required either by the introduction of additional compressed gas or by replacement with tissue or tissue fluids. An effect produced by pressure change is that of "squeeze". Should a diver descend rapidly – for example as a result of falling from an underwater platform – he may travel a great distance through the water before being checked by his lifeline or air-pipe. Thus although pressure within the respiratory system remains at the level of the working platform, external environmental pressure rises rapidly. The increase in external pressure and proportionate decrease in air volume, if there is a large pressure differential, will cause pulmonary oedema and haemorrhage. Other surfaces exposed to air will be subject to similar effects, and the pulmonary manifestations are often accompanied by subconjunctival haemorrhage and oedema. The resultant damage is more extensive, and the condition more dangerous, the nearer to the surface the fall occurs because of the greater relative pressure change.

Pulmonary Barotrauma: if an ascent is undertaken with lungs fully expanded, and air release is inhibited, then as surrounding pressures fall, the enclosed air will expand the lungs. This may tear lung tissue, and air may then leak into the connective tissues of the mediastinum. The interstitial emphysema so produced may rupture into the pleural cavity with the acute onset of pneumothorax. If, as is often the case, air is drawn into the pulmonary circulation, air emobli are produced.

Air Embolism: the most serious presentation is loss of consciousness, although convulsions, visual changes and spastic or flaccid paralyses are quite common, while less dramatic symptoms include vertigo and tingling in the extremities. Treatment of this condition is usually by immediate recompression of the patient: pressure both causes

much of the emoblism to dissolve in the blood, and also causes it to shrink until it may slip past the point of obstruction. Obviously the patient must be repressurised within seconds if treatment is to be effective. Prolonged, slow decompression may be required to prevent the embolus reforming. Following decompression, the patient should be admitted to hospital for medical examination, and an E.E.G. immediately following admission may indicate a focal intracranial lesion. A lung scan should be performed, as this tends to be more informative than straight chest X-ray.

Pneumothorax and Interstitial Emphysema: the patient may complain of pain in the chest or abdomen upon leaving the water. This is commonly accompanied by a cough, epistaxis or slight haemoptysis. Treatment of these conditions presents a different problem. The interstitial emphysema is usually retrosternal and untreated but pneumothorax is drained through an underwater seal.

In the treatment of pneumothorax or interstitial emphysema, because of the possibility of air embolism, full emergency treatment must be given in all cases. If recompression is unavailable, the patient should be placed in either the left lateral or prone position with the body sloping such that the head is kept low, in an attempt to prevent any possible air emboli entering either the cerebral or the coronary circulations. The incidence of burst lung would be reduced by preventing from diving all those with existing lung pathology, for example emphysema or adhesions. If the subject is suffering from an attack of asthma, hay fever or acute bronchitis, he must not dive until resolution is complete.

Pressure Changes and the Ears: otitic compression barotrauma may occur, leading to rupture of the tympanic membrane (possibly complicated by con-comitant serosanguinous transudate in the middle ear) if the pressure in the middle ear space cannot be equalised with the external auditory meatus via the Eustachian tube. This may result in perforation and haemorrhage. Alternobaric vertigo is a further complication, which may be irritated by repeated attempts to equalise middle ear pressure by Valsalva manoeuvres. Many cases of difficulty in equalising pressure result from catarrh within the pharyngo-tympanic tube.

Another syndrome is that of "reversed ears". This can occur when air is trapped in the external auditory meatus, often as a consequence of a tight-fitting rubber hood. The increase in environmental pressure is transmitted to the middle ear by the pharyngo-tympanic tube, and thus the pressure in the outer ear is less than that in the middle ear. The drum tends to bulge outwards, and there is swelling of the lining of the meatus due to oedema. Pain is not a prominent feature, although minor haemorrhage may occur.

Hearing and Balance: any upper respiratory tract infection, or damage to the ear (e.g. hyperaemia or haemorrhage) must be allowed to recover before recommencing underwater activities. In addition, the sea may contain fine particles of sand which often irritate the outer ear with subsequent infection. Wright and Alexander have shown that prolonged exposure to water changed the healthy ear flora from Gram +ve coccii and diphtheroids to Gram -ve bacilli (particularly Psuedomonas pyocyanea) and that this change often preceded acute symptoms.

With regard to balance, the form of vertigo seen in decompression sickness is known as "staggers". The present hypothesis is that gas bubbles are trapped within the microcirculation thus restricting oxygenation of sensory organs within the inner ear.

Pressure and the Sinuses: upon descent, air volume is obviously reduced, and transudate may occupy the resultant space. In contrast to the previous condition, pain is often intense. With re-expansion of air volume during ascent, this may result in mucopurulent discharge or even frank haemorrhage.

Pressure and Bad Teeth: small pockets of gas (from fermentation of food debris) are often found round the roots of teeth. During compression, the gas "bubbles" are reduced in volume, the space being occupied by blood and tissue fluid. Again, re-expansion of air volume results in locally-increased pressure, and subsequent pain.

Pressure may also exert effects on the gas contained within the diver's equipment. If face-mask pressure cannot be equalised, there may be oedema of facial tissues, possibly extending to subconjunctival haemorrhage. Failure to equalise

pressure within a dry suit results in painful "nipping" of the skin. The marks left on the skin must be distinguished from the widespread skin rashes seen in some cases of decompression sickness.

2. HAZARDS FROM GASES

Inert Gas Narcosis: the noble gases, under pressure, may exhibit all the properties normally associated with anesthetic agents. As compressed air remains the most frequently used breathing "mixture", nitrogen is the gas most intimately and continuously associated with the poetically-termed "raptures of the deep". Paradoxically, euphoria is a not uncommon prelude to narcosis.

The signs and symptoms of nitrogen narcosis are as follows:

on descending to	45 m:	Euphoria
" " "	45-60 m:	Joviality
" " "	60-75 m:	Hysteria and numbing of the periphery

Beyond these depths depression, impaired neuro-muscular co-ordination and unconsciousness predominate.

Upon return to the surface, amnesia lasting several hours may follow nitrogen narcosis. Anxiety, alcohol, carbon dioxide retention and fatigue are proven predisposing factors, and ameliorating factors include frequent deep diving and anti-hallucinatory drugs.

When the pressure is released, the excess nitrogen leaves the tissue and recovery is complete. Thus nitrogen should be replaced by a less narcotic gas if dives in excess of 76.2 metres are anticipated. Helium, which is approximately one-eight as narcotic as nitrogen, is used at these depths. Hydrogen and neon have also been used. However argon, krypton and zenon are more potent narcotics than nitrogen.

Oxygen: Problems are encountered with both hypoxia and hyperoxia:

(i) Increased oxygen tension – hyperoxia.

This is differentiated into acute and chronic forms.

(a) Chronic oxygen poisoning (low oxygen concentration prolonged over several hours). The mechanism – which is not well documented – involves a severe inflammatory reaction within the lungs, often leading to pneumonia. General effects include fatigue, decrease in pulse pressure, slowing of pulse, evidence of vasoconstriction in the central nervous system and retinal vessels, and pleuritic chest pain.

The more common danger is that of:

(b) Acute oxygen poisoning, i.e. after brief exposure to high oxygen tensions (2.0 bars and above). The sumptoms, in order of incidence, are: lip twitching, dizziness, nausea, choking sensation, dyspnoea and tremor – which may eventually progress to convulsions. Symptoms normally disappear within a few minutes on returning to air, and no further treatment is required. However, convulsions may cause the self-contained diver in open water to lose his mouthpiece, with possibly fatal consequences. In this syndrome:

(1) as oxygen pressure is increased, the time of exposure before onset of symptoms is proportionately decreased:

(2) tolerance to high pressure oxygen is greatly reduced if exercise is performed:

(3) tolerance varies both between individuals and within the same individual from day to day. The prevention of oxygen poisoning is dependent upon the ability of the equipment to maintain the oxygen partical pressure with safe limits.

Diving on pure oxygen is now restricted to a depth of 8 metres.

(ii) Hypoxia:

In acute hypoxia, loss of consciousness may occur in a few seconds, the collapse often being preceded by sever convulsions. Although rapid recovery takes place if normal oxygen supply is restored before the intervention of anoxic brain damage, amnesia for events immediately prior to the hypoxic incident is a common sequel. Regrettably, unconsciousness may be the first symptom of hypoxia, and syncope in water will result in drowning unless there is immediate rescue. Another similar condition, seen in the subject who

hyperventilates and follows this by prolonged exertion, is latent hypoxia. The sudden loss of muscle tone (subsequent to the exercise) enhances peripheral pooling of blood, giving decreased venous return and reduced cardiac filling with consequent slight diminution in cardiac output. This may prove sufficient to precipitate unconsciousness in the already hypoxic subject.

Both hyperoxia and hypoxia usually result from malregulation of the closed circuit breathing apparatus, in which oxygen in the compressed air is recycled, carbon dioxide being removed by soda lime, thus permitting longer continuous periods under water.

Carbon dioxide: although carbon dioxide *per se* is not used for any specific purpose in diving, in many underwater accidents an increase in carbon dioxide tension is often beyond the control of the diver, and thus he should be able to recognise the preliminary symptoms and signs. These include increased ventilation, mental confusion, severe headache (possibly accompanied by vomiting), decreased blood pressure, lack of co-ordination and slowing of the pulse rate. If allowed to continue this leads to unconsciousness, paralysis of respiratory and cardiac centres, and death.

When man is under pressure the partial pressure of carbon dioxide in the lungs (which is determined by venous tension proportionate to the amount produced by the tissues) is approximately 40 mm Hg. i.e. equal to the normal value on the surface, provided that there is no carbon dioxide present in inspired air and that adequate ventilation is maintained. The percentage of carbon dioxide in alveolar air decreases with depth, but the partial pressure in relation to other gases remains constant. Any air supplied to the diver at depth, contaminated with CO_2, will raise the alveolar partial pressure and carbon dioxide contamination can be magnified greatly, possibly to a fatal outcome.

The use of soda lime introduced another complication. Should sea water leak into the system, the diver may inhale a caustic mixture. The breathing apparatus should be removed as quickly as possible and the diver's mouth washed liberally with a diluted solution of a weak acid, such as vinegar. Any exposed skin must be treated similarly and a drop of sterile liquid paraffin placed into the victim's eyes. The diver should be examined at a future date for any residual lung damage which may have been caused by the caustic inhalation.

Other Gases: (i) carbon monoxide poisoning is an occupational hazard which is usually the result of failure to ensure that the air supply (pump or cylinder is free from traces of this gas. In many cases this is because the inlet of the compressor is situated close to the exhaust! The condition is often manifested by dizziness and fatigue, progressing to syncope. An important diagnostic feature is that lips and mucous membranes often appear bright red. Diagnosis is usually confirmed by examination of the inhaled gas or spectroscopic examination of the blood.

Oxygen is of fundamental importance in the treatment of this condition, although – depending on the circumstances – artificial resuscitation may be a preliminary requirement. Oxygen plus added carbon dioxide is preferable to pure oxygen, as the CO_2 increases the ventilatory drive. Severe hypoxic brain damage is indicated by failure to respond to this treatment within one hour.

(ii) Compressed air may also be contaminated by oil vapour. Oil fumes, under pressure, irritate the membranes of the lungs: in mild cases this may merely precipitate a slight cough, but if exposure to such contamination is prolonged, lipid pneumonia may result.

3. DECOMPRESSION:

There must be a complete reversal of processes by which adaptation to depth is achieved if the subject is to return safely to the surface. Decompression sickness effectively encompasses many diverse features, including any abnormality which is the direct result of environmental pressure. The commonest presenting symptom is severe arthralgia – frequently flitting in nature. Any abnormality often depends on the site of air emboli, and theoretically a wide spectrum of neurological deficits is possible, but for practical purposes the most common presenting sign is sudden loss of consciousness.

Pneumothorax, a frequent consequence of lung overdistension, may well remain undetected until dyspnoea is experienced during decompression from a therapeutic recompression.

(i) Acute Decompression Sickness is sub-divided into Types I and II. The former includes peripheral manifestations and the latter, the central and potentially more dangerous effects.

Type I (a) Pain is the most common feature, ranging from mild pain in the limbs (the "niggles") to a very severe joint pain (the "bends"), and is frequently accompanied by generalised fatigue.

(b) Skin involvement tends to be widespread and includes transient pruritis, subcutaneous oedema — especially of the limbs, and areas of cutaneous vascular stasis with central cyanotic areas, particularly related to the trunk.

(c) Fatigue, malaise and anorexia.

Type II (a) Pulmonary effects, which divers refer to as "the chokes", include substernal pain especially on inspiration, acute dyspnoea, and cough. These frequently precede collapse, shock and asphyxia. The pulse becomes feeble and increasing cyanosis develops.

(b) Neurological decompression sickness shows characteristic effects on the spinal cord, the patient commonly becoming paraparetic. Other neurological deficits encountered in this condition include vertigo, visual defects, monoparesis, mental disturbances, migrane and girdle pains of the trunk.

(c) Postural hypotension may result from the fluid shift which occurs secondarily to the increased capillary permeability. Although haemoconcentration (due to relative hypovolaemia) may not be obvious, the associated rheological changes will almost certainly exacerbate consequences of intravascular pockets of gas.

(ii) Chronic Decompression Sickness: The more extreme cases, involving the spectrum of hemiplegia or paraplegia, condemn the victim to years of disability. Recently, attention has been directed to aseptic bone necrosis, which may result from chronic decompression sickness. This may in turn cause joint deformity and arthritic changes. It is assumed that the condition is caused by minute gas bubbles with surrounding platelet aggregation acting as micro-emboli in the end-arteries of bones.

Diagnosis is normally made on the symptoms and signs already mentioned; however this may only be satisfactorily confirmed by further subjecting the patient to increased pressure, when the symptoms should disappear. Essential treatment for this condition — or air embolism — is to reduce the size of "pressured" air bubbles by immediate recompression to an adequate depth, followed by slow decompression. Any delay in instituting such a procedure will render the patient less responsive to subsequent recompression. In the absence of any recompression facility, a more complex regime of intravenous therapy — as suggested by Saumarex, Bolt and Gregory — may be commenced: however the efficacy of such treatment has not yet been determined.

During recompression, changes in air volumes — for example, within intravenous giving sets — must be anticipated. In the same context, the balloon of a cuffed endotracheal tube must be filled with water to prevent volume changes paralleling those of external pressure. Nitrous oxide and opiate analgesics are contraindicated during the transfer of the patient: however, oxygen can be safely administered. Ancillary treatment includes Dexamethasone, Dextran, and Heparin (the effectiveness of the latter being related to its anti-lipaemic activity).

4. IMMEDIATE CARE OF THE DIVING CASUALTY

Many cases of drowning are complicated by concomitant acute onset of hypothermia. Both salt- and fresh-water drowning — although physiologically distinct — have similar consequences, i.e. acidosis, hypoxaemia and hypercapnia.

Immediate effects of the inhaled fluids include an acute imflammatory reaction in the alveolar capillaries and subsequently accumulation of a plasma-rich exudate within the alveolus. Areas of

atelectasis may result if the inhaled water destroys normal surfactant. In addition, pulmonary or cerebral oedema may ensue, and the risk of pulmonary infection is greatly increased when the water is polluted.

The major aims of treatment of such a condition must include correction of acid base imbalance, rewarming, and — most important — restoration of acceptable levels of ventilation and circulation.

After clearing the mouth and oropharynx, artifical resuscitation should be performed as soon as possible, and, in pulseless patients, this should be accompanied by closed chest cardiac massage (although the mere restoration of adequate ventilation will often result in a satisfactory cardiac rhythm). One must *not* perform closed chest cardiac massage on the hypothermic patient. Ventricular fibrillation commonly follows the slightest mechanical irritation in hypothermia victims, and in such patients the most successful regime includes prevention of further heat loss plus maintenance of ventilation. More recent forms of immediate treatment are:

(i) The "Space Blanket", which is an aluminium coated plastic sheet, intended to prevent further heat loss. This has several disadvantages. It prevents only radiation heat loss (not that by convection or conduction) and the patient may also lose a great deal of both core heat and moisture by normal breathing, a facet of temperature exchange totally unaffected by the space blanket.

(ii) A modern, and controversial, item of equipment in this sphere is one which does not yet even possess a name! This "apparatus" allows the patient to breathe CO_2 through a soda-lime open circuit, resulting in the inhalation of both moisture — an essential requisite in these situations — and heated air thus directly acting upon core temperature. The equipment has not been fully tested, and complications discovered to date include inflammation and sloughing of pharyngeal epithelium; however the extent of usage required before this arises is not known.

When the patient arrives in hospital, an intravenous line should be set up using 8.4% $NaHCO_3$ to correct the acid base imbalance; normally 150 mEq is given, then 70 mEq every subsequent ten minutes until adequate ventilation and circulation return. An E.C.G. is taken on arrival, and if the patient is in ventricular fibrillation D.C. counter shock is applied (unless, as already stated, the patient is hypothermic).

The treatment of hypothermia in hospital is controversial. Myocardial rewarming can be accomplished by means of cardio-pulmonary bypass, although this is obviously limited in its practical potential. The most widely-used technique is probably immersion in a hot bath at 45°C with the limbs *out* of the bath, always ensuring that the water remains at that temperature; there is an unfortunate tendency to place the patient in the bath forgetting that as his temperature increases, that of the surrounding water will proportionately decrease, thus reducing the efficacy of the treatment. Present forms of anti-arrhythmic therapy have proved ineffective in the treatment of cardiac arrhythmias resulting from hypothermia. These usually revert to normal within 12 hours of restoring core temperature.

Other forms of management include peritoneal dialysis with warmed fluid; breathing heated humidified air; enemas with warmed fluid; and instilling warm water into the stomach. Deeply hypothermic patients may suffer little anoxic brain damage during periods of circulatory standstill: at 37°C the maximum safe period is about 3 minutes at 32°C 8-10 minutes and there is one documented case of a patient with a core temperature of 15°C surviving after 1 hour of circulatory arrest. Return of spontaneous respiration obviously depends on the amount of anoxic brain damage present.

Patients should be intubated and ventilated with 100% oxygen. Normally the oxygen is given by IPPV, however recently PEEP has been found to be effective in preventing pulmonary oedema, and this is now being increasingly used. In non-hypothermic patients who are being ventilated but do not have an acceptable heart action, an intracardiac injection of 2-4 ml. 1/10,000 adrenaline and 10 ml. of $CaC1_2$ should be given.

Thus the problems of initial treatment, both immediate and subsequently in hospital, have been

briefly considered. The later management of the patient includes problems of "secondary drowning" and a condition resembling the respiratory distress syndrome. Secondary drowning is characterised by tachycardia, anoxaemia, fall in pulse pressure then in overall blood pressure, a rapidly increasing pulmonary oedema and changes in cardiac rhythm. Both the respiratory distress syndrome, in this instance, and secondary drowning are treated by early ventilatory support accompanied by concurrent circulatory support with the infusion of up to one litre of plasma (under central venous pressure control). Methyl prednisolone is usually given to counteract any pulmonary or cerebral oedema secondary to anoxia.

It can be seen that the underwater environment may encompass additional features, within the emergency situation, to those commonly encountered on the surface. All possible aspects of this form of accident must be thoroughly investigated: later complications often result from omissions in initial investigation, rather than inherent difficulties in subsequent treatment regimes.

References –

Wright and Alexander

Saumarez, Bolt & Gregory

REFERENCES

1. Cochleo-Vestibular Disturbances in Diving Audiology 15(4) 273-8 July-Aug 1976.
2. Skeletal Changes in Divers, Australas Radiol 20(1) 83-84 March 1976.
3. EEG Criteria for Diving Fitness, Corriol J. Aviat. Space Environ. Med. 47(8) 868-73 Aug. 1976.
4. Haematological Changes in Man during Decompression: Relations to Overt Decompression Sickness and Bubble Scores – Goad R.F. J. Aviat. Space Environ. Med. 47(8) 863-7 Aug. 1976.
5. Editorial: High Pressure Medicine, BMJ 4(5996) 541-2 6.12.75.
6. Letter: Medical and Surgical Emergencies in Divers, McCallum BMJ 1(6931) 832 3.4.76.
7. Medical Aspects of Scuba Diving and Underwater Photography – Mayer B.W. Del. Med. Jour. 48/6) 331-5 June 1976.
8. Management and Treatment of Osteonecrosis in Divers and Caisson Workers – Walder et al Washington; US Dept. of Health, Education and Welfare publication.
9. Stress in the Deep; A Study of Undersea Divers in Controlled Dangerous Situations, J. Applied Psycho. 61(4) 507-12
10. Decompression Sickness; A Clinical Series – Erde A. et al J. Occup. Medicine 17(5) 324-8 May 1975
11. Resp. Heat Loss and other Thermal Problems of Deep Diving – de Hanson et al J. Roy. Nav. Med. Service 61(11) 16-21 Spring 1975
12. Intro. to Scuba Diving Belanke et al J. Sports Med. 2(5) 276-90 (Sept/Oct
13. Inves. of Scuba Deaths – McAniff J.J., J. Sports Med. 2(4) 199-208 Jul/Aug 1974
14. Air Embolism and Decompression Sickness in Scuba Divers – Whitecraft D. et al JACEP 5(5) 355-61 May 1976.
15. Underwater Deceleration Curves in Relation to Injuries from Diving – Albrand D.W. Surg. Neurol. 4(5) 461-4 Nov. 1975.
16. Otolaryngeal Complications of Skin and Scuba Diving: Causes and Treatment – Wilson Trans Pac. Coast Otophthalmic Soc. 55; 61-76 1974.
17. The Distribution of Vascular Bubbles in Multiple Dives – Gait D. Undersea Biomed. Res. 2(1) 42-50 Mar. 1975
18. Responses of Scuba Divers via Undersea Telemetry – Godden B.A. Undersea Biomed. Research 1(1) 45-58 March 1974

19. General History of Vestibular Disorders in Diving – Kenned D.S., Undersea Biomed. Res. 1(1) 573-81 Mar. 1974
20. Nitrogen Elimination in Man During Decompression – Kindwall E.P. Undersea Biomed. Res. 2(1) 285-97 Dec. 1975.
21. Diver. Temp. and Performance Changes During Long Duration Cold-Water Exposure, Undersea Biomed. Res. 2(2) 75-88 June 1975.
22. Causes, Mechanisms and Prevention of the High Pressure Nervous Syndrome – Hunger W., Undersea Biomed. Res. 1(1) 7-28 March 1974.
23. Effects of Water Immersion on Renal Haemodynamics in Normal Man – Epstein M., J. Applied Physiol. 41(2) 230-3 Aug. 1976
24. Narcotic Potency of N_2 and N_2O evaluated by the Physical Performance of Mouse Colonies at Simulated Depths – Ralin H. et al, Undersea Biomed. Res. 3(1) 25-34 March 1976.

MEDICAL MARACAS

by
James Owen Drife
Senior Registrar in Obstetrics and Gynaecology, Bristol

For ten years I was one of Edinburgh's few medical maraca players. I hope that's clear: I was a medical player of maracas, not a player of medical maracas. A maraca is a Latin-American musical instrument constructed originally, I suppose, out of a coconut with beans inside. Little natural aptitude is required to shake one in time to the music, but because they come in pairs, two hands are needed. Thus Latin-American orchestras have a limited number of openings for musical illiterates who own their own dinner-jackets, which is why your intrepid correspondent eventually ended up shaking bean-filled coconuts at the revellers at ten consecutive Medical Faculty Balls.

Our band, the Unbelievable Brass, was born in the Physiology Library in 1968. In those days the library was equipped with high shelves, ladders and a variety of mini-skirted research workers, and we perspiring undergraduates were forced to sublimate by doing crosswords, writing songs and producing revues. To one such revue the class's own trumpet-player brought along half the brass section of the University Orchestra, and I found myself part of the ensemble, doubling as maraca-player and lady vocalist. My debut involved rushing out to the tiny toilet to change into wig, balloons and dress, and tottering back to reveal myself to an appalled and largely silent audience.

In the natural scheme of things our career would have ended there, in that little hall which the creative genius of the University has since converted into a car park. However, as luck would have it we also guested that night at the Fourth Year Dance, where our mixture of trumpeters, trombones and transvestism was an unexpected triumph. Our colleagues came to jeer but remained to get stoned out of their minds, and when we told them next day that we had been a resounding success, none could remember enough to disagree. Our showbusiness career was under way.

The case-history outlined so far may be very familiar to you. Nowadays the stages of London's famous West End and Edinburgh's celebrated Festival swarm with medical undergraduates — and indeed registered practitioners — and an observer has the impression that showbiz features on the modern medical curriculum, somewhere between sex and sociology. But back in the pioneering days of the sixties the Edinburgh medical student was a creature of sombre dignity who looked askance at the musical stage. Bagpiping was acceptable; violin-playing was dangerously eccentric, and maracas were instruments of the devil. So at first the Unbelievable Brass bent over backwards to maintain a low profile (as "A Spokesman" might phrase it), but as we became older and acquired experience — not to mention degrees and cirrhosis of the liver — our confidence grew. What did not grow, however, was our repertoire. Although the personnel of the band changed during the decade and drummers came and went, we steadfastly retailed the same two or three dozen tunes whether at a barn dance, a Jewish wedding, on top of a horse-drawn carriage or on our home

territory, the George Street Assembly Rooms.

The Assembly Rooms were the natural home of balls, which were popular at the outset of our career. The Medical Faculty Ball was the highspot of the social year for doctors and medics, and the Charities Ball inspired other students to camp out all night to be sure of the opportunity of buying tickets — which at that time could be purchased without a mortgage and allowed the bearer to dance till dawn to the music of star performers. Watching from the wings, as it were, we noted that the *modus operandi* of big-name musicians varied: some arrived (in James Cameron's phrase) "tired as a newt" while others were reliable journeyman — and best value of all, if you're interested, were Geno Washington and the Ram Jam Band. Well down the bill, the UBB (as we in the Unbelievable Brass now suavely called ourselves) acted as warm-up men and then joined the frolics.

This was the zenith of our career: around this time an apparently sober Kenny Ball was heard to remark that our performance was "not bad". Then an even more impressive accolade came our way: one night at a Union Palais I looked down from the bandstand to see two heavy teenagers dancing together near my feet, casting come-hither leers up at us through their mascara. I felt that life had little more to offer, though in fact the band was never organised enough to keep groupies — and the longsuffering girlfriends who came along to carry drums for us objected to being called "bandies". Still, I now have a lot of insight into the temptations strewn in the path of Daniel Barenboim or the Boomtown Rats.

I learned a few more of life's poignant lessons during our climb to fame. In particular, I learned what not to do. For example, intoxicated with my first audience during that memorable revue, I had cried out briskly, "One, two, three, four!" at the start of a number. I vaguely imagined that musicians did this to boost morale, rather like the late John Wayne hollering, "Let's go, fellas!" as the posse gallops out of the corral. I realised my error as the horrified musicians thundered *prestissimo* through a normally sedate tune, unable to apply the brakes. When the rendition ended (thirty seconds later, including choruses), the purple-faced trumpeters suggested politely that I might leave counting-in to them in future. I learned also that one would no more dream of talking to a musician when he's working than one would chat to a cholecystectomist or a plumber struggling with a U-bend. Formerly I had thought that drummers must feel a bit lonely, sitting at the back with nobody to speak to. In fact, although drummers concentrate with their eyes open, they are in no less of a trance than their colleagues who shut their eyes and blow things. If you essay bright small-talk with a drummer, he can usually only manage two words in reply — and one-syllable words at that.

I learned a little of the delicate art of negotiation. When the UBB arrived at a hall to find the piano missing or unplayable we coped by giving the pianist some compensatory pocket-money and a stool at the bar — no-one could normally hear him anyway. But when Kenny Ball's road manager arrived to find the Assembly Rooms' stage pianoless, he decreed with a shrug that without a piano his boys could not perform. Immediately a dozen students manhandled the Corporation's priceless Steinway Grand from its hiding-place, with only mild or moderate damage to building, piano and students. Watching in admiration of the roadie's technique, I realised that when a thousand dinner-jacketed punters are already smoothing in through the front door, the show must go on.

We learned too that at the Medical Faculty Ball, the final spot — 2 a.m. to 3 a.m. — was the best one to play. By that time the weaker customers have gone to the wall, and the more robust couples remaining, having carried out the necessary duties of meeting old friends, complaining about the food and dancing the Dashing White Sergeant, are at last ready to enjoy themselves. During a Ball there is normally a lull just after midnight as people's normal diurnal rhythm struggles to assert itself, but after about 1.30 a.m. the superego gives up and the id surfaces. Professors fall over, their wives succumb to the primitive jungle beat, and invariably some idiot takes his clothes off. We gave up playing "The Stripper", ostensibly because the trumpeters lost the music, but in reality because the audience participation — regrettably invariably male — became more and more degenerate year by year. The last time we tried the tune a Vet (it was always a Vet) leaped on to a table, took off his jacket, swung it sensuously around his head and

let go. All very amusing, but unfortunately he had thoughtfully provided himself with a carry-out and his pockets were full of cans of McEwan's Export: the jacket, acting like a South American *bolas,* narrowly missed decapitating some Faculty members, and the band swiftly slid into "White Christmas".

The sun has now set on my maraca-playing career — and just as well, probably. At the extremes of medical life (as a student or as a professor) membership of a dance band is like a woman's preaching as defined by Dr Johnson — "the wonder is not that it is done well, but that it is done at all." As a senior registrar, however, a man is judged without indulgence, and with due allowance for natural modesty I feel that as a maraca-player I could have achieved competence but not greatness. I shall never follow the James Galways of the world to the position of First Maraca to the Berlin Philharmonic and tax-exile in Lucerne. Last time out with the UBB I had the uncomfortable new feeling that the old rapport with the student audience was fading, and that the oddly short-haired listeners were asking themselves why these members of assorted Royal Colleges were enjoying themselves so much on the band-stand. Students hadn't changed, of course — students never change — but we had. Or perhaps we were worrying that we might have changed. Or perhaps we hadn't had enough to drink.

It had been fun — albeit rather expensive fun. (Traditionally the UBB led a drunken debauch by example as well as precept, and we usually paid more to the barman than we collected as a fee.) But the trumpeters enjoyed themselves, playing and watching the girls — even the most ascetic jazzman opens his eyes when a spectacular decolletage boogies past the stand — and the maraca-player, sensitive flower that he is, enjoyed himself most of all. Free of the worry of playing bum notes and with the drummer leading the tempo, the maraca-player can shimmy around the bandstand, participating as an Associate Member in the unique rapport that exists between musicians, but able also to watch the audience. Audiences are great entertainment. "Strange how potent cheap music is", wrote Coward, and at 3 a.m. our music (not cheap, but competitive) was as potent as any. Happy tunes had the floor bopping creatively, while smoochy numbers produced buttock-clutching embraces and dreamy expressions that might equally signify lust or exhaustion. Any national anthem except "God Save the Queen" would, at the drop of a hat, fill the appropriate ethnic group with solemnly homicidal fervour, but with "Auld Lang Syne" we were all buddies again, moist-eyed with ethanolic nostalgia for a great night. It was a privilege to hide behind a microphone and watch it all. Thank you, revellers one and all: and now, most sincerely on behalf of all the boys in the band, may I wish you goodnight, God Bless, and a safe journey home. Drive carefully, won't you.

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BOOK REVIEWS

Title: Geriatrics (Guidelines in Medicine – Vo.1)
Authors: A.N. Exton-Smith, P.W. Overstall.
Publishers: MTP Press Limited. **Pages** 344.
Publication Date: 1979

Geriatric medicine is becoming a more important discipline and occupies a greater proportion of the undergraduates' clinical experience. The student requires an accompanying textbook from which to fortify his clinical knowledge, but until recently the choice of reasonably priced texts has been poor and it is good to be able to welcome a newcomer to the field.

The book, in hardback, is written by two distinguished geriatricians and is well laid out in a structured manner. It commences with a chapter entitled "Theories of Ageing" and ends appropriately with a chapter entitled "Care of the Dying". This last chapter would have been enhanced by a small section relating to the legal disposal of the dead, a topic which is often bewildering to the newly qualified.

Between the aforementioned chapters the authors approach the subject in a systematic manner with chapters on special geriatric problems interspersed throughout. Subjects included are, nutrition, and rehabilitation. The rehabilitation chapter concentrating on strokes is welcome as the subject is an often neglected aspect of undergraduate teaching.

The chapter relating to the Principles of Drug Therapy has been relegated to the back of the book, a site I consider totally inappropriate for such an important subject. This chapter would have been better following the chapter on "Special Features of Disease in Old Age" thus emphasising to the reader the need to be especially careful when prescribing for the elderly.

The social aspects of geriatric care have been scattered throughout the text making it difficult for the reader to assess what services are available for the elderly. The authors would probably have achieved a more comprehensive account had they entitled a chapter: Social Services for the Elderly.

Psychogeriatric welfare is reviewed briefly with the emphasis being correctly placed on the common geriatric problems, namely toxic confusional states and dementia.

I have a brief criticism to make relating to the layout of the index. Most subjects can be found under their own heading but unfortunately this is not so in the case of dermatological problems where the reader is required to wade through the "skin diseases" section to find the relevant topic. I would have thought Pressure Sores was an important enough subject to warrant a separate entry.

In summary, this is a welcome addition to the fold of geriatric medicine textbooks but requires to be fortified with information from other larger and smaller works.

I also wonder if the authors have found a new cure, or psychosomatic disorder:– "Radiography may provide great symptomatic relief for superior vena caval obstruction . . . ." This may well be so in psychiatric practice but I very much doubt it in the case of bronchial carcinoma.

A.J. Grant

Title: The M.R.C.G.P. Examination.
Authors: A.J. Moulds, T.A. Bouchier Hayes & K.H.M. Young. **Publishers:** M.T.P. Press Limited, International Medical Publishers. **Pages** 131.
Publication Date: November 1978. **Price:** £4.95

With about 1000 candidates now presenting themselves annually for the M.R.C.G.P. examination it was only a matter of time before this hardback, or something like it, appeared in medical bookshops. It is subtitled "A comprehensive guide to preparation and passing" and the authors, who are the organisers of some of the most successful courses for the M.R.C.G.P. exam and all serving officers in Her Majesty's forces, should know what they are talking about.

In his foreword, the ubiquitous Dr. John Fry, explains that the failure rate – now approaching 40% – in the exam, is due, at least in part, to unfamiliarity with the special nature and form of the exam – with MCQ's (Multiple Choice Questions), MEQ (Modified Essay Question), TEQs (Traditional Essay Question) and oral tests.

The book has been written to prepare and familiarise candidates with these various parts of the examination. It does this admirably. It is not a textbook. Six short chapters deal with each section of the examination – what to expect and how best to prepare. Also included are a few vital statistics concerning General Practice in the National Health Service, a recommended reading list (books, periodicals, journals and papers) and an eight point work-plan for the candidate. All of these make up rather less than half of the book. The major part is a full length mock examination with answers at the back.

At the moment the majority of candidates presenting themselves for the examination are young doctors at the completion of their training or soon after becoming principals in general practice. The examination has therefore become increasingly oriented to an assessment suitable for these candidates. It is possible that the present form of the examination may change in the foreseeable future, but until it does, all candidates would be well advised to read this book. Whether or not each should be encouraged to spend a fiver to secure his own copy is doubtful. The book can be borrowed, read at leisure and returned after two or, at the most, three evenings and even then leave the reader enough time to catch his favourite T.V. programme.

Anyway, if the book serves its purpose it shouldn't need to be read again.

J.S.K. Stevenson

Title: Local Analgesia – 2nd edition.
Author: Dr. Clive Jolly. **Publishers:** H.K. Lewis & Co. Ltd., London. **Pages:** 152 (illusr. – 19)
Publication date: 1979. **Price:** £5.00 nett

The second edition of Dr. Jolly's small book on 'Local Analgesia' appears at an opportune time when there is a resurgence of interest in analgesic techniques for surgery as well as for pain relief in non-surgical conditions. As a primer, the book gives a useful outline of the principal techniques available, their indications and contraindications and a description of the various complications which may be anticipated. There is a useful chapter on points of technique to be observed in the performance of any kind of analgesic block and a reminder that analgesic techniques may be, but are not necessarily, safer than general anaesthesia.

The final chapter on 'Therapeutic Uses of Nerve Blocks' is, however, rather patchy and unbalanced. It is admittedly difficult to summarise briefly the whole field of the work of Pain Clinics but it would have been worthwhile extending this chapter to give a more comprehensive outline of the blocks used and the conditions for which they are indicated. The author has attempted to cover the subject by mentioning only a few techniques to illustrate how local analgesia is being used outwith the operating theatre.

This book is a helpful introduction for the junior anaesthetist to the subject of regional blocks. For those who wish to delve deeper into the subject, there is a select list of articles for 'Further Reading' in addition to a comprehensive list of references.

While the book can be recommended for the junior anaesthetist, it is a pity that there is so much evidence of slipshod proof reading in the form of misspelling, grammatical errors and omitted apostrophes.

A.H.B. Masson

SOCIETY NEWS

(MEMBERS OF COUNCIL for the 244th SESSION 1980/81)

The following members of Council were elected at the Annual Extraordinary General Meeting held on Wednesday, 7th May 1980:

Senior President: Christopher Ingamells
1st Junior President: Alex Hamilton *2nd Junior President:* Brian Montgomery
3rd Junior President: Hamish McRitchie *Senior Secretary:* Dorothy Russell *Junior Secretary:* Lyn McLarty
Convenor of the Business Committee: Alastair McKinlay
Convenor of the Publicity Committee: Roderick Elliott
Convenor of the House Committee: Iain Lindsay
Convenor of the Library Committee: Elizabeth Boyter
Convenor of the Museum Committee: Sandra Brown
Convenor of the Entertainments Committee: Lizbeth Jordan

Non Council posts:
Editor of Res Medica: Graeme Duncan
Convenor of the Annual Dinner Committee: Lizbeth Jordan

The Appeal Fund which was established to purchase new premises for the Society, has now been converted to a Trust Fund. Registered as a charity with the Inland Revenue, its purpose is to administer the financial assets of the Society. The Trustees include: –

Sir Derrick Dunlop *(Chairman)*
Prof. D.C. Simpson/Mr. Ian McLaren – *Joint Treasurers*
Prof. Archie Duncan *(Univ. Repres.)* Sir John Croom *(Royal College of Physicians)*
Prof. John Gillingham (Royal College of Surgeons) Dr. Beverley Norton *(Past Members)*
and the current Senior President

The Society is deeply grateful to its Solicitor, Mr. Robin Martin, for his services in the recent formation of the Trust Fund. Mr. Martin was elected an Honorary Life Member at the 1979/80 Annual Extraordinary General Meeting.

Financial assistance was given by the Trust Fund this year to two notable educational visits:–

i) Sixteen members of the RMS spent one week visiting the Leiden Medical School, Holland in March under their Mentor Dr. Simon Glover. This visit continues the friendship between medical students in Holland and the RMS.

ii) Eighty Finnish medical students from Helsinki were the guests of the RMS during their recent visit to Edinburgh, under the theme "Primary Health Care in Scotland", although under the direction of Mr. Alex Hamilton a wide range of activities was organised. It is hoped that this new friendship will be continued in future years.

Dr. Jack Cormack (Librorum Custas) with the assistance of Mr. Alan Boyd is cataloguing all the remaining valuable books in the historical library, which the George Square Library has previously very kindly stored for us. The collection is to be displayed in the Malcolm Low Room.

Prof. D.C. Simpson (Hon. Fellow) has retired as Executive Dean from the Faculty of Medicine.

Engagements:–

Surg. Lieut Simon Glover R.N. (Olim Praeses) to Miss Jane Petrie (Soc. Od)
Mr. Robin Mitchell (Praeses) to Miss Diana Robertson (Praeses)
Mr. Alex Hamilton (Scriba) to Miss Shona Ross (Aedilie)

Marriage:–

Mr. John Rainey (Life Member) to Miss Linda King (Life Member)

Deaths:–

Tragically, Mr. Marshall Johnson – November 1979

Three oil paintings owned by the Society, and at present hanging in the National Portrait Gallery, have been reproduced photographically on canvas, and are to be hung in the Society's Rooms. They are the portraits of James Black, William Cullen and Andrew Duncan.

Printed by Port Seton Offset Printers Ltd., Seton Works, Cockenzie, East Lothian