EDITORIAL / VAN DIE REDAKSIE

Continuing medical education in obstetrics and gynaecology

The present century was only 5 years old when Sir William Osler observed to an undergraduate audience in Oxford that the course upon which they were embarking was not a medical course, not a college course, but a life course. Throughout the world, this life course is generally made up of three sequential phases: undergraduate education supervised by universities, postgraduate education supervised by national colleges or councils and continuing medical education (CME), whose supervision is best described as varied. CME may be defined as that phase of medical education which begins, or should begin, when an individual completes his or her postgraduate training and obtains, from the relevant licensing body, accreditation to function as an independent practitioner. CME is our equivalent of what most other professions call continuing professional education (CPE). Call it what you will, the bedrock upon which this phase of education is built is the belief that education should not end with accreditation but rather, should last for a professional lifetime. In Europe,[1] and the USA[2] and Australasia[3] there is a general movement towards the concept that CME is not an optional extra but a professional duty which we owe to ourselves, to our juniors and to our patients. Medical education should, in fact, be seen as a continuum whereby the undergraduate years at medical school lay the foundations for postgraduate specialist training, which in turn determines the agenda for CME. Like medical audit, continuing education should actually be a state of mind. The thoughtful doctor, like the good scientist, is perpetually dissatisfied with the state of clinical knowledge and technical practice in his field and is naturally interested in relevant new data or technical developments. Equally, the truly educated colleague is one who has been equipped to receive such data or claims with a critical eye, and who is capable of drawing inferences from them in proportion to their validity and relevance. It can therefore be seen that the clear duty of the undergraduate schools is not, as is often the case at present, to produce a mini-specialist in a large range of disciplines, but rather so to educate an individual that maximum benefit will be obtained from subsequent postgraduate and continuing education. Medical students should be gently encouraged to become physically addicted to education so that even temporary withdrawal causes symptoms of anxiety and a desire for satisfaction.

Of the three phases of medical education CME is by far the longest and the most neglected. However, there is a general trend towards continuing professional accountability in most developed countries, perhaps reinforced by governmental and legal pressures, which has led to a major development with regard to CME in Autralia and an imminent one in the UK.

The discipline of obstetrics and gynaecology, perhaps because of its relatively small size and well-defined clinical area, has long been a pioneer in both undergraduate and postgraduate educational development. To take a South African example, the biennial meeting of the SASOG now incorporates a full session on undergraduate education in the specialty; this year, the session was attended by representatives of every medical school in the country.

In 1978 the newly formed Royal Australian College of obstetricians and gynaecologists (RACOG) announced that its fellowship, the accepted qualification for specialist practice in Australia, would be granted on a time-limited basis and would be subject to recertification through participation in a CME programme. In other words, the RACOG saw itself not just as a gatekeeper, controlling entry into the consultant grade but also — the analogy is irresistible — as a gamekeeper monitoring and sustaining the educational health of the flock.

In the UK, the Royal College of Obstetricians and Gynaecologists (RCOG), after conducting a survey of its members and fellows, has taken the decision that mandatory, time-limited and CME-based recertification should be established. Not wishing to reinvent the wheel, the College has taken pains to establish the practical stengths and weaknesses of CME programmes in the USA and Australasia, with a view to achieving a running rather than a standing start to the programme.

The UK programme is likely to operate on a cognate points system similar to that used in Australia. Cognate here means 'of equal value', e.g. 150 points gained over a 5-year cycle is the Australian target. A key ingredient in a successful CME programme is flexibility. The system must be able to cater for consultants in isolated areas, those with large private practices, academics and those working in teaching hospitals. The key to flexibility is the use of a broad definition of what is educational and hence pointsworthy (neologisms may be necessary here). For example, it is clearly educational to give, as well as to receive, a lecture or seminar; it is educational to publish a paper in a refereed journal and it is educational to construct multiple-choice questions for an undergraduate examination. All the above activities are likely to earn points and will complement attendance at accredited meetings and workshops. Another essential source of points is activities at home base. A mandatory CME programme is a golden opportunity to provide a useful reward for a colleague's participation in clinical audit meetings, quality assurance groups and perinatal morbidity meetings. It is likely that, say, 25% of the required points total may be acquired in this way, the balance coming from accredited meetings away from home base. Some of the latter would take place at district or regional medical societies, which would have more purpose as a result of accredited and hence point-bearing meetings. The author, on a recent visit to South Africa, had the pleasure of addressing and being interrogated by the Pietermaritzburg Intellectual Gynaecology Society (PIGS). The accreditation of such meetings could not but encourage the flow of ideas and data, the critical examination of which is the essence of education. Equally, attendance at multidisciplinary groups or at individual sessions for instruction in new surgical techniques could be rewarded. For many colleagues, the CME programme will simply be a codification of what they already do, but there is evidence from the RCOG survey of UK consultants that a substantial number take part in little organised educational activity and would welcome a formalised programme. The Australian experience is certainly encouraging. At the RACOG in Melbourne this year, the author informally assessed the perception of the CME programme by 35 of its participants. In general, there was a highly positive attitude, the colleagues approving the fact that the process was mandatory for all. They felt that it had provided an instructive and indeed enjoyable new dimension to their clinical lives, while generally reassuring them that their knowledge and practices were informed by the latest developments. Areas requiring improvement, however, included the need for an even greater flexibility in point-bearing activities and a *cri de coeur* from some academic staff that their pedagogic and scientific activities were insufficiently rewarded.

Based on an address delivered at the 26th Biennial Congress of the South African Society of Obstetricians and Gynaecologists at the Wild Coast Sun, Transkei, 14-18 September 1992.

EDITORIAL / VAN DIE REDAKSIE

With regard to sanctions, the Australian experience has been remarkably bloodless. Of the 708 specialists in the CME programme only 6 failed to convince the RACOG that they should be re-accredited, and reverted from 'fellowship' to 'membership' status.

Does CME work? In a recent review of 50 randomised controlled trials, Davis *et al.*[4] reported that where CME programmes were practice-enabling or reinforcing, then an improvement in physician performance was to be expected. An improvement in the ultimate end-point, health care outcome, has proved more elusive but evidence of such improvement has been forthcoming from some,[5,6] but not all[7,8] studies.

In summary, then, CME is probably here to stay. It already exists everywhere, although it is often voluntary, with much of the activity unquantified. In each society the regulatory authorities will have an opinion as to whether it is likely that the performance of the physician and, ultimately, the care of the patient is likely to be influenced by a mandatory CME programme. If such an opinion is accepted, it is the duty of the profession to set up a CME programme from within rather than from without, given the inevitable resentment which the latter course would attract.

Would you fly with an airline whose pilots might, or might not, participate in mandatory regular checks on their competence? It is unlikely. As doctors we should determine if, by education alone, we may account our fitness to practise. Ultimately, it may be that, like airline pilots, we will have to be examined critically on a regular basis via tests of knowledge, simulated emergencies and the like. Yet, with a universal CME programme that focuses on quality assurance at home and state-of-the-art presentations away from home, it should be possible for us to regulate ourselves to a degree acceptable to our health authorities, our peers and, most importantly, our patients.

DAVID W. PURDIE
Postgraduate Education Centre
Hull Royal Infirmary
Kingston Upon Hull
UK

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The present and future of obstetrics and gynaecology in South Africa

The first audit of human resources in obstetrics and gynaecology in South Africa was recently reported.[1] This audit is discussed in this editorial.

Summary of the audit

Pertinent aspects of the demography of South Africa relating to the specialty of obstetrics and gynaecology include the composition and size of the population, its age distribution, level of education and financial strength. A massive process of urbanisation is taking place, mainly in the PWV area, followed by Durban, Cape Town and Port Elizabeth. Eighty per cent of the population is black (1985 census) and this proportion is expected to increase in the future at the expense of the other three population groups. The population growth rate is unacceptably high, with an unsatisfactory decrease in the birth rate. The mean age of the population is 25 years with almost 40% under the age of 16 years. Educational standards are low; 30% of the population are without training and 37% have had primary school education only. Per capita spendable income (income minus income tax) has decreased significantly since 1975. Furthermore, the country's financial growth rate has remained at the low level of 2,8 - 3% during this period.

In 1990 South Africa had one hospital bed per 216 people. Twenty-eight per cent of the beds were in private hospitals (21% if the TBVC countries are included). The cost per bed was high, ranging from R41 000 in the Cape Province to R23 000 in Natal. The reason for this difference is probably the influence of the former mission hospitals (e.g. in KwaZulu) where a less expensive health service has been provided for many years.

The total number of medical practitioners is rising steadily, and this trend is expected to continue into the next century. In 1991 there was 1 specialist for every 2,8 general practitioners. Eighty per cent of medical practitioners were white (and only 5% were black). More than 95% lived in urban areas, the majority in the PWV area, Cape Town and Durban. The distribution of medical practitioners in the four provinces was as follows: 43% in Transvaal, 28% in the Cape Province, 16% in Natal and 8% in the Orange Free State; 5% were living elsewhere. The percentage increase in the number of medical practitioners since 1988 was largest in the PWV area, followed by Durban, Port Elizabeth and Cape Town.

Obstetrics and gynaecology is the fourth largest specialty after medicine, anaesthetics and surgery. In 1991 there were 642 gynaecologists, 562 in active practice. Of these, 97% were white and 85% male.

A questionnaire was sent to all gynaecologists; there was a 52% return rate. Seventy per cent of this sample had qualified since 1970, the mean age was 50 years, 70% were in private practice and 23% in full-time hospital practice. The majority of their patients were white, with blacks the next largest group. The standard of training was generally considered satisfactory, especially with regard to obstetrics, abdominal gynaecological surgery and female infertility. However, training in ultrasound, advanced treatment methods for infertility, oncology and, to a lesser extent, vaginal gynaecological surgery was considered inadequate. The general opinion of the respondents was that there were enough gynaecologists in South Africa, if not too many. Should private practice be abolished in South Africa, at least one-third would consider emigration.

There were 6 million people on medical aid in South Africa in 1991 (57% white, 23% black, 15% coloured, and 5% Asian). There were therefore 270 people on medical aid per medical practitioner, 370 per general practitioner and 1 000 per specialist.

EDITORIAL / VAN DIE REDAKSIE

In the seven academic departments of obstetrics and gynaecology there were 120 specialist posts (12% vacant) on 1 January 1992, 166 registrar posts (only 1 vacant), and 84 medical officer/senior house officer posts (only 2 vacant). A need was expressed for 35 additional specialist posts, 27 registrar posts, 43 for medical officers, and 27 other posts — mainly for research assistants. Of the registrars, 80% were male, 60% white, 21% black, and 18% Asian. The number of registrars per consultant was 1,4 with 812 deliveries per registrar per year (132 deliveries per final-year medical student per year).

The cities and surrounding areas where there were sufficient (or almost sufficient) gynaecologists were Pietermaritzburg, Bloemfontein, Cape Town, Pretoria, Johannesburg and Durban. Areas with a capacity to absorb more private gynaecologists were the West and East Rand, Highveld Ridge (east of the East Rand), northern Natal, northern Orange Free State and western Transvaal. There were about 25 full-time specialists outside academic medicine, but the total number of posts available was not identified.

Discussion

An excess of gynaecologists has certainly not been identified. Both the private and hospital sectors can accommodate additional gynaecologists. Furthermore, the mean age of the South African population is only 25 years and there is a high growth rate — two aspects that will increase the need for obstetric and gynaecological services. There is, however, an imbalance between the private and hospital sectors. Seventy per cent of respondents to the questionnaire were in private practice, while only 28% of all hospital beds (TBVC countries excluded) are private. Furthermore, only 6 million people are on medical aid (20% of the population), of whom 3 million are women (1 800 000 16 years and older). For women on medical aid, there is 1 gynaecologist for every 5 000 women (1 for every 3 000 women 16 years and older). In contrast, for the 12 million (7 200 000 16 years and older) not on medical aid there are only about 130 gynaecologists – a ratio of 92 300:1 (55 400:1 for women 16 years of age and older).

Although postgraduate training in obstetrics and gynaecology was generally satisfactory, certain deficiencies were identified. These were ultrasound, advanced infertility care, oncology and laser treatment methods — all highly technical activities. In addition, a growing awareness of the need for an extended (5-year) postgraduate training period has been identified. The question is, however, how relevant is current postgraduate training?

In response to the above question, two objectives in postgraduate training can be identified. On the one hand, gynaecologists want to comply with First-World standards which are universally held to be the 'gold standard' for top-quality obstetric and gynaecological care. On the other hand, such a style of medicine might prove too expensive for South Africa, especially in the future. In addition, does the 80% of the population with a low income and a low level of education want First-World medicine? Both these objectives — compliance with First-World standards or Third-World needs — have merit and each objective will receive support from the gynaecological community. Future training should therefore accommodate both objectives for which there are, again, two possibilities. The first is an extended (5-year) course incorporating training in advanced technological techniques, as well as the teaching of skills in basic, almost primary care (e.g. the organisation of a community obstetric programme). Secondly, the 4-year course which excludes the superspecialties can be maintained. After this basic course, further formal training should then be available in the highly technical subdisciplines. Accreditation for units capable of such training should be a prerequisite.

In conclusion then, although there is not an excessive number gynaecologists and although the standard of training is generally acceptable, the direction in which obstetrics and gynaecology should develop needs revision. The realities and needs of the Third-World component of our population are at present not adequately addressed by this specialty. These needs should not only be identified, but also included in future training programmes.

H. S. CRONJÉ
Department of Obstetrics and Gynaecology
University of the Orange Free State
Bloemfontein

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The case for an increased tobacco tax in South Africa

The health and economic impact of tobacco smoking has been examined in detail in South Africa: 25 450 smoking-related deaths were reported in 1988; 110 856 potential years of life were lost in the age group 35 - 64 years. In economic terms, tobacco use resulted in R1,1 billion from lost productivity due to premature death and hospitalisation, while an additional R289,6 million was due to direct health care costs.[1]

Government intervention is required for tobacco control since the monopolistic nature of the tobacco industry has meant that the industry has a strong influence on consumer demand.[2] For example, while the industry spends R150 million per year on tobacco advertising (excluding sports sponsorship), less than R5 million per year is spent on all health promotion — this results in the population's knowledge being determined by the industry. Government intervention is required not only because of market imperfections, but also because of the strong externalities caused by tobacco control programmes, i.e. since tobacco affects non-smokers (through passive smoking) a control programme will have a multiplier effect on the population.[2,3] There is therefore a case for an increased tobacco tax, which should be part of an integrated, comprehensive control programme.[1-3]

Price is the single most important determinant of the demand for tobacco products and thus taxation has a great potential as a deterrent of smoking.[4-5] While estimates of the price elasticity of cigarettes (i.e. how responsive demand is to price changes) vary between countries, there seems to be a growing consensus in developed countries that a 10% increase in price will result in at least a 4% decline in demand. Teenagers are particularly price-sensitive, with evidence in the UK suggesting that a 10% increase in price is associated with a 14% decline in cigarette consumption among teenagers.[4]

In South Africa, since the retail price of cigarettes has

EDITORIAL / VAN DIE REDAKSIE

lagged behind the overall consumer price index over the past decade, cigarettes have become more affordable![2] Several studies have shown that the demand for cigarettes appears to react asymmetrically to falling as opposed to rising prices; the demand is twice as responsive to falls as to increases in real price.[5,6]

Tobacco tax has many benefits. The least important of these is an attempt to cover the true costs induced by smoking. Smokers are likely to die at twice the rate of non-smokers (even under the age of 65 years); during their working years, energy and productivity of workers are wasted. But the real reason to try and reduce consumption is not usually economic but the increased disease and suffering caused by tobacco products.

The benefits of tobacco tax include increased revenue gained by the government, i.e. taxation constitutes good fiscal policy. Secondly, it results in the decline in the prevalence of smoking and thus the costs associated with smoking-related death and disease. It does this by increasing the quit rate; stopping new recruits entering the market; and causing those who smoke heavily to reduce their smoking habit. Thus, taxation constitutes good health policy. Thirdly, there is public support for taxation. This has been found internationally in studies carried out in Canada,[5] Michigan State (USA),[7] and, more recently, in South Africa, where it was shown that 56% of all adults (smokers included) support an increased tax.[8]

Who supports an increased tax on tobacco? Besides support from the general public (the most important support), in recent months a range of organisations have called for an increased tax on tobacco. These include a group of academics from the University of Natal;[9] the editor of the *South African Medical Journal* and a senior member of the Medical Association;[10] the Past President of the College of Medicine of South Africa; progressive health organisations such as NPPHCN and SAHSSO;[11] and on several occasions the Tobacco Action Group and the Medical Research Council.

Who opposes an increased tax? Against these strong calls for taxation is the tobacco industry, represented by the Tobacco Institute, the Tobacco Manufacturers Association and selected media. A previous Director-General in the Department of National Health and Population Development commented that the tobacco lobby is the strongest lobby group in Parliament. That is the reason why no legislation has yet been tabled. The strong monopoly that the Rembrandt/Richmont group have in South Africa and its strong links to the business sector means that they have had an undue impact on the introduction of progressive legislation. Some of these forces are starting to weaken; and the need for extra revenue to balance the budget means that the probability of increased taxation will grow.

Fixing the level of taxation. In South Africa there is a great potential for an increase in tax on tobacco products. This is in line with the 1987 Margo Commission recommendation that excise duties be regularly adjusted. South Africa and the USA are almost alone in having extremely low tax on their cigarettes. In both cases less than 30% of the retail price is made up through taxation compared with around 50% in Mexico, Zimbabwe and Australia and 70% in Canada, New Zealand and Brazil.[2]

Tobacco taxes would need to constitute at least 50% of the pack price if they are to have a real impact on sales.[5] Increasing the proportion of an average-price pack to 50% would result in the sale price of cigarettes rising to around R3,20 for a pack of 20. The total excise revenue collected for 1991 (R818 million) would rise dramatically to R2,356 billion.[1] If it was assumed that consumption decreased by at least 20% (i.e. 0,5 price elasticity), a total amount of R1,885 billion would be received, resulting in an extra R1 billion revenue gained.

Concern has been voiced about the regressivity of the tax in developing countries. Evidence from developed countries suggests that there is a strong relationship between price elasticity and social class; for example in the UK the price elasticity for social class 1 is just about zero, for social class 2 it is –0,12, rising to –0,50 for social class 3 and –0,96 for social class 5.[4] Further, the same UK study showed that elasticity for women is greater than for men. This indicates that the poor and women are more likely to reduce consumption more rapidly as prices increase and thus the distributive effects of increased tax on the poor are likely to be minimised.

Regressive aspects of the tax could be overcome by ensuring that additional tax revenue is used to specifically target the poorest in terms of the provision of prevention and quit programmes.

Substitution effects of an increased tax. One might argue that an increase in the tax would result in a substitution effect. Smokers may switch to cheaper cigarettes or pipe tobacco or snuff. In South Africa the taxation on pipe tobacco is particularly low and over the last decade has decreased substantially relative to the taxation on cigarettes. Further, there is no taxation on snuff. Smokeless tobacco is currently being marketed in large amounts in the USA. A pre-emptive ban should be placed on the large-scale commercial production of this product. Increasing taxation on snuff, pipe tobacco and cigars will ensure that substitution is less likely. The increased revenue gained from taxation on these products is likely to be around R50 million.

Declines in consumption and revenue gained. There is always a concern that the tax will be so successful that smokers would cut consumption rapidly. The experience of countries like Canada, the UK and the USA shows that this is not the case. For example, between 1980 and 1986 in the UK the real price of cigarettes increased by 39%, consumption decreased by 24%, the number of smokers decreased by 15% but the total tax revenue increased by 14%![4] In Canada, over the period 1982 - 1991, with consumption decreasing by 40% government revenue increased from $2 to $7 billion.[5]

Earmarking the tax for health and social investment. The use of extra revenue needs to be carefully planned. None of the South African organisations who support an increase in tax have defined where the funds should be spent. It is crucial not to allow them to go into existing health expenditure areas such as tertiary care treatment of chronic diseases, but rather to use them to start health promotion programmes and fund key areas in primary health care not receiving attention.

Overall the recommendation would be that of the additional R1 billion, 50% be used by the Treasury for areas of high-priority social investment while the other 50% be used by the health sector.

The budget of the Department of National Health should not be reduced and merely replaced by extra tax revenue. Rather, the new tax revenue should be used for additional preventive/promotive programmes. One specific way of doing this would be to earmark at least R250 million of the remaining R500 million for health promotion activities, similar to those in Victoria State, Australia, California in the USA, and Canada. (In these places additional revenue has resulted in a windfall for the health sector, so that they were able to replace tobacco sports sponsorship with health promotion sponsorship.) The remaining R250 million could be used to extend primary health care in rural and peri-urban areas.

EDITORIAL / VAN DIE REDAKSIE

To control tobacco requires the strong support of the Ministries of Finance, Health and Education. In addition, the Ministry of Agriculture needs to be convinced that it should no longer subsidise tobacco production and tobacco research in a country where the health consequences are clearly apparent.

DEREK YACH
Essential Health Research Group
Medical Research Council
Pretoria

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Implications of bacterial resistance for the use of beta-lactam agents in clinical practice

The β-lactam antibiotics, e.g. penicillins, cephalosporins, monobactams and carbapenems, have been among the most useful available. Only allergy and rapidly emerging resistance have limited their clinical utility. Because bacteria have consistently modified their defences to match each new drug development, confusion exists among clinicians as to which antibiotic to use and when.

Resistance develops via one or more of three mechanisms. These involve alterations in penicillin-binding proteins (PBP) or the outer membrane structure or the elaboration of β-lactamase (BLA) enzymes.[1]

The PBPs. These are enzymes involved in bacterial cell wall synthesis and are responsible for growth, maintenance of shape and replicative function. β-lactam antibiotics bind to PBPs and, by competing with their natural substrates, cause cellular fragility and induce lysis.[2,3] Reduced affinity of PBP for β-lactam antibiotics is seen in those organisms unable to produce BLA, such as pneumococci or *Staphylococcus aureus* where BLA production has been limited by use of BLA-resistant antibiotics such as methicillin.[2,4] This also occurs in Gram-negative bacteria, exposed to β-lactams for years but in which BLA production has been rare, e.g. *Neisseria gonorrhoeae* and *Haemophilus influenzae*.[2,5] Coincidentally the alteration of PBP structure in these mutants also reduces affinity for its natural amino acid substrates so that the cell wall may be weakened to the point of non-viability.[5,6]

Porins. These are channels in the outer membrane of Gram-negative organisms through which small water-soluble molecules like β-lactams pass to reach the PBP; the PBPs of Gram-positive organisms are open to the environment.[2,7] If an antibiotic cannot penetrate the porins it obviously cannot interact with PBPs. Relative impermeability commonly coexists with enhanced BLA activity, but the loss of a specific porin protein may make an organism completely impermeable to an antibiotic such as imipenem. This form of imipenem resistance cannot, however, confer resistance to antibiotics other than the carbapenems.[8,9]

Beta-lactamases. These are capable of hydrolysing the β-lactam ring of β-lactam antibiotics. Although modification of adjacent rings or side chains affords protection against some or all of these enzymes and increases potency and spectrum, resistance still develops, particularly in Gram-negative organisms. BLA enzymes are encoded either by the bacterial chromosome or by transferable extrachromosomal plasmids which are acquired on contact with resistant species.[10-12] These enzymes differ biochemically, genetically and functionally. The rapid emergence of plasmid-mediated resistance to the broader spectrum antibiotics, such as ampicillin and the first-generation cephalosporins, stimulated the production of a new generation of antibiotics largely unsusceptible to hydrolysis by these enzymes, viz. the ureidopenicillins, the second- and third-generation cephalosporins, the monobactams and the carbapenems.[10,11] In response, some Gram-negative bacteria began to produce excessive amounts of chromosomally mediated BLA which were capable of hydrolysing all of the new antibiotics except imipenem.[12,13] On the basis of the type of BLA elaborated, there exist two distinct groups of Gram-negative bacteria.[1,10,11,13]

Group I includes the common community-acquired organisms such as *Escherichia coli, Streptococcus pneumoniae, Proteus mirabilis* and *H. influenzae* which, because they cannot increase their small basal output of chromosomally mediated BLA, are dependent upon the acquisition of plasmids.[1,10] Those members that have not acquired plasmids retain susceptibility to amoxycillin and first-generation cephalosporins, while those that have are still susceptible to second-generation cephalosporins or amoxycillin/clavulanate combinations (clavulanate, while having little intrinsic antibacterial function, binds irreversibly to plasmid-mediated BLA and, like sulbactam and tazobactam, is termed a suicide inhibitor). Plasmid-mediated resistance is largely an all-or-nothing phenomenon; it is either present or it is not and it cannot be increased in response to the presence of an antibiotic. Recently, some of this group have acquired plasmids that code for extended spectrum BLA capable of hydrolysing many of the newer antibiotics that are stable in the presence of the older variety.[1] These have been identified primarily in nosocomially acquired *Klebsiella* species and should not alter present prescribing practices among those managing community-acquired infection.[1]

Group II includes the common nosocomially acquired organisms such as *Enterobacter* spp., *Citrobacter freundii, Pseudomonas* spp., *Proteus vulgaris, Morganella* spp., *Serratia* spp. and the various non-fermenters such as *Acinetobacter* and *Alkaligenes* spp., which are a major problem in intensive care units.[11,15] These organisms

EDITORIAL / VAN DIE REDAKSIE

produce a basal amount of chromosomal BLA which can be increased exponentially in the presence of certain antibiotics by means of a process termed induction.[11-15] The strongest inducers of BLA are ampicillin, the first- and second-generation cephalosporins (excluding cefuroxime) and imipenem; with the exception of the latter, which has a great natural resistance to hydrolysis, these should not be used against group II organisms.[11,16,17] Mutants exist within this group and modify a regulatory gene by means of a process called derepression to express high levels of BLA constitutively.[11,15,17,18] While this stably derepressed organism is resistant to most β-lactam antibiotics, it retains susceptibility to imipenem.

The classification of β-lactam antibiotics. David Livermore has divided these drugs into four groups in terms of their ability to induce chromosomal BLA in group II organisms and on their sensitivity to hydrolysis.[11]

1. *The labile strong inducers* are best characterised by ampicillin and cefoxitin. These drugs are ineffective against group II organisms as they are hydrolysed by BLA of stably derepressed mutants and those that they induce.

2. *The labile weak inducers* are best characterised by the ureidopenicillins (e.g. piperacillin), aztreonam, the second-generation cephalosporins (excluding cefoxitin) and the third-generation cephalosporins. Members of this group, while extremely susceptible to hydrolysis by BLA, do not themselves induce production of significant amounts. It is axiomatic that none of these agents should be used in conjunction with strong inducers such as cefoxitin, ampicillin or imipenem. This group has the potential to select for stable derepressed mutants because they are protected by their constitutive high level of BLA production. This mechanism is the commonest cause of emerging resistance to most newer β-lactam antibiotics except imipenem.[11]

3. *The stable strong inducers* are represented only by imipenem at present, though new carbapenems such as meropenem are on the horizon.[19] Because of its resistance to hydrolysis both inducible strains and derepressed mutants remain susceptible, with no selective advantage for the latter.

4. *The stable weak inducers* are potentially the ideal drugs and it is unfortunate that no good examples thereof exist.

Recommendations. These cannot satisfy every scenario, and consequently an understanding of the antibiotics available to us and awareness of the susceptibilities of organisms within each environment are essential.

If the organism is, or is likely to be, Gram-positive, or of the group I, Gram-negative, non-inducible variety (i.e. those responsible for the great majority of community-acquired infections), amoxycillin or first-generation cephalosporins remain the β-lactams of choice except in cases of staphylococcal infections where cloxacillin is preferred. The use of clavulanate combinations or second-generation cephalosporins, e.g. cefuroxime, is especially indicated in situations where the most commonly encountered organisms are resistant to amoxycillin or the first-generation cephalosporins. Third-generation cephalosporins, particularly the new oral preparations, are only occasionally required for community-acquired infections, regardless of their severity.[19] In addition, the use of 4-quinolones as first-line therapy for community-acquired infection cannot be recommended, both for reasons of expense and antibiotic resistance spectra at this time.

If the organism is, or is likely to be, a group II (inducible) variety, therapy should be based on the pattern of susceptibility within each unit. Therapy is usually initiated with piperacillin or third-generation cephalosporins if susceptible and if not, the 4-quinolones or imipenem must be considered. Aminoglycosides are particularly indicated as combination therapy in neutropenic patients or in proven or presumptive *Pseudomonas* or enterococcal infection.[20-23] If *Klebsiella* infections with extended spectrum BLA are encountered in a particular unit, it is possible that they may retain susceptibility to cefoxitin or to combination therapy with a β-lactamase inhibitor such as clavulanic acid, despite resistance to third-generation cephalosporins.

This has been an overview of the current status of β-lactam antibiotics. Far from being outdated they remain the mainstay of antimicrobial chemotherapy and if used correctly, may remain so for many years.

G. A. RICHARDS
K. P. KLUGMAN

Departments of Medicine and Medical Microbiology
University of the Witwatersrand
Johannesburg

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EDITORIAL / VAN DIE REDAKSIE

Quality of care — and debate

A vitally important piece of legislation which, when it comes into effect, will have a profound effect on the provision of health services in South Africa, was recently debated in Parliament. This was the Medical Schemes Amendment Act which, as the Minister of Health explained during the course of the debate, 'aims to deregulate medical schemes so that free-market principles can operate. This is essential to enable medical schemes to offer health care cover suited to the real needs and financial means of their members'. Clearly, this was a piece of legislation of crucial interest to doctors, paving the way as it did for alternative methods of health care delivery such as HMOs to operate, and also, again as the Minister explained, 'to extend the ability of the private sector to provide cover for a larger percentage of the population in order for the State, on the other hand, to provide more adequately for the indigent by means of primary and secondary health care'. Whether one agrees with the philosophy expressed by the Minister is of course open to debate, given the current rapidly changing social and political scenario in the country, particularly by those who are not wedded to the notion of the free-market system as applied to health care, and who see in such an outlook the inherent dangers of a two-tiered system in health care delivery. However, given the terms of the Act, a tremendous amount of work needed to be done by the MASA, and the midnight oil was consumed in vast quantities while the minutiae of the Act were gone through with a fine toothcomb to ensure that the Act did not have any unexpected stings in its tail which could interfere with the basic social contract between the individual doctor and patient to the detriment of them both. There is little doubt that the MASA made a great impact on the debate in Parliament, largely due to the meticulous care with which it had done its homework, and its understanding of the somewhat convoluted way in which Parliament carries out its business.

It was therefore extremely unfortunate that what should have been a debate carried out at a high level of technical expertise and understanding of the needs of patients should have been marred by allegations made by the Minister at the beginning of the debate when she was reported as saying, '. . . As 'n voorbeeld hiervan wil ek graag 'n opname gebruik van die ongeveer 200 geneeshere wat die hoogste inkomste uit mediese skemas eis. Ek is nie van plan om die name van die geneeshere te noem nie, maar as 'n mens na die dokument kyk, is dit duidelik dat die vanne wat daar staan 'n weerspieeling is van die volle spektrum van die bevolking'. What was particularly damaging about this statement is that by making such an allegation without submitting any evidence for it, without calling for an investigation and without naming the doctors concerned, the Minister placed every single doctor in the country under suspicion. The situation had also been confused at a media briefing held prior to the debate in Parliament by the Minister giving the impression that she would give the names of the doctors concerned to the South African Medical and Dental Council for investigation, but afterwards denying that she was going to do so.

Naturally, the mass media, who were in fine doctor-bashing form, had a field day, and the Minister's allegations were given widespread publicity. Not surprisingly, the vast number of doctors who customarily put in long and arduous hours of devoted patient care were incensed at being tarred with the same brush that had been used on the alleged black sheep, and have been looking to the MASA for an appropriate response which, however, was not as easy to make as might at first appear. By simply taking the easy way out and condemning the Minister's statement out of hand, it could fall into the trap of appearing to condone the actions of which the Minister has accused the alleged small minority of 'trading' doctors who, it is being implied, have been bending the medical aid system to their own financial advantage. On the other hand, to do nothing could be taken as evidence of tacitly agreeing with what the Minister had said — an equally untenable position.

In actual fact, the ball does not at present lie in the MASA's court at all but in the Minister's, who has not finished what she started. If she actually possesses the names of 200 doctors together with evidence that they have been engaged in what amounts to fraud, then her clear duty is surely to forward those names together with her evidence to the South African Medical and Dental Council for investigation and appropriate disciplinary action. If the evidence of fraud is strong enough, she might even consider sending her evidence to the Attorney-General. What she cannot do, however, is to smear an entire profession by making serious allegations about the ethical standards of some of its members and then not following them up with definitive action.

By refusing to indulge in the traditional knee-jerk response to ill-judged criticism of the profession, the MASA is exhibiting commendable judgement, restraint and political maturity, preferring to devote its not inconsiderable energy towards tackling health problems at their roots. It is fortuitous that the Association is just about to unveil a major programme, which addresses the quality of medical care that patients receive and how it can be maintained at a high level. Such an initiative comes at a critical juncture, when reallocation of available resources with its concomitant uncertainties and upheavals could easily lead to a deterioration in the standard of individual patient care. The main objectives of the quality of care programme will be, firstly, to improve the quality of medical care available to the South African public, secondly, to improve the self-regulatory capacity of the medical profession and, thirdly, to assist the profession to play its part in any initiative towards equity and cost containment. Announcing the programme is one thing. Making it work is another, but if it can be made to work as planned, it should have a major impact on standards of medical care in this country, and should convince even the Minister that the MASA is serious about its sincere dedication to the health needs of patients.

N. C. LEE

EDITORIAL / VAN DIE REDAKSIE

Alcohol and brain damage

Alcoholism may constitute the major health problem in many parts of the world, with its effects on morbidity and mortality grossly underestimated in world health statistics.[1] Alcohol-related neuropsychiatric disorders constitute a particularly large and incapacitating subset of the medical complications of alcoholism.[2] There are a number of well-described neuropsychiatric syndromes associated with alcohol withdrawal (e.g. withdrawal delirium, hallucinosis, seizures) or concomitant nutritional deficiencies (e.g. Wernicke-Korsakoff syndrome, polyneuropathy, pellagra).[3]

Alcoholic brain damage has traditionally been viewed in straightforward terms. Thus, Wernicke-Korsakoff syndrome has long been regarded as the classic form caused by vascular lesions in di-encephalic structures as a result of thiamine deficiency. Wernicke's encephalopathy is the acute component, manifesting clinically as impairment of consciousness, accompanied by ataxia and ophthalmoplegia, while Korsakoff's syndrome is the chronic sequel of the same pathological process and is characterised by profound retrograde and anterograde amnesia.[4] Research has shown that memory deficits of patients with Korsakoff's syndrome are not entirely pure and that other cognitive functions, especialy visuoperceptive and problem-solving capacities, are also impaired. There appears to be marked variability in the degree of cognitive impairment, with female patients particularly severely affected.[5] Wernicke-type pathology is far more common than is generally recognised, and may develop surreptitiously. In a neuropathological study in Western Australia, morphological evidence of the disorder was found in 1,7% of *all* autopsies performed, only the minority of whom had been diagnosed as such during life. The Wernicke-Korsakoff syndrome was, until fairly recently, thought to account for the majority of alcoholics who suffered lasting cognitive impairment, and those who escaped this development were regarded as being essentially 'intact'.[6]

Evidence from neuropathological and neuroradiological studies was, however, rapidly accumulating to show that diffuse brain damage can occur in alcoholics.[3] This brain damage is not always clinically obvious, but refined psychological tests have been able to demonstrate that a large proportion of alcoholics actually do have impaired cognitive functioning.[6] Heavy consumption of alcohol results in the increasing danger of cognitive impairment. In fact, there is some evidence to suggest that such impairment may be detectable even in the so-called social drinker![7] The frontal lobes appear to be most severely affected by chronic, excessive consumption of alcohol.[8] Frontal lobe dysfunction may manifest with symptoms such as decreased ability to make abstractions, lack of insight and impaired impulse control — functions that are of critical importance in any attempt to rehabilitate the alcoholic. In this way the development of even subtle brain damage may be a significant factor in the perpetuation of the alcoholic's dependency problem.

Computed tomographic (CT) studies have shown that alcoholics have enlarged ventricles and widened cortical sulci.[9] These changes are broadly associated with cognitive impairment.[5] A great number of alcoholics — perhaps the majority of severe alcoholics — impair their brains quite early on. This impairment, although structurally demonstrable, appears in most cases to remain relatively benign over many years and, to some degree, is potentially reversible by abstinence.[10] As the alcoholic gets older, however, perhaps as the result of the alcoholism's interaction with other pathological conditions (e.g. ageing, trauma, hepatic dysfunction, vascular changes), he may develop a more severe global impairment of cognitive function — so-called 'alcoholic dementia'.[4]

It has been proposed that two separate pathological processes may contribute to brain damage in alcoholics: (*i*) severe thiamine deficiency because of dietary neglect, poor absorption and utilisation for the metabolism of alcohol, with consequent di-encephalic damage and memory impairment; and (*ii*) cortical shrinkage caused directly by alcohol neurotoxicity, which leads to widespread cognitive impairment.[11] Thus, Korsakoff's syndrome may in fact represent only the tip of an iceberg; many other alcoholics may be affected to a lesser degree by the same pathological processes.[6]

Unfortunately, the causes of alcoholism are still largely unknown, and its treatment remains unsatisfactory. Priorities are primary prevention and early identification and treatment of those at risk. The public at large needs to know of the hazards involved. Therapeutic intervention needs to take place before physical dependence and cognitive impairment compound an already difficult task.

ROBIN EMSLEY
Department of Psychiatry
University of Stellenbosch

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