Selective posterior rhizotomy in the treatment of spasticity

Spasticity as a clinical phenomenon is generally considered to entail a state of hyperactive deep tendon reflexes, increased resistance to stretch as the velocity of stretch increases, the clasp-knife phenomenon, the presence of clonus and a reduction in the range of limb movements. All these elements indicate an exaggeration of the reflex response to muscle stretch, the degree and severity of which depend on the suprasegmental structures involved.

Sherrington[1] found that interruption of the posterior nerve roots would neutralise the characteristic hypertonus in decerebrate cats. On the basis of this work, Otfrid Foerster[2] sectioned the posterior nerve roots for relief of spasticity, particularly in cerebral palsy (static encephalopathy) but also in other conditions. The aim of rhizotomy was to relieve disabling spasticity but retain residual neurological function. Foerster[2] reported on 159 cases, including 88 of spastic diplegia treated by posterior rhizotomy. For the procedure to be successful, it had to be performed on patients in whom the basic morbid process was static. Spastic symptoms were relieved by the operation, but not the paralysis itself, and care had to be taken not to convert a spastic into a flaccid paralytic. The importance of 'useful' spasticity of the quadriceps femoris, the innervation of which was identified by electrical stimulation, was emphasised. A long, dedicated postoperative regimen of training and exercise was needed to rehabilitate these patients.

Persistent and disabling sensory disturbance as well as trophic ulceration following posterior root section brought this operation into disfavour. A modified anatomically selective posterior rhizotomy in which four out of five rootlets were sacrificed in order to preserve proprioceptive sensation was introduced by Gros *et al.*[4] Some sensory loss and weakness still occurred in a significant number of patients, as some remained spastic postoperatively.

Anatomical selective hemisection of posterior nerve roots in preserving sensation[5] suffered the same drawbacks as Gros' procedure. Fasano *et al.*[6] introduced posterior rootlet stimulation and, on the basis of abnormal electromyographic responses, identified those nerve roots involved in abnormal feedback activities that were regarded as dispensable by means of selective sectioning.

This technique was refined in Cape Town[7] and the present upsurge in its popularity is the consequence of the promising results obtained by Peacock and coworkers.[8] Innovations such as a modified laminectomy, clarification of the anatomical landmarks in rootlet localisation and refinement of intra-operative electromyographic (EMG) monitoring, have resulted in excellent selective rootlet section and reduced the undesired negative aspects, yet achieved adequate relief of spasticity.

Patient selection is as vital to the success of this operation as rootlet section. More objective modes of functional assessment, such as gait analysis and motional assessment, EMG and force plate assessment, are being used more widely and information will be of assistance in objective assessment pre- and postoperatively.

The patient must have true spasticity as other manifestations of cerebral palsy will not be improved by rhizotomy. This operation is a significant intervention in the life and functional ability of patients with variable degrees of disability. Surgeons, patients, relatives and all other therapists involved in management must have a clear understanding of reality, expectations, risks and future roles of all participants. Relief of spasticity is but one aspect of the total management of these patients.

Reduction of spasticity has been the experience of the vast majority of workers in this field[7,9,10] and most of them noted an improvement in the ambulation of those who had been able to walk pre-operatively.[11]

Weakness in the early postoperative phase may be due to the unmasking of pre-existing weakness or to inappropriate rootlet section. Weakness usually improves in the course of time with intensive physiotherapy.

Sitting, standing and walking are usually improved, but occasionally may be worsened if there is excessive flaccidity or loss of 'useful' spasticity. Occasionally, improved motor function may be demonstrable with the relief of spasticity which had masked such function preoperatively. Unexpected apparent improvement in functions, such as better seizure control, improved sphincter control and improved motivation,[10] needs to be confirmed by careful postoperative assessments.

Even in selective functional rhizotomy, sensory disturbance in the legs and sides of the feet is common immediately after operation. Touch and paraesthetic discomfort may last up to a year and even be permanent in some patients.

Postoperative complications are few in selective posterior rhizotomy. In the large Cape Town series there was no mortality and the occasional postoperative cerebrospinal fluid leak did not prove a problem. Incontinence is not a problem with root section above S2.

It was feared that spinal deformity might be a complication of long lumbar laminectomy (L2-S1), and scoliosis and hyperlordosis occur with equal frequency in children with cerebral palsy following rhizotomy and those not operated on.[12] Spondylolysis and spondylolisthesis do, however, occur in some children who have had a lumbar laminectomy, and are probably attributable to their greater postoperative mobility. They seem to be static conditions, but a longer follow-up is necessary to establish how they will progress.[13-15]

Rapidly progressive subluxation of the hip has been reported[16] but has been found only in those children with pre-operative hip dysplasia.

Meticulous patient selection that aims to identify those patients who will benefit most from surgery is important. Patients who are able to stand and walk, albeit with difficulty, are those who fare best. Reduction of extreme spasticity in order to render a bedridden patient more manageable or more easy to nurse, are factors difficult to assess, although real for those who have to live with them from day to day. Most relatives and therapists who have given an opinion after the operation have been in favour thereof.

Intra-operative monitoring of nerve root function by electrophysiological means has greatly improved the selection of rootlets to be sectioned but has not been standardised,[17] and quantification of these modalities would improve evaluation of results.

The significance of spasticity as a contributing factor in the disability suffered by some cerebral palsy patients has been questioned, but this is difficult to understand as postoperative clinical evaluation seems to prove that it does.

It has been suggested[17] that the improvement observed after surgery in young children with minimal dysfunction would have occurred in any event with maturation of the nervous system and physiotherapy. The beneficial effects of selective posterior rhizotomy in teenagers and young adults[7] and the improvement in adults,[3,18] however, indicate that improvement is not only due to maturation and physiotherapy. To conduct controlled trials on patients with a disease as variable as this will not be easy.[19]

In the USA where, to an ever greater extent, the clinical piper's tune is called by those who pay, it was questioned whether this procedure was experimental or

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investigational. The best evaluation of the procedure was probably the one provided by the American Society of Pediatric Neurosurgery.

Selective posterior rhizotomy is not an experimental procedure; it is safe and effective for the reduction of spasticity, with documented long-term functional benefit, and is useful in the management of some patients with spastic cerebral palsy.

The management of patients with cerebral palsy is best carried out by a team of health care providers and selection criteria for selective posterior rhizotomy are best decided via team evaluation of patients. Major operations on the nervous system of children should be performed at centres that provide all aspects of paediatric care with active paediatric neurosurgery, orthopaedic and rehabilitative services. Selective posterior rhizotomy and orthopaedic intervention are not mutually exclusive in the surgical management of cerebral palsy.

Posterior rhizotomy is therefore a safe and effective procedure for alleviating, at least to some extent, the plight of selected patients with cerebral palsy.

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1. Sherrington CS. Decerebrate rigidity and reflex co-ordination of movements. *J Physiol (Lond)* 1898; **22:** 319-332.
2. Foerster O. Uber eine neue operative Methode der Behandlung spastischer Lähmungen mittels Resektion hinterer RM Wurzeln. *Z Orthop Chir* 1908; **22:** 202-223.
3. Foerster O. On the indications and results of the excision of posterior spinal nerve roots in men. *Surg Gynecol Obstet* 1913; **16:** 463-475.
4. Gros C. Spasticity — clinical classification and surgical treatment. *Adv Tech Stand Neurosurg* 1979; **6:** 55-97.
5. Fraioli B, Guidetti B. Posterior partial rootlet section in the treatment of spasticity. *J Neurosurg* 1977; **46:** 618-626.
6. Fasano VA, Broggi G, Barolat-Romana S, Sguazzi A. Surgical treatment of spasticity in cerebral palsy. *Childs Brain* 1978; **4:** 289-305.
7. Peacock WJ, Arens LJ. Selective posterior rhizotomy for the relief of spasticity in cerebral palsy. *S Afr Med J* 1982; **62:** 119-124.
8. Peacock WJ, Arens LJ, Berman B. Cerebral palsy spasticity. Selective posterior rhizotomy. *Pediatr Neurosurg* 1987; **13:** 61-66.
9. Fasano VA, Broggi G. Functional posterior rhizotomy — State of the Art Reviews. *Neurosurgery* 1989; **4:** 409-412.
10. Peacock WJ, Staudt LJ. Selective posterior rhizotomy: evolution of theory and practice. *Pediatr Neurosurg* 1991; **17:** 128-134.
11. Palmer FB. Physical therapy: objective assessment. *J Pediatr Orthop* 1991; **11:** 689-690.
12. Peter JC, Hoffman EB, Arens LJ, Peacock WJ. Incidence of spinal deformity in children after multiple level laminectomy for selective posterior rhizotomy. *Childs Nerv Syst* 1990; **6:** 30-32.
13. Peter JC, Arens LJ. Selective posterior lumbosacral rhizotomy for the management of cerebral palsy spasticity: a 10-year experience. *S Afr Med J* 1993; **83:** 745-747 (this issue).
14. Peter JC, Arens LJ. Selective posterior lumbosacral rhizotomy in teenagers and young adults. *J Neurosurg* (in press).
15. Peter JC, Hoffman EB, Arens L. Spondylolysis and olisthesis after 5 level lumbo-sacral laminectomy for selective posterior rhizotomy in cerebral palsy. *Childs Nerv Syst* 1992; **8:** 158.
16. Greene WB. Dietz FR, Goldberg MJ, Gros RH, Miller F, Sussman MD. Rapid progression of hip subluxation in cerebral palsy after selective posterior rhizotomy. *J Pediat Orthop* 1991; **11:** 494-497.
17. Newberg N. Gooch JL, Walker ML. Intraoperative monitoring in selective dorsal rhizotomy. *Pediatr Neurosurg* 1991/92; **17:** 124-127.
18. Laitinen LV, Nilsson S, Fugl-Meyer AR. Selective posterior rhizotomy for treatment of spasticity. *J Neurosurg* 1983; **58:** 895-899.
19. Goldberg MJ. Measuring outcomes in cerebral palsy. *J Pediatr Orthop* 1991; **11:** 682-685.
20. Reigel DH. Editorial. *Pediatr Neurosurg* 1990/91; **16:** 1-2.

Prevention of congenital syphilis by effective maternal screening at antenatal clinics

Syphilis remains a significant health problem in South Africa. A serosurvey conducted by the Department of National Health and Population Development (DNHPD) at the end of 1991 showed that the prevalence rate of maternal syphilis was 6,6%.[1] The primary aim of the survey was to determine the point prevalence of HIV infection in women attending antenatal clinics.[1] The survey sampled all population groups within the four provinces of South Africa and the self-governing and independent states; 16 370 subjects were tested.[1] The prevalence rates for syphilis for the black and coloured groups were 7,1% and 8,1% respectively, while the Asian and white groups had prevalence rates of 1,2% and 0,7% respectively.[1]

In the past, untreated maternal syphilis at delivery was regarded as a disease of pregnant women who had received no antenatal care during pregnancy. However, data from two studies conducted at Kalafong Hospital have shown that over 90% of mothers with untreated syphilis at the time of delivery had indeed received antenatal care (S. D. Delport — unpublished data). Similar findings have been reported from Pelonomi Hospital, Bloemfontein, where 15,6% of patients attending the antenatal clinic had positive serological tests for syphilis (STS) and 43% of these had received no treatment from their antenatal caregivers.[2] At the Khayelitsha clinic in Cape Town a mere 51% of 70 patients received the complete course of treatment, because of attrition rates at each stage of the diagnostic and treatment process.[3]

Although screening for syphilis is an undisputed and essential part of antenatal care, this principle is often not applied or applied ineffectively. At present, in the majority of cases, blood is taken during the first antenatal visit and dispatched to a referral laboratory for STS. Mothers are generally advised to return 2 weeks later, by which time the results should be available. Unfortunately many mothers may not return, especially if, during their first visit, they were not counselled about the importance of the test. Another problem is that many mothers only start to attend well into their third trimester. If they were to experience delays in receiving treatment, these could prove fatal to the fetus. In addition, results are often lost, and repeat testing can result in a further delay in the provision of therapy, thereby adding to the risk of vertical transmission of syphilis to the fetus.

The significance of the morbidity and mortality caused by congenital syphilis was brought to the attention of the DNHPD. As a result congenital syphilis became a notifiable disease in 1991.[4] The feasibility of testing for syphilis on site in antenatal clinics was then seriously considered.

A screening test for syphilis performed on site with results available immediately eliminates the delay in treatment. The rapid plasma reagin (RPR) card test, originally developed to be performed under field conditions without sophisticated equipment[5] was suggested as the ideal screening test. This test is easy to perform, cheap and widely available.

A study to evaluate the sensitivity, specificity and utility of an RPR test performed on site in a referral hospital is reported in this issue of the *SAMJ*.[6] Ninety-two per cent of pregnant women with positive STS were identified during their first visit to an antenatal clinic by means of an on-site RPR test. Appropriate therapy was administered immediately, the cost of which was minimal.[6] The study was subsequently repeated in two peripheral clinics where no sophisticated laboratory equipment was available. Under these conditions the test proved as sensitive but with improved specificity. Treatment could be provided during the first antenatal visit to 86% of patients who required it (S. D. Delport — unpublished data).

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As a result, a small working group was formed by the DNHPD to consider proposals for a national policy for the diagnosis and treatment of maternal syphilis in order to prevent congenital syphilis more effectively. The suggested policy for consideration and debate is as follows:

1. Screening for syphilis with the RPR card test should be available to all clinics providing antenatal care, so that results can be made available immediately. If clinics have immediate access to laboratory facilities, the latter should be responsible for performing the RPR test. The results should be reported the same day. At peripheral clinics, the clinic staff should perform the RPR test on site and report it as positive or negative. Positive serum should be dispatched to referral laboratories for titration. Results should be made available to the clinic within 1 week. RPR titres should be documented on patient-retained records for future reference.
2. Patients with positive on-site RPR tests should receive counselling about the implications of the result and the importance of treatment.
3. Treatment should be provided immediately to all patients with positive tests. An intramuscular injection of 2,4 mU benzathine penicillin G should be administered and patients should return at weekly intervals for two further injections.
4. Ideally, consorts should be screened, counselled and treated by the clinic treating the mother.
5. Patients with negative on-site RPR tests need no serological confirmation at a referral laboratory.
6. All patients should be rescreened at delivery to diagnose seroconversion in previously seronegative mothers and to monitor changes in RPR titres which could indicate either reinfection or successful treatment.
7. In groups with a low prevalence of syphilis a positive on-site RPR test should be confirmed with a positive specific test for syphilis before treatment is commenced.

Before official implementation of these guidelines, pilot studies will be undertaken to assess performance and feasibility of on-site testing in different settings and to establish quality control measures.

Financial and manpower constraints are major factors leading to poor compliance in population groups with a high prevalence of syphilis. Free clinic services which are publicised and accessible to the majority of the population, and which operate within flexible time schedules, may accommodate the needs of these groups. However, until such flexibility exists, a concerted effort should be made to meet their needs and address their constraints with the resources available. The RPR test performed on-site at antenatal clinics throughout the country may form part of a solution to the problem of inadequate testing for syphilis in pregnant women.

The high rates of morbidity and mortality caused by congenital syphilis in South Africa should not be tolerated, given the availability of an inexpensive, easy test and effective treatment. Despite the fact that considerable funding is at present being channelled into antenatal screening for maternal syphilis in South Africa, the rates of congenital syphilis are still unacceptably high. Those involved in providing antenatal services should consider testing the above proposals in their own settings. Feedback regarding individual experiences will be appreciated.

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1. Department of National Health and Population Development. Syphilis in women attending antenatal clinics October/November 1991. *Epidemiological Comments* 1992; **19** (10): 165-170.
2. Barn RH, Cronjé HS, Muir AR, Griessel DJ, Hoek BB. The prevalence and outcome of positive serology for syphilis in antenatal patients. In: Rotherberg A, ed. *Proceedings of the Eleventh Conference on Priorities in Perinatal Care in South Africa*. Johannesburg: University of the Witerwatersrand, 1992: 6-7.
3. Swingler GH, Van Coeverden de Groot H. The antenatal prevention of congenital syphilis in a peri-urban settlement. *S Afr Med J* 1993; **83:** 34-35.
4. Department of National Health and Population Development. Declaration of medical conditions to be notifiable medical conditions in terms of section 45 of the Health Act, 1977 (Act No. 63 of 1977). *Government Gazette* 1991; No. R.328.
5. Portnoy J, Brewer JH, Harris AD. Rapid plasma reagin card test for syphilis and other treponematoses. *Public Health Rep* 1962; **77:** 645-652.
6. Delport SD. On-site screening for syphilis in an antenatal clinic. *S Afr Med J* 1993; **83:** 723-724 (this issue).

Some reasons for the failure to notify congenital syphilis

Congenital syphilis remains a significant cause of pregnancy loss and neonatal morbidity and mortality in South Africa.[1] Approximately 10% of pregnant women attending antenatal clinics at academic institutions are Wassermann reaction (WR)-positive. In 1990, paediatricians successfully lobbied the Department of National Health and Population Development to have congenital syphilis declared a notifiable disease in order to improve data collection and facilitate assessment of the problem. This would enable South Africa to allocate resources appropriately, increase public awareness, alert health personnel, encourage on-site testing and prevent subsequent cases by following up and treating affected women and their consorts.[2] The Center for Disease Control (CDC) classification and treatment of congenital syphilis[3] have been recommended for use locally.[2] A recent survey indicates that less than 10% of cases are actually being notified,[4] and discussions at the 1993 Annual Conference on Priorities in Perinatal Care revealed that there is, in particular, a lack of consensus on the management and notification of *asymptomatic* congenital syphilis (babies born to mothers whose actual or recorded treatment is inadequate, irrespective of the findings in the baby). In order to evaluate the various interpretations of the guidelines recommended for South Africa, a survey was conducted at nine teaching hospitals (Baragwanath, Johannesburg, Tygerberg, Peninsula Maternal and Neonatal Services, H. F. Verwoerd, MEDUNSA/Ga-Rankuwa, Pelonomi/Universitas, King Edward VIII and Kalafong Hospitals).

It was generally agreed that notification of congenital syphilis is worth while for the reasons stated above. However, one unit feels that affected *mothers* should be notified instead, and another that notification is so

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poorly done as to be meaningless. While there is general agreement on the notification and treatment of *symptomatic* congenital syphilis, the only point of note is that in no case is the treatment altered by cerebrospinal fluid findings. This calls into question the recommendation that lumbar puncture (to detect neurosyphilis) be performed in all symptomatic infants.

The problem then lies with cases of *asymptomatic* congenital syphilis. In terms of the CDC classification, the South African situation is different from that in the USA. Here we have far greater numbers of affected infants, a less well-developed health service and severe financial limitations. Within the local context, the notification and treatment of asymptomatic cases can potentially result in an excessive case load and significant cost. According to the CDC classification, the asymptomatic category includes infants of mothers who have been partially treated or who have been fully treated but whose records are inadequate.[3] Many of these infants are actually uninfected; consequently there is the potential for overreporting and overtreatment. For these reasons four of the units surveyed would not notify asymptomatic cases of congenital syphilis. However, if one considers the goals of notification, then failure to notify these cases will result in an underestimation of the extent of the problem and represents a missed opportunity to follow up these infants and prevent infection in subsequent pregnancies. Notification of all WR-positive *mothers* may indeed be more appropriate but would actually increase the case load substantially.

The *management* of asymptomatic congenital syphilis is equally unclear. Some units do not investigate asymptomatic infants, others use maternal WR titre or adequacy of treatment as a guideline, and the remainder carry out a variety of investigations ranging from neonatal serological tests to lumbar puncture. Treatment varies from a single dose of bicillin to a full course of intravenous penicillin G. It is interesting to note that the four units which would not notify asymptomatic congenital syphilis would nevertheless treat the infants concerned; this indicates that they are indeed considered to be at risk. The majority (5/9) would not follow up asymptomatic cases. Others recommend follow-up, by a medical officer, clinic sister or local authority, for 6 weeks to 6 months.

While we have not established the precise reasons for the poor notification of congenital syphilis, we do have an idea of the confusion that exists in the case of asymptomatic neonatal disease. There is consensus on the need to notify symptomatic congenital syphilis and on the management of symptomatic cases. The approach to *asymptomatic* cases is unclear. If the goals of notification are to be met, then inclusion of all cases that fulfil the CDC criteria is necessary; however, this may well overburden the health services. As a step towards solving the problem, records need to be kept *and used* so that there is no unnecessary treatment of infants whose mothers have been adequately treated. Expectant mothers must be encouraged to attend antenatal clinics. Collaboration between members of the health team is essential; midwives have an important role to play, particularly in the management of asymptomatic cases. Perhaps interdepartmental differences in the notification and management of the asymptomatic infant will disappear if these goals are met. With regard to one aspect of treatment of the asymptomatic case, there is an indication that, contrary to CDC recommendations, treatment of asymptomatic cases with bicillin alone is considered acceptable and that such cases do not need follow-up;[5] however, this should be properly evaluated in a prospective manner. Results of this survey call into question the need for lumbar punctures in cases of symptomatic congenital syphilis as these do not influence management. Finally, on-site testing is obviously under-utilised and should be encouraged in all institutions, particularly where there is a high incidence of unbooked patients.

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1. Delport SD, Adhikari M, Van der Elst CW, Venter A. *S Afr Med J* 1989; **76:** 297-298.
2. Delport SD, Rothberg AD. Congenital syphilis — now a notifiable disease. *S Afr Med J* 1992; **81:** 288-289.
3. Centers for Disease Control. Congenital syphilis: New York City 1986-88. *MMWR* 1989; **38:** 825-829.
4. Ballot DE, Rothberg AD. Congenital syphilis as a notifiable disease. *S Afr Med J* 1993; **83:** 721-723 (this issue).
5. Paryani SG, Vaughn AJ, Lawrence S, Crosby M. Treatment of asymptomatic congenital syphilis: bicillin versus procaine penicillin. *Pediatr Res* 1991; **29:** 285A.

Towards a future policy for transplantation in South Africa

Transplantation is now universally accepted as the treatment of choice for end-stage organ failure. For patients suffering from corneal blindness or end-stage renal failure, transplantation is the only hope for a cure. In patients with hepatic, cardiac, pulmonary and pancreatic island failure, transplantation could provide a new lease on life.

The current restructuring of the health care system in South Africa is resulting in stringent financial restraints on tertiary care hospitals, necessitating a drastic cutback of services and staff. The future holds no hope of a reprieve. Despite the ever-increasing demand for health care services, priority will have to be given to housing, education and social upliftment.

Clearly the rationalisation of medical services in South Africa is essential and long overdue. Unfortunately, precious financial resources are still being wasted because of bureaucratic inefficiency, and the political legacy of duplicated health care systems. Sadly, even when savings are achieved or income is generated from private patients, academic hospitals do not benefit, as funds disappear into state coffers.

Transplantation is one of the aspects of tertiary care currently under scrutiny, and risks being severely affected in a future primary care-orientated system. Without doubt, transplantation is an expensive commodity, but it remains a far more cost-effective option than long-term methods of treatment such as haemodialysis. In the USA kidney transplantation costs a total of US$77 000 per patient by the end of the third post-transplant year. Cost per dialysed patient amounts to US$35 000 per year, i.e US$105 000 after 3 years. The difference in cost after 3 years is $28 000, rises to $75 000 by the end of the fifth post-transplant year, and continues to increase each year, clearly demonstrating that kidney transplantation is more cost-effective than ongoing dialysis.[1]

In a Third-World country like Pakistan, an estimated 10 000 new patients with end-stage renal failure require treatment yearly. Because of poor health infrastructure

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only 25% of these patients reach a dialysis facility. The cost of dialysis in Pakistan is US$2 400 per patient per year, whereas renal transplantation costs about US$4 000 during the first post-transplant year, US$1 500 in the second year and then levels off to around US$1 000 yearly. The cost of dialysis, however, continues to rise, due to foreign exchange fluctuations which dictate the price of imported disposables.[2]

In South Africa the cost per patient is estimated at R30 000 per annum for dialysis, versus R10 000 per annum for transplantation, calculated over a 3-year period (Tygerberg Hospital Renal Unit). It clearly makes sense, therefore, to continue with transplantation.

Unfortunately many misconceptions still exist among the general public as well as the medical profession regarding the role of transplantation. One of these is that only privileged members of society are selected to benefit from organs which are donated mostly by the underprivileged. This is simply not true, and although lack of facilities has necessitated stringent admission criteria in some transplant centres, organs are allocated equitably and fairly, based on internationally acceptable scientific methods. In the western Cape the ratio of transplant recipients among different population groups correlates well with the ratio in the general population.

Another common misconception is the transplantation is not very successful. The truth is that advances in medical technology and innovations in immunosuppressive therapy have resulted in a significant overall improvement in life expectancy and quality of life for all transplant recipients. Long-term survival rates now approach 85 - 90%. Remarkable rehabilitation is possible after transplantation, providing patients with an opportunity to return to work, to support their families and take their place in the community again.

Transplantation can no longer be regarded as high-tech or experimental medicine. Renal transplantation has been performed for almost 4 decades and cardiac transplants for more than 25 years — both have stood the test of time. Without access to transplantation facilities, an academic hospital cannot provide adequate training to postgraduate physicians, surgeons, urologists, anaesthetists and cardiologists, not to mention nursing, technical and paramedical staff. Without an active renal transplantation programme, there is no point in maintaining a dialysis programme, and without dialysis facilities, an academic hospital cannot function.

The commonly held perception that expansion of primary care facilities will prevent serious disease and cause tertiary care facilities to become redundant, does not strictly apply in cases of organ failure. Even in highly sophisticated Scandinavian and North American countries, despite adequate care, the number of transplant patients is increasing every year. There are currently almost 20 000 patients waiting for renal transplants in the USA.[3]

Cultural, ethnic and religious objections to organ donation and transplantation in this country are much less prevalent than they are made out to be. A survey by Pike *et al.*[4] showed that more than 70% of people from all population groups (urban and rural) are prepared to donate their organs.[4] The shortage of donor organs does, however, remain one of the major problems facing transplant units all over the world. Less than 20% of all potentially suitable donors (brain-dead individuals on life-support systems) are eventually utilised.[5] Lack of consent for donation is the reason for this wastage in approximately 25% of cases (unpublished data).

Furthermore, many organs are not utilised because of medicolegal requirements, signs of infection or other unpreventable problems. However, a large number (± 25%) of potential donors are never recognised as such, or simply not referred to transplant centres because of apathy among medical personnel. More than 1 000 patients in South Africa are on waiting lists for organ transplants at any given time, and many more are in need of corneas.

The transplant fraternity can do much more to improve donor utilisation in this country, by ensuring that organs (especially hearts and livers) which are not used locally, are offered to centres where these organs can be transplanted. Donor organs are a national resource and ideally a system should exist where equitable distribution of organs based on human leucocyte antigen matching, can be co-ordinated by an independent national body. Unfortunately geographical and financial constraints have prevented this from becoming a reality.

Legislation regarding organ donation is suboptimal, especially in cases where the name of the donor is not known or when the relatives cannot be located. Furthermore, a critical shortage of corneas for transplantation exists since a moratorium was placed on the removal of corneas in mortuaries.

Fortunately, through the effects of the Organ Donor Foundation, the proposed card-format driver's licence will include an area where willingness to be an organ donor can be indicated. This may improve public awareness and partly alleviate the shortage of donor organs.

The role of transplantation in the private sector is rather controversial and it is feared that many private patients will eventually become a burden to the state when medical aid funds run dry. The possibility that a financial motive may influence patient selection and organ allocation cannot be ruled out. Transplantations in private hospitals could, however, alleviate the patient load at state institutions, and be a viable option if sensibly planned in co-operation with local academic units.

Representation to the Minister of Health and other health policy-makers must be made, in order to obtain a commitment to maintain state funding of transplantation.

The proposed implementation of 'framework autonomy' in academic hospitals is unlikely initially to provide adequate funds to sustain a transplant programme. National funding, perhaps on a supra-regional basis, such as is seen in the British health care system, is essential to ensure a future for transplantation in South Africa.

The alternative would not only be to the detriment of the thousands of patients waiting for transplants, but would also lead to a disastrous deterioration in standards of medical care, and a further loss of professional expertise.

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1. Mendez R, Aswad S, Dessonk A, *et al.* Cost and financing of kidney transplantation in the United States. *Transplant Proc* 1992; **24:** 2127-2128.
2. Naqvi A, Hussain M, Askari H, *et al.* Economics of renal rehabilitation in Pakistan: a case for increasing transplantation activity. *Transplant Proc* 1992; **24:** 2125-2126.
3. Stuart FP, Veith FJ, Cranford RE. Brain death laws and patterns of consent to remove organs for transplantation from cadavers in the United States and 28 other countries. *Transplantation* 1981; **31:** 238-244.
4. Pike RE, Odell JA, Kahn D. Public attitudes to organ donation in South Africa. *S Afr Med J* 1993; **83:** 91-94.

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only 25% of these patients reach a dialysis facility. The cost of dialysis in Pakistan is US$2 400 per patient per year, whereas renal transplantation costs about US$4 000 during the first post-transplant year, US$1 500 in the second year and then levels off to around US$1 000 yearly. The cost of dialysis, however, continues to rise, due to foreign exchange fluctuations which dictate the price of imported disposables.[2]

In South Africa the cost per patient is estimated at R30 000 per annum for dialysis, versus R10 000 per annum for transplantation, calculated over a 3-year period (Tygerberg Hospital Renal Unit). It clearly makes sense, therefore, to continue with transplantation.

Unfortunately many misconceptions still exist among the general public as well as the medical profession regarding the role of transplantation. One of these is that only privileged members of society are selected to benefit from organs which are donated mostly by the underprivileged. This is simply not true, and although lack of facilities has necessitated stringent admission criteria in some transplant centres, organs are allocated equitably and fairly, based on internationally acceptable scientific methods. In the western Cape the ratio of transplant recipients among different population groups correlates well with the ratio in the general population.

Another common misconception is the transplantation is not very successful. The truth is that advances in medical technology and innovations in immunosuppressive therapy have resulted in a significant overall improvement in life expectancy and quality of life for all transplant recipients. Long-term survival rates now approach 85 - 90%. Remarkable rehabilitation is possible after transplantation, providing patients with an opportunity to return to work, to support their families and take their place in the community again.

Transplantation can no longer be regarded as high-tech or experimental medicine. Renal transplantation has been performed for almost 4 decades and cardiac transplants for more than 25 years — both have stood the test of time. Without access to transplantation facilities, an academic hospital cannot provide adequate training to postgraduate physicians, surgeons, urologists, anaesthetists and cardiologists, not to mention nursing, technical and paramedical staff. Without an active renal transplantation programme, there is no point in maintaining a dialysis programme, and without dialysis facilities, an academic hospital cannot function.

The commonly held perception that expansion of primary care facilities will prevent serious disease and cause tertiary care facilities to become redundant, does not strictly apply in cases of organ failure. Even in highly sophisticated Scandinavian and North American countries, despite adequate care, the number of transplant patients is increasing every year. There are currently almost 20 000 patients waiting for renal transplants in the USA.[3]

Cultural, ethnic and religious objections to organ donation and transplantation in this country are much less prevalent than they are made out to be. A survey by Pike *et al.*[3] showed that more than 70% of people from all population groups (urban and rural) are prepared to donate their organs.[4] The shortage of donor organs does, however, remain one of the major problems facing transplant units all over the world. Less than 20% of all potentially suitable donors (brain-dead individuals on life-support systems) are eventually utilised.[3] Lack of consent for donation is the reason for this wastage in approximately 25% of cases (unpublished data).

Furthermore, many organs are not utilised because of medicolegal requirements, signs of infection or other unpreventable problems. However, a large number (± 25%) of potential donors are never recognised as such, or simply not referred to transplant centres because of apathy among medical personnel. More than 1 000 patients in South Africa are on waiting lists for organ transplants at any given time, and many more are in need of corneas.

The transplant fraternity can do much more to improve donor utilisation in this country, by ensuring that organs (especially hearts and livers) which are not used locally, are offered to centres where these organs can be transplanted. Donor organs are a national resource and ideally a system should exist where equitable distribution of organs based on human leucocyte antigen matching, can be co-ordinated by an independent national body. Unfortunately geographical and financial constraints have prevented this from becoming a reality.

Legislation regarding organ donation is suboptimal, especially in cases where the name of the donor is not known or when the relatives cannot be located. Furthermore, a critical shortage of corneas for transplantation exists since a moratorium was placed on the removal of corneas in mortuaries.

Fortunately, through the effects of the Organ Donor Foundation, the proposed card-format driver's licence will include an area where willingness to be an organ donor can be indicated. This may improve public awareness and partly alleviate the shortage of donor organs.

The role of transplantation in the private sector is rather controversial and it is feared that many private patients will eventually become a burden to the state when medical aid funds run dry. The possibility that a financial motive may influence patient selection and organ allocation cannot be ruled out. Transplantations in private hospitals could, however, alleviate the patient load at state institutions, and be a viable option if sensibly planned in co-operation with local academic units.

Representation to the Minister of Health and other health policy-makers must be made, in order to obtain a commitment to maintain state funding of transplantation.

The proposed implementation of 'framework autonomy' in academic hospitals is unlikely initially to provide adequate funds to sustain a transplant programme. National funding, perhaps on a supra-regional basis, such as is seen in the British health care system, is essential to ensure a future for transplantation in South Africa.

The alternative would not only be to the detriment of the thousands of patients waiting for transplants, but would also lead to a disastrous deterioration in standards of medical care, and a further loss of professional expertise.

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1. Mendez R, Aswad S, Dessonk A, *et al.* Cost and financing of kidney transplantation in the United States. *Transplant Proc* 1992; **24:** 2127-2128.
2. Naqvi A, Hussain M, Askari H, *et al.* Economics of renal rehabilitation in Pakistan: a case for increasing transplantation activity. *Transplant Proc* 1992; **24:** 2125-2126.
3. Stuart FP, Veith FJ, Cranford RE. Brain death laws and patterns of consent to remove organs for transplantation from cadavers in the United States and 28 other countries. *Transplantation* 1981; **31:** 238-244.
4. Pike RE, Odell JA, Kahn D. Public attitudes to organ donation in South Africa. *S Afr Med J* 1993; **83:** 91-94.

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to cancer in the intervention group becomes significant'.[9]

Understandably, many authorities have called for intensive scrutiny of present beliefs and practices, and the overall benefits that accrue therefrom. Thus Oliver[10] has written of his 'doubts about preventing CHD', and Ramsay *et al.*[11] on 'dietary reduction of serum cholesterol concentration'. Smith and Pekkanen[12] have raised the question, 'Should there be a moratorium on the use of cholesterol lowering drugs?' and Collins *et al.*[13] have suggested the 'need for larger trials'.

In a recent editorial, Hulley *et al.*[14] made a number of radical suggestions: (*i*) 'there is an association between low blood cholesterol and non-cardiovascular deaths in men and women . . . and a cholesterol-lowering diet may not be prudent for those adults whose cholesterol levels place them on the left-hand limb of the total mortality U'; (*ii*) 'there is no association between high blood cholesterol and cardiovascular deaths in women . . . With the exception of those who already have CHD or other reasons for being at a comparable very high risk of CHD death, it no longer seems wise to screen for and treat high blood cholesterol in women'; (*iii*) 'primary prevention trials of cholesterol intervention reveal an increase in non-CHD death rates that is similar in magnitude to the decrease in CHD death rates . . . For primary prevention in patients who do not yet have manifestations of coronary disease (or other reasons for being at a comparable very high risk of CHD death), it now seems unwise to treat high blood cholesterol with drugs'; and (*iv*) 'the new evidence on non-CHD causes of death makes it clear that this pediatric policy (the universal testing of children) is unwise, and indicates that we should draw back from universal screening and treating of blood cholesterol for primary prevention in adults as well'.

The effects of these views could be far-reaching, certainly with regard to the primary prevention of CHD. There will certainly be a measure of confusion in the mind of the public. Surely, in all preventive and therapeutic endeavours there should be regular and intensive examination of their effectiveness. It is necessary to be circumspect in the common practice of overblaming risk factors, e.g. cholesterol intake and cholesterol level, and overclaiming the benefits from control of risk factors, e.g. the excessive restriction of natural foods.[15]

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1. Shaper AG. Blood cholesterol: who to test. Part 1. *Br J Hosp Med* 1992; **47:** 639-641.
2. Naylor CD, Basinski A, Frank JW, Rachlis MM (the Toronto Working Group on Cholesterol Policy). *Asymptomatic Hypercholesterolemia: A Clinical Policy Review*. Toronto: Pergamon Press, 1990.
3. Naylor CD, Basinski A, Frank JW, Rachlis MM. Asymptomatic hypercholesterolaemia: a clinical policy review. *J Clin Epidemiol* 1990; **43:** 1029-1121.
4. Sanders FN. Congress report. Second National Congress of the Lipid and Atherosclerosis Society of Southern Africa. *CME/VMO* 1992; **10:** 1077-1084.
5. Betteridge JD. Blood cholesterol: who to test. Part 2. *Br J Hosp Med* 1992; **47:** 643-644.
6. Grover SA, Abrahamowicz M, Joseph L, Brewer C, Coupal L, Suissa S. The benefits of treating hyperlipidemia to prevent coronary heart disease: estimating changes in life expectancy and morbidity. *JAMA* 1992; **267:** 816-822.
7. Muldoon MF, Manuck SB, Matthews KA. Mortality experience in cholesterol-reduction trials. *N Engl J Med* 1991; **324:** 922-923.
8. Jacobs J, Blackburn H, Higgins M, *et al.* Report of the Conference on Low Blood Cholesterol: mortality associations. *Circulation* 1992; **86:** 1046-1060.
9. Skrabanek P, McCormick J. Prevention of coronary artery disease. *N Engl J Med* 1991; **325:** 203-205.
10. Oliver MF. Doubts about preventing coronary heart disease. *BMJ* 1992; **304:** 393-394.
11. Ramsay LE, Yeo WW, Jackson PR. Dietary reduction of serum cholesterol concentration: time to think again. *BMJ* 1991; **303:** 953-957.
12. Smith GD, Pekkanen J. Should there be a moratorium on the use of cholesterol lowering drugs? *BMJ* 1992; **304:** 431-434.
13. Collins R, Keech A, Peto R, *et al.* Cholesterol and total mortality: need for larger trials. *BMJ* 1992; **304:** 1680.
14. Hulley SB, Walsh JMB, Newman TB. Health policy on blood cholesterol: time to change directions. *Circulation* 1992; **86:** 1026-1029.
15. Walker ARP, Labadarios D, Benadé AJS, Vorster HH, Glatthaar II. Natural foods — are current restrictions excessive? (Editorial). *S Afr Med J* 1991; **80:** 311-312.

The neuroleptic malignant syndrome — still a conundrum

Neuroleptics, also known as antipsychotics and major tranquillisers, are commonly prescribed pharmacological agents. While in psychiatric practice they are used in the treatment of the psychoses, they are also employed in general medicine as antiemetics, antihistamines and sedatives. Apart from their therapeutic properties, unwanted side-effects may arise as a result of simultaneous α-adrenergic, histaminergic, cholinergic and dopaminergic receptor blockade of the peripheral and central nervous system. Such adverse effects include postural hypotension, reflex tachycardia, over-sedation, dry mouth, blurring of vision, constipation, urinary retention and extrapyramidal syndromes such as parkinsonism and dystonia. Occasionally a rare but potentially life-threatening condition occurs during the course of neuroleptic therapy; this has been termed the neuroleptic malignant syndrome (NMS). The disorder was described by Delay and Deniker;[1] they used the term in reporting a severe and idiosyncratic reaction to haloperidol. Since this initial description, a burgeoning literature, mainly in the form of case studies, has not only resulted in increased recognition of the syndrome but has provided the clinician with disparate views on pathophysiology, risk factors and management strategies. Many of these determinants are unresolved but recent prospective studies are providing more consistent data about the syndrome.

The clinical features universally recognised as pathognomonic of the disorder include generalised muscular rigidity, pyrexia (> 37,5°C), an altered state of consciousness and autonomic instability (labile blood pressure, tachycardia, tachypnoea, intermittent diaphoresis). The associated laboratory findings are nonspecific and are therefore considered supportive of the diagnosis which is made primarily on the clinical findings. They include a raised serum creatine kinase (CK) concentration, leucocytosis and a low serum iron concentration. It is evident that certain cases of suspected NMS do not fulfil textbook criteria and it is therefore probably reasonable to assume that the syndrome is a spectrum disorder with mild to severe manifestations and so-called 'formes frustes' or atypical variants.

The two major competing theories that attempt to explain NMS are central dopaminergic blockade versus a direct toxic effect of neuroleptics on skeletal muscle. Several writers have suggested that NMS is caused by a

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hypodopaminergic state in the mesostriatum that results in the extrapyramidal features; a similar state in the hypothalamus results in derangement of thermoregulation.[2,3] A recent study found that in those patients with leucocytosis and raised serum CK concentrations there was an associated low serum iron concentration during the acute phase of NMS; it was postulated that the acute phase reaction may play a role in the disorder.[4] Furthermore serum iron may play a role in the maintenance of striatal dopamine receptor function.[5]

Controversy exists concerning a possible pathophysiological link between NMS and malignant hyperthermia (MH). Both syndromes are characterised by fever, an altered state of consciousness, muscular rigidity and rhabdomyolysis. The possibility of an aetiological connection has been strengthened by reports of an abnormal contractile response of skeletal muscle *in vitro* to halothane exposure in patients who developed NMS.[6] On the other hand, others have denied MH susceptibility in NMS by noting that the tissue from NMS patients did not respond abnormally to halothane, caffeine or other agents.[7-9] The pathophysiological link appears tenuous, particularly because clinical evidence indicates that NMS patients tolerate anaesthetic agents without incident, and to my knowledge there has not been a single case of MH reported in such patients to date.[10,11]

Delay and Deniker[1] characterised the syndrome as 'malignant' because of its possible fatal outcome. In the early literature the mortality rate for NMS is cited as 7,7 - 20%.[12,13] Recent studies have reported reduced morbidity/mortality and attribute the improved outcome to earlier recognition and treatment of the syndrome.[14,15] It is interesting to note that fatalities are usually the result of recognisable medical complications that arise during the course of the disorder (e.g. pneumonia, renal failure, sepsis and pulmonary embolism), rather than the result of a specifically neuroleptic-induced mechanism.[16] The great majority of patients are treated with neuroleptics and other psychotropic medication without major sequelae. The determinants that predispose a small percentage of patients to NMS are unknown, but given the potential lethality of the illness there is an urgent need to identify possible predisposing or risk factors. It must be emphasised that patients presenting with a symptom cluster suggestive of NMS should be thoroughly examined and investigated to exclude other causes of a pyrexial and stuporous state such as CNS infection, heatstroke, anticholinergic toxicity or sepsis in a patient with neuroleptic-induced parkinsonism. Recent studies have linked NMS to several possible preconditions.

Psychomotor agitation (restlessness) has been noted as a frequent antecedent behavioural disturbance prior to the onset of NMS.[15-18]

A diagnosis of an affective disorder, such as mania and, less frequently, depression, is accompanied by a higher incidence of NMS than one of schizophrenia, which has been noted only rarely as an antecedent state.[15,18]

An increasing dose of a neuroleptic and the initial introduction of neuroleptics to vulnerable patients can be a precondition for NMS.[15]

Dehydration has been cited as a possible risk factor but it is possible that this finding may be a consequence rather than a cause of the disorder.[19]

Central nervous system disorders, other than psychiatric disorders, may increase the risk of NMS, including mental retardation, Alzheimer's disease, seizure disorders, HIV encephalopathy and neurosyphilis.[3,15,20]

A diagnosis of catatonia characterised by alternating episodes of mutism/immobility and acute psychomotor agitation may predispose a patient to NMS.[21]

In our experience at Groote Schuur Hospital we have observed the frequent occurrence of an antecedent catatonic syndrome in patients who developed NMS prior to the administration of a neuroleptic agent. It is possible that the psychomotor agitation commented on by other investigators may have been a state of catatonic excitement in the patients observed. It is suggested that certain patients present to hospital in a state of agitation (catatonic excitement), are given neuroleptics to control their behaviour and then go on to develop NMS.

Various treatments have been proposed but the mainstay of management is early recognition of the syndrome with immediate discontinuation of neuroleptics and supportive treatment. The use of pharmacological agents remains unresolved, with conflicting reports about the efficacy of dopamine agonists, such as bromocriptine, or skeletal muscle relaxants, such as dantrolene. It is interesting to note that a recent study reports that the use of these agents may, in fact, have prolonged the duration of illness in certain patients.[22] In certain cases, the use of benzodiazepines may provide transient relief of symptoms and ease discomfort.[15,23] The rationale for the use of various drugs is based on hypotheses of underlying aetiopathogenesis but, to date, it is evident that none of these pharmacological agents has any effect on the core pathophysiological mechanism of the disorder.

A fatal outcome has been associated with failure to recognise the syndrome and the continued use of neuroleptics.[23,24] Fortunately, with early diagnosis and treatment, the natural course of the illness is towards recovery. The average duration of the acute phase is approximately 9 - 14 days and we have found that a useful indicator of favourable progress is a declining CK level. Once the laboratory findings have returned to baseline levels, the patient is in the recovery phase. It is important to note, however, that muscular rigidity and autonomic dysregulation may persist for several weeks after the acute illness. There is concern about the reintroduction of neuroleptics in those patients who have experienced an episode of NMS, but there is evidence to suggest that most patients resume neuroleptics with no sign of recurrence. A period of over 2 weeks before challenge has been significantly associated with success.[25-27] It is suggested that neuroleptics should not be used until there is marked improvement in clinical status and the CK level and white cell count are within normal limits.

Given our observation that a catatonic state may predispose to NMS, we believe that the presence of catatonic signs (e.g. extreme agitation/excitement) should be a contraindication to the use of neuroleptics as sedation and that benzodiazepines are the agents of choice in this setting.

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1. Delay J, Deniker P. Drug-induced extrapyramidal syndrome. In: Winken PJ, Bruyn GW, eds. *Handbook of Clinical Neurology. Vol. 6. Diseases of the Basal Ganglia.* New York: Elsevier North Holland, 1968.
2. Burke RE, Fahn S, Mayeux R, Weinberg H, Louis K, Willner JC. Neuroleptic malignant syndrome caused by dopamine depleting drugs in a patient with Hungtington's chorea. *Neurology (NY)* 1981; **31:** 1022-1026.
3. Fricchione GL. Neuroleptic catatonia and its relationship to psychogenic catatonia. *Biol Psychiatry* 1985; **20:** 304-313.
4. Rosebush PI, Mazurek MF. Serum iron and neuroleptic malignant syndrome. *Lancet* 1991; **338:** 149-151.
5. Ben-Schachar D, Finberg JP, Youdim MB. Effect of iron chelators on dopamine D2 receptors. *J Neurochem* 1985; **45:** 999-1005.
6. Caroff SN, Rosenberg H, Fletcher JE, Helman-Patterson TD, Mann SC. Malignant hyperthermia susceptibility in neuroleptic malignant syndrome. *Anesthesiology* 1987; **67:** 20-25.
7. Tollefson G. A case of neuroleptic malignant syndrome: in vitro muscle comparison with malignant hyperthermia. *J Clin Psychopharmacol* 1982; **7:** 339-341.

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8. Scarlett JD, Zimmerman R, Berkovic SF. Neuroleptic malignant syndrome. *Aust NZ J Med* 1983; **13:**70-73.
9. Merry SM, Werry JS, Merry AF, Birchall N. The neuroleptic malignant syndrome in an adolescent. *J Am Acad Child Psychiatry* 1985; **25:** 284-286.
10. Levenson JL. Neuroleptic malignant syndrome. *Am J Psychiatry* 1985; **142:** 1137-1145.
11. Hermesh H, Aizenberg D, Weizman A. A successful electroconvulsive treatment of neuroleptic malignant syndrome. *Acta Psychiatr Scand* 1987; **75:** 237-239.
12. Caroff SN. The neuroleptic malignant syndrome. *J Clin Psychiatry* 1980; **41:** 79-83.
13. Addonizio G, Susman VL, Roth SD. Neuroleptic malignant syndrome: review and analysis of 115 cases. *Biol Psychiatry* 1987; **22:** 1004-1020.
14. Deng MZ, Chen GQ, Phillips MR. Neuroleptic malignant syndrome in 12 of 9792 Chinese in-patients exposed to neuroleptics: a prospective study. *Am J Psychiatry* 1990; **147:** 1149-1155.
15. Rosebush PI, Stewart T. A prospective analysis of 24 episodes of neuroleptic malignant syndrome. *Am J Psychiatry* 1989; **146:** 717-725.
16. Levinson DF, Simpson GM. Neuroleptic-induced extrapyramidal symptoms with fever. *Arch Gen Psychiatry* 1986; **43:** 839-848.
17. Itoh H, Ohtsuka M, Ogita K. Malignant neuroleptic syndrome: its present state in Japan and clinical problems. *Folia Psychiatrica et Neurologica Japonica* 1977; **31:** 565-576.
18. Keck PE, Pope HG, Cohen BM, McElroy SL, Nierenberg AA. Risk factors for neuroleptic malignant syndrome: a case control study. *Arch Gen Psychiatry* 1989; **46:** 914-918.
19. Harsh HH. Neuroleptic malignant syndrome: physiological and laboratory findings in a series of nine cases. *J Clin Psychiatry* 1987; **44:** 328-333.
20. Breitbart W, Marotta RF, Coll P. Aids and neuroleptic malignant syndrome. *Lancet* 1988; **2:** 1488-1489.
21. White DAC, Robins AH. Catatonia: harbinger to the neuroleptic malignant syndrome. *Br J Psychiatry* 1991; **158:** 419-421.
22. Rosebush PI, Stewart T, Mazurek MF. The treatment of neuroleptic malignant syndrome. *Br J Psychiatry* 1991; **159:** 709-712.
23. White DAC. Catatonia and the neuroleptic malignant syndrome: a single entity? *Br J Psychiatry* 1992; **161:** 558-560.
24. Kish SJ, Kleinert R, Minauf M, Gilbert J, Walter GF, Slimovitch C. Brain neurotransmitter changes in three patients who had a fatal hyperthermia syndrome. *Am J Psychiatry* 1990; **147:** 1358-1363.
25. Mann SC, Caroff SN, Bleier HR, Welz WK, Kling MA, Hayashida M. Lethal catatonia. *Am J Psychiatry* 1986; **143:** 1374-1381.
26. Levenson JL, Fisher JG. Long-term outcome after neuroleptic malignant syndrome. *J Clin Psychiatry* 1988; **49:** 154-156.
27. Pope HG, Aizley HG, Keck PE, McElroy SL. Neuroleptic malignant syndrome: long-term follow-up of 20 cases. *J Clin Psychiatry* 1991; **52:** 208-212.

Healthy cities for a future South Africa

With rapid urbanisation occurring worldwide, it is estimated that by the year 2000, half of the world's population will live in urban areas.[1] In response, a project aimed specifically at improving urban health, the Healthy Cities Project,[2] was initiated by the World Health Organisation. Here we review this project with particular regard for its relevance to South African cities.

The 'Beyond Health Care' Conference held in Toronto, Canada, in 1984 aimed at reviewing progress made in the public health movement during the previous 10 years; it was here that the concept of a 'healthy city' was first formulated.[3] This concept was developed further by the European office of the WHO in Copenhagen, which saw in it an opportunity for translating the 'Health for All' strategy into a tangible programme of action at local level within cities.

The Healthy Cities Project was officially launched 2 years later at an inaugural conference in Lisbon, Portugal, in March 1986; the underlying aim was to bring the public, the business and voluntary sectors and the communities together in a partnership to focus on urban health and health-related issues. The project has become one of the WHO's notable successes,[2] involving a large network of cities, and has expanded to eastern and central Europe and the developing world. Partnerships with international organisations and agencies have been formed, and feasible, practical strategies aimed at ensuring appropriate mobilisation of resources have been developed.

The ultimate goal of the project has been to improve the health of all people living and/or working in cities, through commitment to and visibility for health, together with institutional change and innovative action with regard to health and the environment.

In terms of the Healthy Cities approach, a city is viewed as a complex organism that is living, breathing, growing and constantly changing.[2] Healthy Cities Projects throughout the world have a number of characteristics in common. They affirm the holistic nature of health, recognising the interaction between its physical, mental, social, economic and ecological dimensions. They also require mechanisms for political commitment and decision-making for health, and are based on intersectoral collaboration and community participation. They recognise that housing, transport, planning, economic development, education, social services and other aspects of city government have a major effect on the environment and state of health of people in cities. Essential to the success of these projects has been broad political commitment from city parliaments (from all parties on a non-party platform).

Among the key principles of a healthy city are a clean, safe, physical environment of high quality, a stable and sustainable ecosystem, a strong and mutually supportive community, a high degree of participation and control by the public over decisions affecting their health and well-being, the meeting of the basic needs of all the city's people, an optimal level of appropriate environmental health and clinical care services that are accessible to all.[2]

A rethinking of WHO strategy in Europe has been achieved through the Healthy Cities Project. The demedicalisation of health, and the principles of the Ottawa Charter[4] on health promotion, which highlight the environmental and social aspects of health, are being emphasised more.

This conceptual framework has resulted in the reorientation of health services to meet the broader needs of the population. For example, local authorities have (*i*) changed their policies and approaches to focus on the planning and provision of services to vulnerable and underserved social groups; (*ii*) established health accountability mechanisms; and (*iii*) targeted health services to underprivileged groups, women and young children. Environmental projects have ranged from community action to clean up city streets to the development of city transport policies and comprehensive environmental monitoring systems. Several projects have implemented action to 'green' cities. Communities in general have become involved in shaping their environments by responding to developers' plans and by participating actively in the development of the places in which they live.[1]

It is expected that by the year 2000 a large proportion of the population of South Africa will live in cities.[5] In the light of increasing concern about the state of the environment, the housing crisis, the health impacts of rapid urbanisation and industrialisation, pro-active and holistic policies and planning mechanisms are needed to ensure more cost-efficient delivery systems. There is better understanding of the need in the country for a reorientation of policies towards equity, health promotion, disease prevention, environmental enhancement and, more importantly, a broader understanding of the concept of health.

A multi-faceted approach to environment and health enhancement, which integrates the various factors that

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support the creation of optimal health, needs to be adopted. The Healthy City concept, based on the recognition that health is multi-dimensional, has the potential to improve and maintain the quality of life for citizens living in urban areas in South Africa.

In the long term a successful Healthy Cities Project could mean a reduced need for expensive curative health services and medical care, fewer drug prescriptions for preventable conditions, lower work absenteeism and decreased loss of productivity, among other improvements in the health status of South African cities.

The establishment of such a project in this country would, like those in Europe, need to take into account city-specific environmental, social, cultural, economic and political circumstances. New administrative processes that strengthen communication and co-operation between departments of city government will be essential for the achievement of intersectoral action. Community groups and neighbourhood associations will need to become more actively involved in making the city a healthier place in which to live.

Cities themselves will have to be strengthened in order to allow for true community empowerment at local level with regard to issues that affect people's lives. In some cities, such as Johannesburg, there are many existing intersectoral programmes and committees of city government to ensure that a Healthy Cities Project can easily be developed and expanded upon, facilitating the synthesis and cementing of the building blocks that already exist.

Fundamental to a successful Healthy Cities Project in South Africa is political commitment, in order to ensure a high profile and visibility as well as support for the project. The formulation of intersectoral health promotion plans with a strong environmental heath component with real political power placed at the lowest possible level will decrease fragmentation. Project teams and technical committees need to be formed to link professionals from different disciplines and departments so that co-ordinated and integrated environment and health plans can be developed and implemented. Mechanisms for public participation must be strengthened to enhance health at city level, encouraging commitment from community-based organisations and placing health high on their political agendas.

Community empowerment could be facilitated by the targeting of specific groups and small local communities, e.g. young children, the youth, single parents, women, the elderly and the poor. Morbidity and mortality profiles of the community and linked environmental and social demographic databases are needed to promote the cost-effectiveness of community-based health promotion and disease prevention programmes. It would also be essential to develop appropriate indicators for monitoring and evaluating the project's activities in order to ensure that the city's resources are effectively utilised.[6]

Such a project would be advantageous for business and tourism, and could help place cities on the international map, with enhanced possibilities for links with other cities and communities throughout Africa and the world. A project of this scope would enable cities in South Africa to play a meaningful role in promoting the quality of urban life for all people in the region.

We look forward to a healthy debate on the Healthy City concept as South Africa moves towards a new vision with the hope of attaining health for all.

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1. World Health Organisation. *City Networks for Health*. Geneva: WHO, 1991.
2. World Health Organisation (Tsouross D, ed.) *World Health Organisation Healthy Cities Project: A Project Becomes a Movement*. Copenhagen: WHO Regional Office for Europe, 1991.
3. Ashton J, ed. *Healthy Cities*. Philadelphia: Open University Press, 1992.
4. Ottawa Charter for Health Promotion. Paper presented at the International Conference on Health Promotion. Ottawa, Canada, 17 - 21 Nov 1986.
5. Schoeman B. The southern African population problem. In: Mostert WP, Lötter JM, eds. *South Africa's Demographic Future*. Pretoria: HSRC, 1990.
6. World Health Organisation. *Twenty Steps for Developing a Healthy Cities Project*. Copenhagen: WHO Regional Office for Europe, 1992.

OPINION / OPINIE

Compensation for Occupational Injuries and Diseases Bill, 1993

Since 1941 the Workmen's Compensation Act (Act 30 of 1941) (WCA) has provided limited compensation to certain categories of employees who sustain occupational injuries or develop one of a limited schedule of occupational diseases. A variety of long-standing problems associated with medical, legal and administrative aspects of the WCA has led to the publication of proposed new legislation in the form of a Draft Bill[1] published in December 1992, entitled the 'Injured Employees Compensation Draft Bill, 1993', and subsequent public distribution, in May 1993, of a Bill with amendments, including a new title, the 'Compensation for Occupational Injuries and Diseases Bill, 1993' (COI&D Bill).

We take this opportunity to bring these developments to the attention of the medical profession, and to comment on their positive and negative implications, should this Bill become law. Of most significance to the medical profession is an extensive overhauling of existing provisions for compensation of occupational diseases. Under the previous system the approach to the compensation of occupational diseases was, in many instances, inconsistent and unclear. Few people were able to gain access to the compensation system. In addition, the system was very slow in its response to claims.

The new definition of an occupational disease in the Bill incorporates any disease, proved to the satisfaction of the Commissioner to be work-related, whether listed in an attached schedule or not. Only scheduled diseases were usually compensable under the WCA. The concept of scheduled diseases has been retained, but greatly expanded. A legal implication for an individual contracting a scheduled disease is that there is presumption as to the work-relatedness of the disease, thus reducing the burden of proof on the individual.

The expanded list of scheduled diseases and the broad definition of a work-related disease will bring South Africa into line with, and in some cases improve upon, prevailing international standards regarding compensation of occupational diseases. For example, lung cancer in asbestos workers and tuberculosis in health care workers were two of many regrettable omissions in the past that will now become scheduled diseases.

The failure of the WCA to cover non-scheduled occupational diseases has been the cause of serious hardship for those individuals who have suffered from