Van die Redaksie/Editorial

Psychiatry for a new South Africa

Psychiatry probably more than any other medical discipline is bound to a psychosocial interface and should have an awareness of the effects of psychosocial distress on mental and physical well-being as well as the dysfunction of families and groups.

It may well be that in the past psychiatry has not spoken loudly enough on issues that may be construed as 'political' or 'economic' and confined itself to the medical or psychological components of practice. Some reasons why psychiatrists may have been silent are that the legislation governing mental hospital staff and state psychiatrists has been more closely aligned to prisons than to hospitals.[1] State psychiatrists were placed in an invidious ethical position by the Internal Security Acts, especially when it came to the treatment of detainees. These Acts (still in force) successfully intimidate psychiatrists from speaking out in public and in the halls of political power.

The Executive Committee of the Society of Psychiatrists of South Africa has issued a 'position statement' in the hope that it will be accepted by psychiatrists and all professionals dealing with the mental health of people in this country, and thus provide guidelines for their practice. While the statement is perhaps tied into the present political dispensation, it can be modified to retain its main principles when damaging and unacceptable apartheid structures are obliterated not only by decree but in clinical practice.

Psychiatry is accepted as one of the five major medical disciplines by the South African Medical and Dental Council (SAMDC) but rarely is this really recognised or practised. It is hard to know who and what should be blamed for this, but the net result is that psychiatric services have suffered badly and appear to have been last in line for the allocation of whatever resources one can name. The patient who has the misfortune to suffer from a *mental* illness, especially if he is poor and disadvantaged, often cannot find adequate treatment.

Some major problems face psychiatry in South Africa and statistics tell the story. The World Health Organisation's figures indicate that seriously incapacitating mental disorders are likely to affect at least 1% of any population at any one time and at least 10% at some time in their lives. In developing countries the major psychoses (schizophrenia and mood disorders) constitute a large part of serious mental disorders and, in addition, mental disorders secondary to infectious illnesses and other organic diseases are relatively common.[2]

Epidemiological research[3,4] in Africa revealed that 4,9% of the total population showed definite psychiatric syndromes, with an additional 20,4% showing evidence of psychiatric distress. In some African studies,[5] 17% of children aged 5 - 15 years were suffering from some form of emotional problem. Extrapolated to the 33 million total population of South Africa these figures mean that psychiatry should be responsible for about 1,5 million people with definite psychiatric syndromes (4,9%). In Soweto alone there would be something of the order of 170 000 children in need. Given the violence, stay-aways and educational disruption of recent years, the figure of 17% is probably not an overestimate.

There are approximately 220 psychiatrists registered with the SAMDC and practising in the country. Less than 10% of these are black (African). Many psychiatrists have emigrated for political reasons. Most psychiatrists are in private practice in the large centres. As an urgent intervention training posts for psychiatrists need to be increased. Black doctors must be encouraged to specialise in psychiatry.

Psychiatric teaching in some medical schools still does not make the qualifying doctor competent to practise psychiatry in family or general hospital practice.[6] This means that in many areas where there are no psychiatrists, the mentally ill have to be managed by nurses unsupported by doctors.[7] Most rural psychiatric patients are managed by nurses with little or no effective medical cover. Psychiatry needs to be taught and practised as part of the spectrum of primary medical care. The College of Medicine of South Africa is introducing a Diploma in Psychiatry in an attempt to address this need.

Although official policy is that there is no racial discrimination in state hospitals, services for blacks in some mental hospitals are bad, and have been so poor for so long that the upgrading is going to require enormous sums of money and great effort. Many black psychiatric patients are housed in patently unacceptable and overcrowded accommodation. The move towards integration and equalisation has to be rapid, although it may be resisted. It is for psychiatrists in such areas to campaign against unethical perpetuation of unequal services based on colour and race.

Child psychiatry is now a registrable sub-specialty with the SAMDC. South Africa has a large population of children and adolescents, particularly in the black communities where more than 40% of the population is under the age of 15 years.

Child psychiatry services are rare in black communities here, as well as in the whole of Africa.[5] There is an urgent need to train more child psychiatrists and to give child psychiatry a more important place within the discipline.

Psychiatry has suffered from neglect through the years. The mentally ill tend to be a stigmatised, voiceless, unwanted group. Much of the stigma is based on folklore, fear and misinformation predating the discovery of psychotropic agents. Given even reasonable follow-up and care many mentally ill people can be helped to cope better, and a significant number can live normal lives. There will, however, always remain a demand for institutions for the chronically severely disabled mentally ill who would otherwise drift into a subhuman existence in subways, slums, prisons, old motor cars and holes in the ground.

The privatisation of institutions caring for the chronically mentally disabled in some 8 000 beds in this country remains a controversial issue and appears to be an abdication of government responsibility. These are, however, but a fraction of the real number who need care and this includes mentally retarded children and adults for whom community care is not a humane option.

The subdivision of health services into 'own affairs' and 'general affairs' along racial lines[8] has had a devastating effect on psychiatric services, particularly community services, in many areas. This was as a result of the reallocation of posts and the multiplication of parallel services.

It may be true that some services improved for a few, but it is more true that psychiatric services have deteriorated badly for the majority of black patients because of these divisions. Psychiatry has probably been more severely affected by the tricameral parliamentary system than any other medical discipline.

Desegregation and equalisation of services within a unitary health system are all good, but it is an illusion — and possibly a delusion — to think that decree and re-organisation alone will

solve the problem. The balances can only be redressed by a massive change in priorities and reallocation of resources in the direction of services to the mentally ill.

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1. Mental Health Act of 1973 (Act 18 of 1973). Sections 14(2) and 66A.
2. World Health Organisation. Organisation of mental health services in developing countries. *WHO Tech Rep Ser* 1975; No. 564; 8.
3. Orley JH, Wing JK. Psychiatric disorders in two African villages. *Arch Gen Psychiatry* 1979; **36:** 513-520.
4. German GA. Mental health in Africa: I. The extent of mental health problems in Africa today — an update of epidemiological knowledge. *Br J Psychiatry* 1987; **151:** 435-439.
5. Odejide AO, Oyewunmi LK, Ohaeri JU. Psychiatry in Africa: an overview. *Am J Psychiatry* 1989; **146:** 708-716.
6. Gangat AE. Call for psychiatric wards in hospitals (Editorial). *S Afr Med J* 1989; **75:** 411-412.
7. Freeman M. *Mental Health Care for a New South Africa* and *Rural Community Mental Health Care* (proceedings of two symposia). Johannesburg: Centre for the Study of Health Policy, Department of Community Health, University of the Witwatersrand, October 1990.
8. De Beer C. A brief history of divisions in health care. *The Leech* 1990; **59:** May, 7-8.

Primary pulmonary hypertension — new perspectives

Pulmonary vascular hypertension has many causes. These include cardiac disease (e.g. congenital, mitral valve disease, left ventricular disease), pulmonary disease (e.g. interstitial fibrosis or disorders causing airflow limitation), impaired control of breathing (e.g. Pickwickian syndrome) and pulmonary vascular diseases (thombo-embolism and primary pulmonary vascular diseases — arterial or venous — including intravenous drug abuse, pulmonary vasculitis and primary pulmonary hypertension). Underlying disorders of the heart, lungs or breathing control mechanisms can usually be diagnosed on the basis of clinical findings and standard ancilliary investigations (chest radiograph, ECG, echocardiogram and lung function tests). Once eliminated, the differential diagnosis includes two common conditions (primary pulmonary hypertension (PPH) and thrombo-embolic pulmonary hypertension (TPH)) and several other rare conditions (pulmonary veno-occlusive disease, pulmonary schistosomiasis and pulmonary vasculitides.

The development of modern treatment strategies has created new challenges for clinicians caring for patients with either PPH or large-vessel TPH. Appropriate selection and timing of therapeutic options is vital for optimal management, which is, *a priori*, dependent on accurate and early diagnosis.

Early studies[1] of patients with PPH suggested an extremely poor prognosis with inexorable deterioration to death within 2 - 3 years of diagnosis. This provided justification for the development of aggressive and sometimes hazardous management strategies. However, it has been recognised more recently that while prognosis in the main remains poor, certain patients stabilise or improve spontaneously.[2,3] This has emphasised the need to match the degree of therapeutic aggression exhibited with the severity of the condition in the individual patient.

Since hypertrophy of the media of the muscular pulmonary arteries precedes the development of other changes seen on histological examination, the triggering of vasospasm by an as yet unidentified mechanism has been suggested as the first step in the development of this condition.[4] This has prompted the use of pharmacological vasodilator agents to arrest and reverse the process before permanent changes develop. Encouraging early reports of the effectiveness of these agents spawned cautious optimism that a significant advance in the treatment of this condition had been made.[5-7] The disappointing response to vasodilator therapy noted in a recent study from Groote Schuur Hospital,[2] however, is in accord with the findings of many other workers.[8,9] It has been suggested that spontaneous variability in pulmonary artery tone accounts for the apparent improvement ascribed to these agents by some workers.[10] In addition, reports of severe adverse effects and occasional deaths have prompted doubts as to the wisdom of their use.[11-13] Nevertheless, there does appear to be a small group of patients who show a true therapeutic response.[14]

Rich and Brundage[15] have convincingly demonstrated that huge doses of calcium antagonists may be necessary to achieve effective pulmonary vasodilatation. Prostacyclin, a new, rapidly acting parenteral vasodilator, has proved safe and effective in identifying patients with a responsive pulmonary vascular bed suitable for long-term vasodilator treatment.[16,17] It is very potent and the dose must be titrated according to the effect achieved. The short half-life enables adverse haemodynamic effects to be reversed rapidly by curtailing the infusion. Haemodynamic monitoring is essential during initial testing of vasodilators to enable early detection and treatment of untoward effects.

Since vasodilator therapy, even when apparently effective, has not been shown to prolong life,[9] heart-lung transplantation is being increasingly favoured as a treatment option.[18] However, despite continuing improvement in technique and results, the procedure still has significant associated morbidity and mortality. The onus is thus on the attending physician to decide which patient has so severe a form of the condition as to justify this aggressive form of therapy. Moreover, correct timing is vital to enable the surgery to be performed while the patient is in a stable clinical state before the development of right ventricular failure, which significantly increases the risks of the procedure. Clear guidelines would be of enormous assistance to the clinician faced with this situation. Numerous workers have attempted to define accurate prognostic criteria to assist in the making of these difficult decisions. A recent review of clinical and haemodynamic features of 22 patients at Groote Schuur Hospital demonstrated an inverse relationship between survival and pulmonary vascular resistance (PVR), pulmonary artery pressure (PAP) and right atrial pressure (RAP).[3] Those who died soon after evaluation had significantly higher PAPs and PVRs. None of the 6 patients with a mean PAP of 75 mmHg or greater survived more than 12 months. These findings are not dissimilar to those reported previously. Various workers have severally suggested that poor prognosis is associated with elevated RAP,[19,20] PAP[20] and PVR[2] and reduced stroke volume index,[19] low cardiac output[2,20] and low pulmonary artery oxygen saturation.[21] However, the correlations in all these studies varied in significance and their application in assessing prognosis of the individual patient is limited. Early, accurate diagnosis, grading of severity and frequent re-evaluation are essential for optimal decision-making and management of what remains predominantly a most malign condition.

The use of specific treatment options for some patients with PPH and the encouraging results of thrombo-endarterectomy in selected cases of large-vessel TPH[22] has accentuated the need for accurate early differentiation of these conditions. For this pur-

pose, the diagnostic value of various features has been evaluated. A recent comparative study from Groote Schuur Hospital,[23] in accord with two previous studies,[24,25] showed that clinical features alone are insufficient to distinguish these conditions. Although the PPH group was younger, with higher PAPs and higher prevalence of Raynaud's phenomenon and ECG evidence of right axis deviation and right ventricular hypertrophy, the considerable overlap between the groups meant that these features were not diagnostic. Radionuclide lung scanning proved most effective: all TPH patients in this study had lobar or multiple segmental perfusion defects whereas normal scans or subsegmental defects only were found in the PPH group. Other authors concur that a normal perfusion scan in the setting of unexplained pulmonary vascular hypertension is strongly suggestive of PPH.[24,26] Since most clinicians, correctly, are reluctant to subject their patients to open lung biopsy because of the risk of the procedure in these patients, pulmonary angiography remains the most accurate means of distinguishing PPH from TPH.[24] It is, however, invasive, not without risk, and requires catheterisation facilities. Radionuclide lung scanning has proved to be a safe, effective alternative means of diagnosing these conditions and guiding physicians into appropriate investigation and management strategies.

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1. Sleeper JC, Orgain ES, McIntosh HD. Primary pulmonary hypertension. *Circulation* 1962; **26:** 1358-1369.
2. Rozkovec A, Montanes P, Oakley CM. Factors that influence the outcome of primary pulmonary hypertension. *Br Heart J* 1986; **55:** 449-458.
3. Chapman PJ, Bateman ED, Benatar SR. Prognostic and therapeutic considerations in clinical primary pulmonary hypertension. *Respir Med* 1990; **84:** 489-494.
4. Edwards WD, Edwards JE. Clinical primary pulmonary hypertension: three pathologic types. *Circulation* 1977; **56:** 884-888.
5. Wang SWS, Pohl JEF, Rowlands JS, Wade EG. Diazoxide in treatment of primary pulmonary hypertension. *Br Heart J* 1978; **40:** 572-574.
6. Lupi-Herrera E, Sandoval J, Seoane M, Biolostozky D. The role of hydralazine therapy for pulmonary arterial hypertension of unknown cause. *Circulation* 1982; **65:** 645-650.
7. Pearl RG, Rosenthal MH, Schroeder JS, Ashton JPA. Acute hemodynamic effects of nitroglycerin in pulmonary hypertension. *Ann Intern Med* 1983; **99:** 9-13.
8. Dantzker DR, D'Alonzo GE, Gianotti L *et al.* Vasodilators in primary pulmonary hypertension with variability of long-term response. *Chest* 1989; **95:** 1185-1189.
9. Robin ED. The kingdom of the near-dead: the shortened unnatural life history of primary pulmonary hypertension. *Chest* 1987: **92:** 330-334.
10. Rich S, D'Alonzo GE, Dantzker DR, Levy PS. Magnitude and implications of spontaneous hemodynamic variability in primary pulmonary hypertension. *Am J Cardiol* 1985; **55:** 159-163.
11. Dalal JJ, Griffiths BE, Henderson AH. Primary pulmonary hypertension: effects of nifedipine. *Br Heart J* 1981; **46:** 230-231.
12. Bush J, Wennevold A. Hazards of diazoxide in pulmonary hypertension. *Br Heart J* 1981; **46:** 401-403.
13. Elkayam V. Vasodilator therapy in primary pulmonary hypertension. *Chest* 1981; **79:** 253-254.
14. Reeves JT, Groves BM, Turkevich D. The case for treatment of selected patients with primary pulmonary hypertension. *Am Rev Respir Dis* 1986; **134:** 342-346.
15. Rich S, Brundage BH. High dose calcium channel-blocking therapy for primary pulmonary hypertension: evidence for long-term reduction in pulmonary arterial pressure and regression of right ventricular hypertrophy. *Circulation* 1987; **76:** 135-141.
16. Rubin LJ, Mendoza J, Hood M *et al.* Treatment of primary pulmonary hypertension with continuous intravenous prostacyclin (epoprosterol): results of a randomized trial. *Ann Intern Med* 1990; **112;** 485-491.
17. Jones K, Higenbottom T, Wallwork J. Pulmonary vasodilation with prostacyclin in primary and secondary pulmonary hypertension. *Chest* 1989; **96:** 784-789.
18. Smyth RL, Higenbottom TW, Scott JP, Wallwork J. Transplantation of the lungs. *Respir Med* 1989; **83:** 459-466.
19. Rich S, Levy PS. Characteristics of surviving and non-surviving patients with primary pulmonary hypertension. *Am J Med* 1984; **76:** 573-578.
20. Glanville AR, Burke CM, Theodore J, Robin ED. Primary pulmonary hypertension: length of survival in patients referred for heart-lung transplantation. *Chest* 1987; **91:** 675-681.
21. Fuster V, Steele PM, Edwards WD, Gersh BJ, McGoon MD, Frye RL. Primary pulmonary hypertension: natural history and importance of thrombosis. *Circulation* 1984; **70:** 580-587.
22. Moser KM, Daily PO, Petersen K *et al.* Thromboendarterectomy for chronic major vessel thromboembolic pulmonary hypertension. *Ann Intern Med* 1988; **107:** 560-565.
23. Chapman PJ, Bateman ED, Benatar SR. Primary pulmonary hypertension and thrombo-embolic pulmonary hypertension — similarities and differences. *Respir Med* 1990; **84:** 485-488.
24. D'Alonzo GE, Bower JS, Dantzker DR. Differentiation of patients with primary and thrombo-embolic pulmonary hypertension. *Chest* 1984; **85:** 457-461.
25. Gray HH, Morgan JM, Nerr IH, Miller GAH. Clinical correlates of angiographically diagnosed idiopathic pulmonary hypertension. *Thorax* 1990; **45:** 442-446.
26. Fishman AJ, Moser KM, Fedullo PF. Perfusion lung scans vs pulmonary angiography in evaluation of suspected primary pulmonary hypertension. *Chest* 1983; **84:** 679-683.

Prescribing for the elderly — a new perspective required

Alarming demographic trends are forecast for South Africa in the years immediately ahead. It is clear that more and more elderly people will be making ever-increasing demands upon our slender health care resources and it requires little imagination to visualise the likely scenario in health care of the elderly by the end of this century. Therefore anything that can be done to reduce the load of unnecessary and preventable disability will be welcome, and better prescribing for elderly patients certainly has that potential.

The multiple diseases so common among aged patients have a pervasive effect upon the therapeutics of this age group, for whom prescribers' lack of sufficient knowledge of the effects of drugs in the senescent years is compounded by insistent patient demand for 'a pill for every ill' and a plethora of easily available medicines.

The potential for adverse drug reactions and interactions is thus great and it is hardly surprising that one of the most fruitful sources of evidence for the cause of a sudden change or deterioration of such a patient's condition is the pharmacological history or the prescription record.

A survey conducted by Hurwitz[1] in teaching hospitals in Belfast in 1969 revealed an incidence of drug reactions of 10,2% in 1 160 patients. This actually amounted to 15,4% of patients aged 60 years and over, compared with only 6,3% in those under 60 years. Williamson and Chopin[2] demonstrated that about 1 in 10 of 2 000 patients consecutively admitted to acute geriatric units in Britain had been admitted because of drug side-effects, the incidence increasing in proportion to the number of drugs prescribed. The Royal College of Physicians report[3] on medication of the elderly suggested that better prescribing would go a long way towards reducing this avoidable load of iatrogenic illness.

In a unit analysis of 93 consecutive cases seen at four hospitals by the Geriatric Unit of the University of Cape Town in 1988,[4] 19 patients were found to have been much improved by the intervention. Among these, 7 owed their improvement to corrections of their drug therapy. There were 2 cases of drug-induced thyrotoxicosis, 2 cases of digoxin toxicity, 2 patients with hypokalaemia precipitated by a combination of diuretics and self-administered laxatives and 1 patient with severe diuretic-induced hyponatraemia.

For some time we have kept records of drug side-effects or adverse drug reactions (ADRs) and among 320 patients recorded

as having been seen by the geriatric unit we noted 32 patients with possible ADRs. However, in 6 of these, either the absence of follow-up or unconvincing or absent improvement on withdrawal made a definite diagnosis of ADR untenable. This left 26 rather more definite cases (8,1%) in which the ADR reported was entirely compatible with the known side-effect profile and abated on withdrawal of the drug or lowering of the dose. These figures themselves suggest under-reporting, which is a well-documented phenomenon,[5] and which is even more likely since some patients seen by us, such as ward consultations, might not have been recorded. It is also hardly surprising in view of the well-known difficulty of separating the effects of ageing and multiple pathology from those of drug side-effects in this age group and thus a number of preventable causes of morbidity probably went undetected.

Three categories of side-effects stood out as the main offenders: *(i)* diuretic problems; *(ii)* relative over-dosage or inappropriate use of L-thyroxine; and *(iii)* side-effects from non-steroidal anti-inflammatory drugs (NSAIDs).

Diuretic problems occurred in 8 patients; these included severe hyponatraemia with or without hypokalaemia from amiloride.

One patient being treated with L-thyroxine for documented hypothyroidism presented in atrial fibrillation and congestive heart failure owing to too high a dose, while in 4 others angina or less symptomatic atrial fibrillation were the symptoms unmasked, provoked or aggravated.

In 2 of the remaining 9 cases, severe cough was induced by captopril, while in the remainder, 1 each of the following side-effects was encountered: captopril-induced rash; enalapril-induced angio-oedema; obstructive airway symptoms from the β-blocking agent atenolol; peripheral vascular symptoms, also from atenolol; severe postural hypotension from cinnarizine; severe postural hypotension from a tricyclic antidepressant; and a case of thiazide-induced gout.

Several empirical lessons can be learned from these and other experiences. Amiloride needs to be used with the greatest circumspection in the elderly, hyponatraemia being a well-documented complication. It should not be forgotten that the thiazide component can override the potassium-sparing properties of the combination resulting in hypokalaemia,especially when the patient is also taking an over-the-counter laxative. Nor should the very dangerous possibility be overlooked of causing hyperkalaemia with this drug in renally compromised patients, although no such case was encountered in those recorded here.

The elderly often require a lower dose of L-thyroxine, which should be introduced and increased very cautiously. Abrahamson[6] advises an initial dose of 25 μg daily, building up very gradually by 25 μg monthly until replacement doses, established by careful clinical and biochemical monitoring, are achieved.

The tricyclic antidepressants are particularly likely to cause postural hypotension. One should therefore start the older adult on no more than half the usual initial adult dose and, again, increase it slowly, monitoring sitting and standing blood pressure readings carefully at each visit.

For chronic pain from the predominantly mechanical causes in osteo-arthritis, long-term use of NSAIDs is best avoided. Better to encourage weight loss, strengthening of muscles and local pain relief by physiotherapy, as well as judicious periods of non-weight-bearing rest each day.

Paracetamol in usual therapeutic doses is a much safer analgesic in such cases and NSAIDs should be reserved for the management, for a few days at a time, of traumatic and inflammatory exacerbations. Where their use cannot be avoided, as in many cases of rheumatoid arthritis, the minimum dose and careful monitoring are essential in the elderly.

Tranquillisers and sedatives (usually benzodiazepines) should never be given on a long-term basis other than for a few possible specific indications, for although none of the side-effects described here were due to one of these drugs, their use has been associated with increased unsteadiness, psychomotor impairment and a tendency to falls, as suggested by another study undertaken by this unit[7] and a number of other reports on the causes and prevention of falls in the elderly. Phenothiazines should be recognised for their well-marked propensity to cause postural hypotension in older adults and their indications should be examined very carefully.

In the elderly, well-known changes in the pharmacokinetics and pharmacodynamics of a number of drugs, altered baroreceptor sensitivity and the effects of age and disease, such as a decline in renal function, conspire to give therapeutics a distinct and unique slant. The need to research, develop and teach geriatric pharmacology as a subspecialty of the main discipline is therefore hardly an extravagant notion. Properly used, drugs are arguably the most cost-effective modality for the maintenance of symptom-free mobility and independence in the ill aged. They do, however, represent a double-edged sword, which may at times amount to a sword of Damocles.

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1. Hurwitz N. Predisposing factors in adverse reactions to drugs. *Br Med J* 1969; **1:** 536-539.
2. Williamson J, Chopin JM. Adverse reactions to prescribed drugs in the elderly: a multicentre investigation. *Age Ageing* 1980; **9:** 73-80.
3. Royal College of Physicians. Report on medication for the elderly. *J R Coll Physicians Lond* 1984; **18:** 7-17.
4. Meiring PdeV. Drug treatment in the elderly. In: Meiring PdeV, ed. *Textbook of Geriatric Medicine.* Cape Town: Juta, 1990: 114-128.
5. Bem JL, Mann RD, Rawlins MD. Review of yellow cards, 1986 and 1987. *Br Med J* 1988; **296:** 1319.
6. Abrahamson MJ. Common endocrine problems in the elderly. In: Meiring PdeV, ed. *Textbook of Geriatric Medicine,* Cape Town: Juta, 1990; 171-183.
7. Meiring PdeV. Falls in the elderly. *S Afr Med J* 1986; **69:** 214-215.