

Van die Redaksie/Editorial

The wider implications of stroke care

As in many other places, care of stroke victims in South Africa leaves much to be desired. Viewed from the perspective of a large metropolitan hospital the position can best be described as parlous. It is little comfort to be reminded that the world-wide mortality from strokes is declining when large numbers of patients still present for care and the quality of life of the survivors becomes the prime consideration.

Whitelaw¹ described the ideal situation as admission of all cases to a stroke unit where time is not critical and rehabilitation can proceed at a pace commensurate with the individual's potential for improvement. He wrote from the perspective of a small pilot rehabilitation unit of a few beds at Eaton Hospital in the Cape Peninsula. The small size of such a unit itself introduces a bias for with very few beds available cases for admission have to be chosen with consummate care for their potential for recovery of independence. While excellent results have been attained the question has not been settled as to whether such patients would not have done equally well had an integrated rehabilitation programme been based elsewhere. However, Whitelaw points out that stroke patients spend very little time even in an acute hospital setting, let alone in any kind of rehabilitation programme, and in these circumstances a team approach to planned discharge becomes doubly important. The reasons become apparent when a common scenario is examined.

In our large metropolitan hospitals the pressure on beds is unbearable. In a medical ward of about 40 beds it is nothing unusual for the registrars on duty to have to admit well in excess of 20 patients in a 24-hour period. Every third or fourth week their firm will take its turn for a week-end duty, the so-called double intake for that week. This is a source of recurring anxiety for the resident staff and usually ends with patients boarded out all over the hospital. When pressure of this sort is being constantly exerted, all but the sickest patients must be discharged each week to make room for the new admissions, and it may be difficult for anyone who has not passed through this particular mill to appreciate the pressure and anxiety to which the resident medical staff are constantly subjected.

In these appalling circumstances it is small wonder that stroke patients sometimes have to be discharged with a nasogastric tube or catheter still *in situ*, after a rapid and rudimentary attempt to instruct a relative how to 'manage', an appointment to return for physiotherapy and a referral letter to the community nursing staff from the nearest day hospital. While the value of the intensive stroke unit is in some doubt and there may even be a

good case for treating some patients at home, all are agreed upon the benefits of a comprehensive team programme for stroke management.³

In this issue of the *SAMJ*, Dewar examines the outcome of acute strokes in 210 elderly hospitalised⁴ patients for whom such a structured rehabilitation programme did not exist. A retrospective review of their hospital case notes was combined with a prospective household follow-up after a minimum period of 19 months. This constitutes one of very few such local studies in which an attempt has been made to establish the facts upon which rational recommendations can be made. She found a very high case-fatality ratio within the first month, by which time only half the patients were still alive. Only 35 patients remained for household follow-up, although the calculated 37% 1-year mortality compares favourably with that in at least one other study.⁵ Of these survivors, only 13 were fully independent in their activities of daily living.

A high degree of social isolation was found, but the presence of depression and urinary incontinence as contributory causes of an indifferent outcome show that there were at least two potentially treatable conditions that could have improved their quality of life, had they received prior attention. It is apparent that mere examination and comparison of figures from various series does little to address this particular aspect among the survivors of strokes. In any case, Brocklehurst *et al.*⁶ have pointed out that as a considerable proportion of stroke patients may not be admitted to hospital, a hospital-based study does not reflect the full extent of the burden on society.

It is clear that no single measure could suffice to improve the quality of life and functional capacity of stroke survivors. There is a need for motivation on the part of professional carers, objective functional assessment and supportive community-based health services, which will enable people to remain in their own homes and communities rather than having to resort to institutionalisation. However, here we come up against the patchy distribution of such community-based support systems and it has been our experience in the geriatric ward at Conradie Hospital in Cape Town that it would have been possible to discharge many patients into the community had such support existed in their area. A similar problem was found when the reasons for the inability to discharge elderly black patients from hospital in Grahamstown were examined.⁷

Cerebrovascular disease is a very common cause of death and disability and this is particularly so in the

elderly. Therefore the provision of proper stroke care in this age group is practically synonymous with the elements of good geriatric care, in which many of the same goals prevail. This will only be achieved by co-ordinated planning and determined implementation. It has been shown that rather than by means of the expenditure of vast capital sums, good geriatric care can stem from reallocation, realignment and co-ordination of people and facilities that are often already in place.^{8,9}

With reform in the air, and therefore the imminent prospects of a huge diversion of resources to redress the inequalities in our society on a broad social front, it is extremely unlikely that we will see the provision of a great financial bonanza for something as unproductive on the purely material plane as geriatric medicine or stroke care.

It therefore becomes doubly important to co-ordinate those resources we already have in a way which avoids duplication and unnecessary waste. It is hardly likely that this can happen while we are supporting the unproductive bureaucracy that goes with a number of separate health authorities. What is needed above all is a

centralised health authority that can assess and allocate our shrunken resources on a rational basis of real need rather than according to the impractical dictates of yesterday's ideology.

P. de V. Meiring

1. Whitelaw DA. Neurological disorders in the elderly — selected topics. In: Meiring PdeV, ed. *A Textbook of Geriatric Medicine*. Cape Town: Juta (in press).
2. Von Arbin M, Britton M, de Faure U *et al.* A study of stroke patients treated in non-intensive stroke units or in general medical wards. *Acta Med Scand* 1980; 208: 81-85.
3. Stevens RS, Ambler NR, Warren MD. A randomised controlled trial of a stroke rehabilitation ward. *Age Ageing* 1984; 13: 65-75.
4. Dewar SR. Stroke outcome in the absence of a structured rehabilitation programme. *S Afr Med J* 1990; 78: 200-202 (this issue).
5. Stevens RS, Ambler NR. The incidence and survival of stroke patients in a defined community. *Age Ageing* 1982; 11: 266-274.
6. Brocklehurst JC, Andrews K, Morris P, Richards BR, Laycock PL. Why admit stroke patients to hospital? *Age Ageing* 1978; 7: 100-106.
7. Meiring PdeV, White G de L, Jameson C, Wylde BR. Geriatric medicine in Grahamstown. *S Afr Med J* 1987; 71: 166-168.
8. Meiring PdeV, Mitchell PJ, Bracken GL, Wingreen B, Kurten M. A geriatric service in East London. *S Afr Med J* 1986; 70: 288-292.
9. Meiring PdeV, Benatar SR. The establishment of geriatric medicine at the University of Cape Town. *S Afr Med J* 1986; 69: 565-569.

Insulin-dependent diabetes mellitus — recent developments

Ever since Bottazzo *et al.*'s¹ classic article describing islet-cell antibodies in sera of patients with insulin-dependent diabetes mellitus (IDDM), there has been a steadily increasing body of evidence to suggest that auto-immunity is the basic pathogenetic mechanism underlying this disease.²

Attention has now been focused on the possibility of immunosuppression in order to reverse or arrest ongoing immunological destruction of the insulin-producing β -cells of the pancreas. Thus prednisone, cyclosporin and azathioprine have been used either alone or in combination in different studies³⁻⁸ in an attempt to prevent ongoing β -cell destruction. The success rate, however, has been far from satisfactory. One particular drawback was that most of the studies were not double-blind. Moreover, while some patients were controlled without insulin up to a year after the onset of symptoms, none showed any significant improvement in endogenous β -cell function as assessed by C-peptide secretion.

Therefore it would seem that if immunosuppression were to be started before the clinical onset of IDDM (pre-IDDM stage), sufficient β -cell function could be preserved so as to prevent clinical manifestation of the disease. Based on the classic description of IDDM, which highlighted an acute onset as being a distinctive feature of the disease,⁹ recognition of a prediabetic phase would certainly be a problem. Recent studies, however, have challenged the concept that IDDM is a disease of acute onset, since like non-insulin-dependent diabetes mellitus (NIDDM) IDDM has also been shown to have a long preclinical phase during which there is

presumably a steadily progressive destruction of the β -cells of the pancreas until there are at most no more than 10% of such cells still functioning when the disease becomes clinically manifest.¹⁰⁻¹²

How then is this preclinical phase ('pre-IDDM') recognised? The presence of islet-cell antibodies without any symptoms or any abnormality in oral glucose tolerance is the hallmark of this stage.¹⁰⁻¹² Of particular interest is that such subjects, in addition to showing a normal glucose tolerance, have a normal insulin response to oral glucose and intravenous glucagon, both of which are pancreatic β -cell secretagogues.² However, many of them have a blunted first-phase insulin response to intravenous glucose.^{2,13} Moreover, these subjects often show abnormalities of immune function commonly seen in newly diagnosed IDDM patients, viz: insulin auto-antibodies, increase in the ratio of helper T cells to suppressor T cells (OK4/OK8) and increase in the number of immune-activated (Ia-positive) T cells.^{2,14} These observations suggest that the process of auto-immune destruction of the β -cells of the pancreas starts long before IDDM becomes symptomatic. Hence it would be rational to intervene actively using immunosuppressive agents during the pre-IDDM phase in order to prevent the clinical IDDM stage. However, the toxicity of drugs available at present is well known and their use in asymptomatic individuals would certainly raise ethical considerations.¹⁵

Therefore the current recommendation is that until the natural history of the prediabetic phase of IDDM

(pre-IDDM) is more clearly understood and delineated, immunosuppression should not be used in such subjects. Moreover, even patients with newly diagnosed IDDM should not be offered such therapy outside a research setting.

M. A. K. Omar

A. A. Motala

1. Bottazzo GF, Florin-Christensen A, Doniach D. Islet cell antibodies in diabetes mellitus with autoimmune polyendocrine deficiencies. *Lancet* 1974; **2**: 1279-1282.
2. Eisenbarth GS. Type I diabetes mellitus: chronic autoimmune disease. *N Engl J Med* 1986; **314**: 1360-1368.
3. Elliot RB, Crossley JR, Berryman CC, James AG. Partial preservation of pancreatic beta-cell function in children with diabetes. *Lancet* 1981; **2**: 1-4.
4. Stiller CR, Dupré J, Gent M *et al*. Effects of cyclosporine immunosuppression in insulin-dependent diabetes mellitus of recent onset. *Science* 1984; **223**: 1362-1367.
5. Feutren G, Assan R, Karsenty G *et al*. Cyclosporin increases the rate and length of remissions in insulin-dependent diabetes of recent onset: results of a multicentre double-blind trial. *Lancet* 1986; **2**: 119-124.
6. Harrison LC, Colman PG, Dean B, Baxter R, Martin FIR. Increase in remission rate in newly diagnosed type I diabetic subjects treated with azathioprine. *Diabetes* 1985; **34**: 1306-1308.
7. Silverstein J, Maclaren N, Riley W, Spillar R, Radjenovic D, Johnson S. Immunosuppression with azathioprine and prednisone in recent onset insulin dependent diabetes mellitus. *N Engl J Med* 1989; **319**: 599-604.
8. Bougneres PF, Cavel JC, Castano L *et al*. Factors associated with early remission of type I diabetes in children treated with cyclosporin. *N Engl J Med* 1989; **378**: 663-670.
9. Oakley WG, Pyke DA, Taylor KW. *Diabetes and Its Management*. Oxford: Blackwell Scientific Publications, 1973.
10. Irvine WV, Gray RS, McCallum CV. Pancreatic islet-cell antibody as a marker for asymptomatic and latent diabetes and prediabetes. *Lancet* 1976; **2**: 1097-1102.
11. Gorsch AN, Spencer KM, Lister V *et al*. Evidence for a long prediabetic period in type I (insulin dependent) diabetes mellitus. *Lancet* 1981; **2**: 1363-1365.
12. Srikanta A, Ganda OP, Jackson RA *et al*. Pre-type I (insulin dependent) diabetes; common endocrinological course despite immunological and immunogenetic heterogeneity. *Diabetologia* 1984; **27**: 146-148.
13. Srikanta S, Ganda OP, Gleason RE, Jackson RA, Soeldner JS, Eisenbarth GS. Pre-type I diabetes: linear loss of beta cell response to intravenous glucose. *Diabetes* 1984; **33**: 717-720.
14. Rabinowe SL, George K, Jackson R. Lymphocyte abnormalities in individuals prior to overt diabetes mellitus or at risk for type I diabetes mellitus. *Diabetes* 1985; **34**: suppl, 167A.
15. Herold KC, Rubenstein AH. Immunosuppression for insulin dependent diabetes. *N Engl J Med* 1989; **318**: 701-703.