

## EDITORIAL : VAN DIE REDAKSIE

## DIABETES IN THE ELDERLY

We will probably live to be at least 65 years old. As far back as 1955 the USA (male) expectation of life at birth was 67.9 years (female 73.5). At around this age there is a 50% chance of becoming diabetic (by present definitions). If one-tenth of the population of the USA are over 60 years and half of these are diabetic, then there are some 10 million diabetics over 60 years in that country. Even if one considers only known diabetics under treatment, a conservative estimate of 5% in the elderly<sup>1,2</sup> gives us over a million in the USA.

The undoubted increase in the amount of old-age diabetes has been illustrated by Joslin,<sup>3</sup> who quoted figures for the percentages of death from diabetes by decades. The over 80s accounted for 5% of deaths before 1932 and for 14.5% from 1950 to 1955. An increasing number of elderly diabetics can be explained by the increased longevity of the population in general; the increasing duration of life of diabetics after diagnosis; and the greater awareness of diabetes as a common disease. In addition there may be a true increase in the prevalence of diabetes itself. This is certainly true for countries and races in the process of 'urbanization' such as the South African Bantu<sup>4</sup> and most probably applies to highly industrialized countries as well.<sup>5</sup> Finally, there are large numbers of asymptomatic elderly people who are being diagnosed as diabetic by virtue of postprandial hyperglycaemia or glycosuria.

Regarding the duration of life of diabetics, the Joslin clinic data show a rise from 4.9 years before 1914 to 18.2 years in 1956-57, when the average age at death rose to 65 years.<sup>3</sup> It is noteworthy how close this is to the average total expectation of life as mentioned above. Nevertheless, even elderly diabetics have a death rate (calculated per 100,000/year) between 2 and 3 times that of non-diabetics. They die mainly from vascular disease, peripheral, cardiac and renal in origin. Furthermore, the elderly diabetic may yet go into coma, can go blind from retinopathy, is likely to be impotent, may lose one or both legs, develops resistance to oral drugs, has urinary problems, suffers from myocardial ischaemia, aggravates some of these conditions by overweight, and becomes a tremendous social problem for various reasons. He or she may also be more liable to hypertension, cerebral vascular disease, osteoporosis, carcinoma of the body of the uterus, severe Dupuytren's contracture, cataract, gout and innumerable other disorders.

Diabetes mellitus may be simply defined as a 'chronic state of too high a blood sugar level'.<sup>4</sup> Until recently this has been the most practicable definition, though it begs the question of what is meant by 'too high'. Now, and especially in relation to the elderly, we have to consider whether we should be thinking of diabetes as present when the blood sugar exceeds some arbitrary level after an artificial stress (usually oral glucose) or only when unmistakable symptoms and glycosuria during the course of an ordinary day are manifested.

Although the scare about the apparently colossal amount of diabetes in older people is recent, the basic facts have really been known much longer. Reports dating

from 1921 have indicated that tolerance to orally administered glucose diminishes with age, so that, judged by usually accepted standards, even mean glucose-tolerance curves become 'diabetic' in old age.

Spence, in 1921, performed glucose-tolerance tests (GTT) on five men over 60 years (4 were over 70 years) and found mean figures very much above his standard young adult curve.<sup>6</sup> Only one could be classed as normal. Porter and Langley<sup>7</sup> in 1926 performed GTT on 50 normal subjects comprising 10 in each decade and found an increasing loss of tolerance to glucose with each decade, up to 70 years. The tolerance curve of the 70-80-year group was rather lower than the 60-70-year age-group. Hale-White and Payne,<sup>8</sup> in the same year, examined 10 healthy subjects over 70 years and found that in 5 of them the blood sugar peaked at above 210 mg./100 ml. and returned more slowly to the fasting level. They suggested that a peak of 220 mg. should be considered normal at the age of 70 years. In 1931 Marshall<sup>9</sup> came to a similar conclusion after testing 28 people over 65 years—the blood sugar in 4 of his subjects actually rose above 240 mg./100 ml. Numerous more recent reports have confirmed the loss of glucose tolerance with age.<sup>1,2,4,10-14</sup>

There is some evidence suggesting that this diminution of mean glucose tolerance with age does not extend (and may even improve) beyond 75 years,<sup>7,10</sup> while clinical and survey estimations have indicated a decline in the incidence of diabetes at about this age.<sup>1,3,10-13</sup> This suggests either that old age is less diabetogenic than 'elderly age' or that most of the potential diabetics have already been diagnosed or have died off. In any event it must be an important argument against the concept that ageing alone leads to diabetes. Certainly subjects over 90 years may have a completely normal oral GTT.

The rising blood glucose with age after a glucose challenge is not matched by a similar rise in the fasting blood-sugar levels; there is a mean rise in fasting blood sugar with age, though of far lesser degree.<sup>4,11,12</sup>

It has also long been known that considerable hyperglycaemia can be present without glycosuria. Reported by Spence<sup>6</sup> in 1921, the concept as applied to elderly people was taken further by Marshall,<sup>9</sup> who concluded that the renal threshold is generally raised in healthy old age. Hence an absence of glycosuria does not exclude gross impairment of glucose tolerance.

More recently discovered, and still more recently appreciated, the blood sugar frequently rises far higher after a glucose load than after a normal, heavy meal, especially in older people. This simple fact has several important corollaries: Any postprandial screening level must be much lower than a postglucose level, and cannot be as sensitive unless carefully quantitated; glycosuria after glucose is much more frequent than after a meal and is consequently a better screening procedure; and it emphasizes the unnatural nature of a sudden glucose load.

Crombie, in the Birmingham survey,<sup>10</sup> rediscovered these truisms when he found that 18% of individuals over 50 years of age who had been aglycosuric during a screening



procedure had a 'diabetic' abnormality of glucose tolerance. In other words, he found 18% to be diabetic on glucose-tolerance test, as against only 1.65% in the same age-group during the main screening survey.

One survey that was based on a blood-sugar level for screening was that of Osserman and Starin in New York.<sup>20</sup> They established a final diagnosis of discovered diabetes in 8.3% of their tested subjects, of whom less than one-third had glycosuria.

It will be noted that the criteria for normality of glucose tolerance are derived from healthy, young subjects. Hence we must recognize either that diabetes is far commoner in older people than we had thought, or that we must change our criteria of abnormality for older people; ideally we should need to define normal standards for each decade. It is still uncertain which of these two alternatives is correct, whether the finding of an asymptomatic 'abnormality' in glucose tolerance in older people carries prognostic significance. Is it a 'disease'? It is certainly not sharply demarcated from normal—taking 2-hour postglucose levels, or in fact any other values, frequency curves obtained from large numbers of people have never shown bimodal distribution. As might be expected there is a tail to the right in all such curves away from a strictly Gaussian distribution, but no double peak. This, however, does not prove anything—it is still possible that such curves are really composed of two overlapping truly Gaussian curves belonging to a normal and a diabetic population. It is also possible that the blood-sugar values under consideration reflect multiple genic influence, so obscuring the distribution between normal and abnormal. It is further possible that the righthand tail represents simply the upper reaches of normality.

Can any abnormality be found in elderly hyperglycaemics apart from those concerning carbohydrate metabolism? So far the evidence is extremely scrappy but does suggest that they have more vascular disease than the normoglycaemics. Butterfield, from the Bedford survey,<sup>11</sup> reported 43.3% of hyperglycaemics to have vascular disease as against 27.9% of the normoglycaemics. The data provided by Ostrander *et al.*<sup>2</sup> are not strictly germane to this issue, since they examined the prevalence of cardiovascular disease in *known* diabetics, which has been shown in innumerable other studies to be greater than normal.<sup>21,22</sup> They also confirmed the frequent presence of asymptomatic hyperglycaemia in both ischaemic heart disease and

peripheral vascular disease. This converse must suggest that the vascular disorder in many individuals is really a manifestation of diabetes in which the carbohydrate abnormality is minimal.

A working hypothesis maintains that hyperglycaemia is important and is likely to indicate diabetes, at least in a proportion of asymptomatic individuals. It is not certain, even so, whether such people should be treated and, if so, how. Butterfield and co-workers<sup>11</sup> have suggested that only those with 2-hour postglucose blood-sugar levels over 200 mg./100 ml. can be called definitely diabetic and are in need of treatment. Follow-up studies on elderly hyperglycaemics are not yet conclusive.

A single abnormal glucose-tolerance test in an asymptomatic elderly person is not sufficient even for the label of hyperglycaemia, much less diabetes, because repeat tests may be normal. We would tentatively suggest that an asymptomatic, healthy but repeatedly hyperglycaemic elderly person should be carefully watched for the future development of overt diabetes or its complications and should be actively encouraged to lose weight if necessary. Apart from this we cannot see any particular indication for change of diet and the use of oral drugs or of insulin.

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