Diabetes in the Elderly

V. S. Ganesan, V. Balaji, V. Seshaiah

INTRODUCTION

Daibetes mellitus and its complications are important health care problems in the elderly. The average life span in India has increased over the years, thanks to general, social, economical and medicare improvements. While in the developed Western world, 11% of the population is 65 years or older, increasing to 20% by the turn of the century[1], in India, by the report of the 1981 census, 3.8 % of the population is above the age of 65 years. As the prevalence of diabetes increases with age, even a low estimate of diabetes in this high-risk population projects about two million elderly diabetics to be caredfor in India[2]. This coupled with the diabetics diagnosed in their macro and micro vascular complications. add on to the magnitude of the problem. Unfortunately till recently this important issue of diabetes in the elderly was not given the due recognition it deserves.

Spectrum of Glucose Tolerance in the Elderly Diagnosis

It is well established that glucose tolerance declines as age advances[3]. In fact the decline begins in the third decade to progress further through the life span [4]. It is interesting to note that while the fasting plasma glucose changes minimally (a rise of 1mg% per decade after the age of 50 years), the 2 hour plasma glucose after glucose load rises by 10 mg% per decade after the age of 50 years. Thus, ' a 40 year old woman might have a fasting plasma glucose level of 90 mg/dl and two hour post prandial plasma of 130 mg/dl initially but by the age of 80. she may have a fasting plasma glucose level of 98 mg/dl (which is of little clinical significance) but also a two hour post prandial plasma glucose level of 190 to 210 mg/dl- a significant increase' [5]. This age-dependent increase in mean blood glucose concentration might represent the upper level of a positively skewed normal distribution or may be due to overlap of a diabetic sub-population, thus provoking the question : Is this age related hyperglycaemia benign, explainable by the normal ageing process or is it of pathologic potential, requiring medical intervention? The Bedford Survey[6] and other studies have established the increase in macrovascular disease in old patients with hyperglycaemia. Realizing the need to accommodate this age related deterioration in

carbohydrate intolerance and its propensity to increase macrovascular disease in the elderly, many age adjustment corrections have been suggested to interpret the GTT in the aged and a nomogram is also available [4]. But by upwardly revising the diagnostic criteria of diabetes mellitus, both NDDG[7] and WHO[8] have obviated the need for any age adjustment in interpreting the GTT in the elderly. Moreover teh increased risk of macrovascular disease due to mild glucose intolerance has been rightfully recognized by creating a seperate class of impaired glucose tolerance (IGT).

The presently accepted criteria for diagnosing mellitus and impaired glucose tolerance is given in

Table 1.Diagnostic Criteria for Diabetes Mellitus

Vend	ous plasma g Fasting	lucose (mg/dl) 2hrs post 75 g oral glucose
Impaired glucose tolerance Diabetes Mellitus	<140 >140	140-200 >200

Based on WHO[8]

It is worth stressing here that while applying these figures, the usefulness of labelling patients diabetic or not must weighed on an individual basis. Mere blood sugar level is not a criterion to define the severity of diabetes in the elderly. It depends on the consequence of diabetes.

Prevalence and the Burden of Diabetes in the Elderly

"Knowledge of the prevalence of a disease is required to estimate its community impact, rank it against competing priorities and calculate the resources to manage it"[9]. In the Geriatric Clinic of Government General Hospital, Madras, oral glucose tolerance picked up 8% of the elderly as diabetics and twice that number (16%) had IGT. Urine sugar screening detected only 5% as diabetic and random blood glucose screening (> 180 mg/dl of venous blood glucose) picked up 11% as diabetic. In another survey done at the Diabetes Clinic of the Government General Hospital, Madras, 12% of all

From Madras Medical College (VSG); and Diabetes Unit, Apollo Hospitals (VB) (VS), Madras. INT. J. DIAB. DEV. COUNTRIES (1994), VOL. 14 diabetics were aged 60 years and over. Pima Indians of the Arizona [10] and the elderly in eastern Finland[11] have the highest prevalence of NIDDM reported: about 40% of the men aged 65 to 79 are diabetic in these populations. In contrast, about 6% of the elderly Danes[12] and Californians [13] are diabetics. Diabetes is more common in the Afro-Americans, with 17.2% of them at 65 t o75 years, being known diabetics, 12.1% previously undiagnosed and 5.4% with impaired glucose tolerance [14].

Pathogenesis

Hyperglycaemia in the elderly may be attributed to one of the three categories:

- 1. Diabetes detected in the middle age live through to become elderly diabetics.
- 2. Diabetics detected for the first time in the geriatric age group.
- 3. Impaired glucose tolerance developing de novo in the elderly.

Most elderly subjects with diabetes mellitus have NIDDM. The pathogenesis of NIDDM in this group is similar to that in other age groups. Thus, diminished insulin secretion in relation to the patient's need and insulin resistance are major factors in the pathogenesis. There are other age related factors like shift to sedentary life style, increased adiposity, coexistent medical illnesses and concomitant use of multiple ' glucose intolerant' contribute drugs that may also to the hyperglycaemia[15].

Insulin Secretion

Studies conducted in older human subjects and in ageing Sprague-Dawley rats (as a model of ageing) have shown a significant defect in glucosestimulated insulin secretion, despite morphological demonstration of an increase in islet beta cell size and an increase in pancreatic islet insulin content[16]. There is a blunted first phase of insulin secretion and a significant decrease in second phase insulin release. This altered insulin dynamics demonstrated in the animal model closely resembles the defects established in human NIDDM patients. Many workers do not entirely agree with this view of a deficit in the total amount of insulin released by the pancreas. For instance, De Fronzo[17] has established that within the physiologic range of plasma glucose concentrations, the pancreatic response to hyperglycaemia is normal. However, with supraphysiological elevations in plasma glucose concentration, the ability of the pancreas to maintain a normal insulin response becomes

impaired in older subjects, revealing the reduced sensitivity of the ageing beta cell to glucose stimuli. There is also a decrease in biologically active insulin with a concomitant increase in the biologically less active proinsulin component [18]. Thus, at present, the age related decline in glucose tolerance is only partially explained on the basis of reduced insulin output by a less sensitive, aged beta cell.

Insulin Resistance

The combined results of the euglycaemic and hyperglycaemic clamp studies provide a strong evidence that the primary cause of deterioration in glucose metabolism with advancing age, is teh result of tissue unresponsiveness to insulin [17]. Several studies have confirmed that though the number and the avidity of the insulin receptors are not reduced, there is demonstrable insulin resistance even in the euglycaemic elderly[19,20]. As to the site of insulin resistance, it has been shown that hepatic responsiveness to insulin remains unaltered through ageing. As adipose tissue disposes of less than 5% of an oral or intravenous glucose load, quantatively most of the insulin resistance resides in the muscle tissue. From limited studies reported in a smaller number of subjects, it is difficult to localize the receptor and post receptor events that may explain the insulin resistance in elderly. The changes in body composition, reduced physical activity, caloric and carbohydrate intake may also secondarily induce changes aggravating the insulin resistance [21].

'Hyperglycaemia in the elderly may be part of spectrum of age related changes in carbohydrates tolerance, which ranges from mild insulin resistance to frank diabetes mellitus. Type 2 diabetes may be more common in the elderly because of age related changes in insulin secretion and action, glucose handling, diet and in physical activity. All these factors in concert may lead to the development of the disease in genetically susceptible persons'

Clinical Features

Presentation of diabetes mellitus in the elderly may be atypical[22]. Non-specific symptoms like fatigue, pruritus vulvae, incontinence or weight loss may be the pointers of hyperglycaemia. In our series, however, 84.6% of the patients presented with the classic triad of ployuria, polydipsia and polyphagia. Periarthritis of the shoulder, dizziness and balanitis were other common symptoms. Preoperative screening yeilds sizeable percentage of patients. While some may present with the serious complication of hypersmolar non-ketotic coma[23], others may be detected with coexisting cataract, nephropathy, peripheral vascular disease or neuropathic ulceration. Because of these multivaried presentation, high index of suspicion is necessary while treating geriatric patients to maximize the diagnostic yield of diabetes.

Types of Diabetes

Majority of the elderly diabetics have NIDDM or Type 2. Ninety three percent of our patients belonged to this category. Two percent of the patients required insulin for correction of hyperglycaemia but only 0.4% were insulindependent diabetics, thus stressing the rare possibility of Type 1 in this age group.

Management

Treatment Goals: There is a widespread, misplaced notion that good glycaemic control may not be necessary in the aged with diabetes, and symptomatic relief may suffice, as they may not live long enough to suffer from chronic complications. Apart from the excellent quality of life it endows, good metabolic control may reduce the incidence of hyperosmolar coma. With increasing longetivity, more elderly diabetics are becoming prone to complications. chronic With DCCT [24] demonstrating the value of good glycaemic control, there is a place for active management of diabetes in the elderly. Thus at one end a simple symptomatic relief may greatly help those with poor physical and cognitive capabilities, but others with preserved organ faculties, but others with preserved organ faculties must not be denied the advantages of good glycaemic control. But the above caveat must be weighed with the iatrogenic problem such good control invariably exposes these patients to, namely, hyperglycaemia. Exercise, diet, oral agents and insulin form the cornerstones of therapy in the diabetics as in other age groups.

The emphasis on diet therapy is on total caloric restriction. Detailed diet sheets or a school room approach on diet therapy are not justified in the elderly. Total caloric restriction can be effectively emphasized by saying 'eat half of what you use to' in obese diabetics. It is difficult to change the habits of lifetime and hence for good compliance, individual food faddisms must be respected and altered little. Due concessions must be given for coexistent oral problems due to loss of teeth and taste and reduced salivation. Relatives caring for the patient may need to be educated and encouraged. Guidance for exercise must be highly individualized. Brisk walking at three kilometers per hour for 45 minutes everyday not only improves glycaemic control but also induces the much needed self confidence and sense of well being. But coexistent degenrative arthitis and coronary artery disease may interfere with this programme and a suitable indoor alternative to keep 'limbs busy' must be worked out. If feasible, exercise tolerance may be determined by suitable tests and the patient advised on this basis.

Oral Hypoglycaemic Agents

These need to be considered only after a fair trial of diet had been given. The elderly may be on many drugs for concomitant illnesses and the interactions of these with OHA must be considered. Logie et al[25] found that about two thirds of the old patients were prescribed one to nine additional medicines and half of these patients were on drugs with documented interaction with antidiabetic drugs.

The Threat of Hyperglycaemia

The major concern in the elderly diabetic, while on OHA or insulin, is life threatening hypoglycaemia. Many studies have confirmed the markedly reduced awareness of the forewarning symptoms of hypoglycaemia in this age group. Impaired autonomic nervous system functions which delay the early warning signals of the impending hypoglycaemia, puts the aged patient at a lifethreatening risk. Hypoglycaemia may be of insidious onset with predominant neuroglycopenic symptoms. Hemiplegic hypoglycaemia responding rapidly to intravenous glucose has been reported [26]. Poor nutrition and cognition, use of multiple interacting drugs and alcohol and coexistent renal or hepatic problems may further predispose these patients to severe hypoglycaemia. Thus the dosage of any hypoglycaemic drug is gradually increased till satisfactory control attained. Tolbutamide is relatively safe owing to its short duration of action but needs multiple daily doses. Long acting first generation sulphonylureas like chlorpropamide and acetonhexamidev are totally avoided as they may accumulate in the presence of reducing renal functions. Second generation sulphonylureas exhibit fewer drug interactions due to their non-ionic binding but their hypoglycaemic potential is high. Tolbutamide is documented to be relatively safer in the elderly so also is glipizide.

Biguanides especially metformin are a safe alternative for overweight diabetics. Renal impairment and cardiac failure are common in the old and caution is needed before prescribing biguanides as they predispose to lactic acidosis.

Use of insulin

Persistent symptoms, ketonuria or continual weight loss while on maximal safe dose of oral agents are indications to introduce insulin. The rare insulindependent elderly diabetic may require complex split and mixed regime of insulin but the majority of insulin requiring diabetics need to be given a single daily dose of an intermediate acting insulin or long acting ultralente insulin in the morning. If fasting hyperglycaemia persists on this regime, some may require small quantities of intermediate acting insulin in the evening. To avoid or postpone insulin because of mental and physical handicaps is understandable, but many elderly would be thankful for the marked relief of nocturia and the good quality of life that insulin bestows on them. The compliance with insulin regimens is usually good among the elderly.

Monitoring Control

Urine glucose testing is unreliable because of raised renal threshold for glucose in the elderly and by the same token, glycosuria usually reflects hyperglycaemia. Self-monitoring with home blood glucose testing, in the presence of failing vision and arthritic hands may not be possible and is usually not necessary. Fasting blood glucose once a month and a glycated Hb once in 2-3 months may be sufficient for the majority.

Complications

Acute: Hyperglycaemia Hyperosmolar Ketotic Coma : [HNKC]

This syndrome of marked elevation in blood glucose with resultant hyperosmolar state, is usually confined to middle aged and elderly NIDDs. More common in patients with renal impairment, HHNKC is precipipated by a combination of factors such as loss of glucose and co-administration of drugs like thiazides, beta blockers and steroids. It might also be the first manifestation of diabetes [23]. Since unusual presentations such as focl deficits [27], dysphagia [28] and urinary retention are not uncommon, HHNKC must enter into the differential diagnosis of any coma in the elderly. As pointed out by Tattersall [9], one reason for delayed diagnosis and referral might be the false assumption by the primary care physician that diet and OHA, failing to consider severe hyperglycaemia as a cause of deterioration. Low dose insulin therapy and sufficient hydration with half-normal saline are the

essentials of management. The mortality is nearly 50% if it is not energetically managed. Arterial thrombosis may complicate the clinical course necessiating the use of hepari. Patients who survive HHNKC may be managed with diet alone or diet in combination with OHA. Some of the risk factors for HHNKC identified include female gender, dementia, intercurrent illness and previously undiagnosed diabetes mellitus[29].

Diabetic ketoacidosis[DKA]

This is associated with a higher mortality in the elderly: forty three percent of the ketoacidotic patients over the age of 50 years had died compared to 34% in those under the age of 30 years [30]. As in other age groups, most cases are precipitated by infection an other forms of stress like myocardial infarction. About a third of all cases of DKA occur in patients aged 60 years and above and hence awareness and prompt management are essential. The treatment protocol is similar to that adopted in young DKA patients.

Chronic Complications

Retinopathy: Diabetic retinopathy in general and maculopathy in particular are the typical problems of the elderly diabetic. About 40% of those diabetics diagnosed after the age of 70 years were demonstrated to have retinopathy [31], stressing the fact that the prevalence of retinopathy (as in diabetes) rises with age. Another study puts the prevalence of background retinopathy at 25% and proliferative retinopathy at 1.6% in elderly diabetics and has shown that poor metabolic control and duration of diabetes were important risk factors [32]. Maculopathy accounts for majority of the cases with visual loss and fortunately photocoagulation is very effective in preservation of sight in this condition. Maculopathy is identified by the presence of hard exudates, microaneurysms or haemorrhages within ine disc diameter of the fovea with a visual activity less than 6/9 on the Snellen chart or a ring of hard exudates with a centrally visible vascular anomally lying within ine disc diameter of the fovea[22]. In view of the beneficial effects of the photocoagulation, it is obligatory for the physician to detect retinopathy early with annual fundic examination through dilated pureation. Other major causes of reduced vision in the aged like cataracts, glaucoma and senile macular degeneration must not be forgotten. The Framingham Eye Study had shown that cataracts were found in 46%, macular degeneration in 28% and glucoma in 7% of their diabetic population [33].

Neuropathy

Absent ankle jerks and reduced vibration sense are common in the non-diabetic elderly making it difficult to study the prevalence of neuropathy in the aged diabetics. But the condition of diabetic proximal motor neuropathy (diabetic amyotrophy) is common in the elderly. There is asymmetric weakness of the pelvic gridle and thigh muscles, with little sensory changes. It may be associated with excruciating pain over the limbs and marked loss of weight. Active physiotherapy and good glycaemic control ameliorates the problem in about 6 to 12 months. Autonomic neuropathy is equally common and may present as postural hypotension, hypoglycaemic unawareness, gastric and urological abnormalities and impotence.

Diabetic Foot Syndrome

' The elderly diabetic is often divorced from his feet unable to see them because of poor eye sight, unable to feel them because of sensory loss and unable to bend down to touch them because of arthritis' [34]. About 20% of diabetic admissions are for diabetic foot problems [35]. Education of patients aimed at preventive foot care could dramatically reduce the amputation rate [36]. Visually handicapped elderly diabetics with peripheral and autonomic neuropathy and peripheral atherosclerotic vascular disease are highly susceptible to diabetic foot syndrome. Recurrent foot ulcers are common. Combined management by a physician, vascular surgeon and an orthopaedician yields the highest salvage of these infected deformed feet.

Macrovascular Disease

Mortality due to cardiovascular complications is increased in elderly diabetics and those with impaired glucose toleranc [37]. Combination of ageing with diabetes exposes these subjects to increased risk of atherosclerosis. Moreover, many aged may present with macrovascular pathologies as shown by the Finnish Study : previous myocardial infarction was twice as common in diabetis women as compared to the normal population [38]. Older individuals are prone tp hypertension and dyslipidaemia and thus teh stage is set for the cardiac, cerebral and peripheral vascular diseases. Obesity, sedentry life style and smoking may also contribute to the morbidity. The Framingham study has demonstrated that cerebrovascular diseases (thrombotic stroke and TIA) are 2.5-4 times more common diabetics [39]. Major studies testing the effects of treatment of these important variables on the course of macroangiopathies in the elderly is

urgently needed, as this high risk population has so far been excluded from most of the major intervention trials.

SUMMARY

Hyperglycaemia in the elderly is an important health care problem.

Even milder degrees of glucose intolerance enhances the morbidity risk in older individuals in whom the ageing process wreaks its own toll. Diabetes clinics specialized in managing the aged are ideal but with a little effort, most of the primary care physicians can deliver the optimal care. Increased awareness is essential to diagnose the complications early and management is advocated. ' Do not go too low on blood glucose is a therapeutic guide. Senior citizens are rightfully concerned about their health and timely delivered educational advice in preventive aspects is desirable. Diabetes in the elderly is another area where a diabetologist, in cooperation with other medical and surgical specialists, could prevent or retard, what was till recently believed to be ' silent deviation from the norm', from deteriorating into a 'fulminating clinical catastrophe' [40].

REFERENCES

- 1. Rowe JW. Health care of the Elderly. NEJM 1985;312:827-35.
- 2. Rao KVR, Seshaiah V. Epidemilogic features of non-insulin-dependent diabetes mellitus in the elderly in a developing country (India). Bulletin: Delivery of Health Care for Diabetics in Developing Countries 1987;8:22-4.
- 3. Bennett PH. Diabetes in the elderly-diagnosis and epidemology. Geriatrics 1984; 39:37-41.
- 4. Andres R. Ageing and Diabetes. Med Clin North Am 1971.
- Lipson LG. Diabetes in the Elderly. Diagnosis, pathogenesis and therapy. The American Journal of Medicine 1986; Supple.5A: 10-21
- Jarret RJ, McCartney P, Keen H. The Bedford Survey: Ten year mortality rates in newly diagnosed diabetics, borderline diabetes and normoglycaemic controls and risk indices for coronary artery disease in borderline diabetics. Diabetologia 1983; 22: 79-84.
- 7. National Diabetes Data Group. Classification and diagnosis of diabetes mellitus and other categories of glucose intolerance. Diabetes 1979; 28: 1039-57.
- World Health Organization. Technical report Series No. 729. WHO Expert Committee on Diabetes Mellitus. 1985.

- 9. David Keer, Taffersall R. Diabetes and old age. The Diabetes Annual 1988; 4:222-33.
- Knowler WC, Bennet PH, Hamman RF, Miller M. Diabetes incidence and prevalence in Pima Indians. A 19-fld greater incidence than in Rochester, Minnesota. Amer J Epidemi 1978; 108:497.
- Tuomilehto J, Nirsinen A, Kivela SL, et al. Prevalence of diabetes mellitus in elderly men aged 65-84 years in Eastern and Western Finland. Diabetologia 1986; 29:611.
- 12. Damsgaard EM, Faber OK, Forlan A, et al. Prevalence of fasting hyperglycaemia and known non-insulin-dependent diabetes mellitus classified by plasma C-peptide. Fredericia Survey of subjects 60-70 years old. Diabetes Care 1987; 10: 26-32.
- Barrett-Corner E. The prevalence of diabetes in an adult community. Am J Epidemiology 1980; 111: 705.
- Harris MI, Hadden WC, Knowler WC, Bennett Ph. Prevalence of diabetes and impaired glucose tolerance and plasma glucose levels in the US population aged 20-74 years. diabetes 1987; 36: 523-34.
- Halter JB. Geriatric Patients . In 'Therapy for diabetes mellitus and related disorders'. American Diabetes Association 1991; 155-60.
- 16. Reaven GM, Gold G, Reaven EP. Effect of age on glucose simulated insulin release by the beta cell of the rat. J Clin invest 1979;64 :591-8.
- 17. De Fronzo RA. Glucose intolerance and ageing. Diabetes 1981;30:77-82.
- Gold G, Reaven GM, Reaven EP. Effect of age on proinsulin and insulin secretory pattern in isolated rat islets. Diabetes 1981;30: 77-82
- Rowe JW, Minaker KL, Pallota JA, et al. Characterization of insulin resistance of ageing. J Clin Inves 1981;71:1581-7.
- 20. Fink RI, Kolterman OG, Griffin J. et al. Mechanism of insulin resistance of ageing. J Clin Inves 1981;71:1581-7.
- McGandy RB, Barrows CH Jr, Spanias A, et al. Nutrient intakes and energy expenditure in men of different ages. J Gerontol 1966;21:581-7.
- Fernando DJS, Boulton AJM. Diabetes management in old age. In: Textbook of Geriatric Medicine and Gerontology. Brocklehurst, Tallis RC, Fillit HM(eds). 4th Ed. 1992; 729-38.
- 23. Podolski S. Hyperosmolar non-ketotic coma in the elderly diabetic. Med Clin North Am 62: 815-27.
- 24. The Diabetes Control and Complications Trial Research Group. The effect of intensive treatment of diabetes on the development and progression of long term complications in insulin-dependent diabetes mellitus. N Engl J Med 1993; 319:977-86.

- 25. Logie AW. Gallavay DB, Petrie JC. Drug interactions and long term antidiabetic therapy. Br J Clin Pharmacol 1976;3:1027-32.
- 26. Gale EA. Hypoglycaemia. Clin Endocrinol Metab 1980; 9: 461-75.
- 27. Grant C. Warlon C. Focal epilepsy in diabetis nonketotic hyperglycaemia. Br Med J 1985;293:1204-5.
- Burke BJ. Hyperglycaemic hypersmolar non-ketotic diabetic coma presentong as severe dysphagia. Br Med J 1980; 1: 1421-2.
- 29. Wachtel TJ. Selliman RA, Hamberton P. Predisposing factors for the diabetic hyperosmolar state. Arch Intern Med 1987;147:499.
- 30. Gale EAM Dorman TH, tattersall RB. Severely uncontrolled diabetes in the over fifties. Diabetologia 1981:21:25-8.
- 31. Dwyer MS, Melton JC, Ballard DJ, et al. Incidence of diabetic retinopathy and blindness. A population based study in Rochester, Minnesota. Diabetes Care 1985;8:316-22.
- 32. Nathan DM, Singer DE, gocine JE, et al. Retinopathy in older Type 2 diabetics. Association with glucose control. Diabetes 1986;35:797-801.
- 33. Kini MM,Heibowitz HM, Colten T, et al. Prevalence of senile cataract, diabetic retinopathy, senile macular degeneration and open-angle glaucoma in the Framingham Eye Study. Am Journ opthamol 1987;8:28.
- 34. Bloom A. Some pratical aspects of management of diabetes. Clin Endocrinol Metab 1977;6:499-517.
- Boulton AJM, Barker JH. The diabetic foot. In : Diabetes mellitus, management and complications. Olefsky JM, Sherwin RS (eds). Churchill Livingstone, New York 1985;255-75.
- Brand PW. The Diabetic Foot. In : Diabetes Mellitus, Theory and Practice. Ellenberg M, Rifkind H (eds).
 3rd Ed. Medical Examination Publishers New York 1983; 829-49.
- Kannel WB. Lipid, diabetes in coronary heart disease. Insights from the Framingham study. Amer Heart Journ 1985; 110: 1100-7.
- Unsitupa M, Siltonen O, Ard A, Pyorala K. Prevalence of coronary heart disease, left ventricular failure and hypertension in middle aged, newly diagnosed Type 2 diabetic subjects. Diabetologia 1985;28:22.
- Kannel WB, Mcgee DL. Diabetes and cardiovascular disease. The Framingham study. Amer Journ Med 1979;241:2035.
- 40. Petri MP, Gatling W, Petri L, Hill Rd. Diabetes in the elderly. An epidemological perspective. Practical Diabetes 1986;3:153