these were probably outweighed by the more frequent admissions to the emergency room by ambulance in the placebo group. Thus excluding costs of non-severe side effects and transportation probably did not influence the overall result.

The most obvious economic gain with long term metoprolol was in the reduction of indirect costs. Direct costs (costs of readmission), however, were also lower in the metoprolol group compared with the placebo group. The difference between the two groups became most evident later in follow up.

As any evaluation of costs and benefits of a treatment requires many different assumptions, we performed a sensitivity analysis using various discount rates. At discount rates ranging between 2·5% and 8·0% metoprolol was associated with reduced overall costs. We did not set out to achieve exact costs for the two different treatment regimens, as the analysis needed to be based on several assumptions, and costs differ in different health care systems. The important finding was that prophylactic treatment with the β blocking agent metoprolol given for three years after myocardial infarction did not increase costs but reduced utilisation of the health care system.

We do not know what the effects of metoprolol might be if it were to be continued beyond three years after myocardial infarction. That patients with infarction treated with β blockade have a longer survival may result in higher utilisation of the health care system in a later phase. These possible future costs, however, will probably include an increased need for health care owing to aging of the patients.

In conclusion, three years of metoprolol treatment given after myocardial infarction improved the prognosis and reduced utilisation of the health care system. Postinfarction treatment with metoprolol therefore appears to be cost effective.

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Type I (insulin dependent) diabetes: a disease of slow clinical onset?

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Abstract

Type I (insulin dependent) diabetes is usually believed to present acutely and it is assumed that metabolic decompensation is sudden. In a prospective family study, however, 10 of 13 subjects developing the disease showed progressive or intermittent development of hyperglycaemia over many months and the others had non-specific symptoms over a long period. All were first degree relatives of a child with type I diabetes; 10 were siblings (aged 5-24) and three were parents (aged 45-58). All possessed HLA-DR4 or DR3, or both, and all but two had been positive for islet cell antibodies for six to 86 months before diagnosis. Ten had non-specific symptoms for two to 14 months before the onset of thirst and polyuria; one remained asymptomatic even when insulin became necessary. Six subjects had an oral glucose tolerance test before clinical onset, of whom five were diabetic by World Health Organisation criteria four, four, six, seven, and 21 months before insulin was needed. Nine showed random blood glucose concentrations above the 97·5th centile (6·3 mmol/l) six to 34 months (median 12) before diagnosis. Two others had a glucose tolerance test result compatible with diabetes but had not reached the stage of needing insulin.

Hyperglycaemia is often of insidious onset in type I diabetes, even in children and young adults. Diagnosis will inevitably be late if considered only when acute symptoms of thirst and polyuria develop.

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Introduction

Traditionally type I (insulin dependent) diabetes is thought to present acutely, especially in childhood. The most recent edition of a standard paediatric textbook states that "the onset of diabetes in childhood is always acute with thirst and polyuria as the presenting symptoms."[1] In practice the presentation of childhood diabetes may be more varied. A survey of 66 children showed that 19 (29%) had symptoms for less than two weeks, 18 (27%) had symptoms for two to four weeks, and 29 (44%) had symptoms for more than four weeks.[2]

More recent studies have shown that the onset of type I diabetes is preceded by a prodromal period, often extending over years,

characterised by the presence of circulating islet cell antibodies[3] and abnormalities of cell mediated immunity.[4] Another feature of this period is progressive loss of the first phase insulin response to intravenous glucose.[5] These observations suggest slow attrition of the numbers or function of β cells culminating in obvious metabolic decompensation only when some 80-90% of cells have been destroyed. If this is correct we could predict that many patients will drift into diabetes and that acute onset will be exceptional and related to acute intercurrent illness or other forms of stress.

We have had the opportunity to study the onset of glucose intolerance and symptomatic diabetes prospectively in first degree relatives of children with type I diabetes. Over the past seven years we have observed the development of diabetes in 15 patients (two not having reached the stage of needing insulin). All had symptoms or biochemical abnormalities long before clinical presentation.

Patients and methods

The Barts-Windsor family study was established in 1978 and recruited 207 families with a diabetic proband diagnosed under the age of 20. Families have been visited every four to six months and blood samples obtained from all members for blood glucose, islet cell antibody, and other measurements.[3]

Blood glucose concentrations reported here were measured from whole blood using a glucose oxidase-peroxidase method (Technicon AAII or Yellow Springs analyser). A normal range for random blood glucose values was obtained using all the samples measured in the non-diabetic family members in the study (roughly 800 samples); the 97·5th centile for these was 6·3 mmol/l. In one patient glucose values were measured in stored serum samples; the correlation of serum glucose with whole blood samples was: serum glucose (mmol/l) = 1·01 × whole blood glucose − 0·34 (r = 0·99; n = 30).

Glycosylated haemoglobin was measured by cation exchange chromatography (Bio-Rad). The non-diabetic range in our laboratory is 3·4-6·1%. Tests of glucose tolerance including oral glucose tolerance tests (1·75 g/kg body weight to a maximum of 75 g) and 25 g intravenous glucose tolerance tests were performed in some subjects.

Results

During the study 10 siblings and three parents developed diabetes; all were established with insulin treatment. In only two was the onset of symptoms sudden, though both had shown abnormal glucose tolerance four and six months previously. The average duration of specific symptoms such as thirst and polyuria in the remainder was short (four weeks), while the duration of non-specific symptoms such as tiredness, weight loss, and irritability was longer (median 26 weeks). One remained asymptomatic even when insulin was required to control sustained hyperglycaemia. In addition, two others had glucose tolerance test results compatible with diabetes but did not require insulin. The table gives the details of all 15 subjects.

CASE HISTORIES

Case 5—An 11 year old boy first complained of tiredness in November 1984. Urine samples showed 2% glycosuria, and a random blood glucose estimation was 15·4 mmol/l. He began following a 200 g carbohydrate diet and subsequent blood glucose concentrations ranged between 6·0 and 7·3 mmol/l. His glycosylated haemoglobin value was 4·5% in June 1985, and an oral glucose tolerance test gave the following results: 0 min 4·4 mmol/l, 30 min 8·3 mmol/l, 60 min 11·4 mmol/l, 120 min 15·0 mmol/l, and 180 min 7·6 mmol/l. Results were similar six months later, and both sets of values were diabetic by World Health Organisation criteria but (because of the normal fasting blood glucose concentration) not by National Diabetes Data Group criteria.[6,7] He remained asymptomatic, with random laboratory blood glucose measurements of 4·2-7·3 mmol/l until January 1986, when he developed thirst and polyuria over a weekend. Home blood glucose measurements showed readings of >22 mmol/l but urine was negative for ketones. He began taking insulin 14 months after the blood glucose concentration was first noted to be abnormal.

Case 12—Both the brother and mother (case 8) of this 15 year old girl had insulin-dependent diabetes. Apart from failure to gain weight (she weighed 35 kg (3rd centile for age) in September 1983 and 38 kg (well below the 3rd centile) in January 1986) she remained asymptomatic. In September 1985 she underwent a minor operation and the wound was slow to heal. Two months later she complained of a sore throat and tiredness; infectious mononucleosis was considered but a Paul-Bunnell test gave a negative result. In January 1986 these symptoms recurred but she did not complain of thirst or polyuria. Blood glucose concentration was 32·4 mmol/l and the urine contained a moderate amount of ketones. Retrospective analysis of glucose values in stored serum samples gave the following results: September 1982 (five samples), 3·3-4·4 mmol/l; April 1983, 29·4 mmol/l; September 1983, 31·1 mmol/l; May 1984, 25·7 mmol/l; and January 1985, 35·0 mmol/l. All of these samples were taken postprandially. Thus severe hyperglycaemia (recognised retrospectively) had been present for nearly three years before specific symptoms occurred.

Case 3—This 24 year old man shared at least one HLA haplotype with his

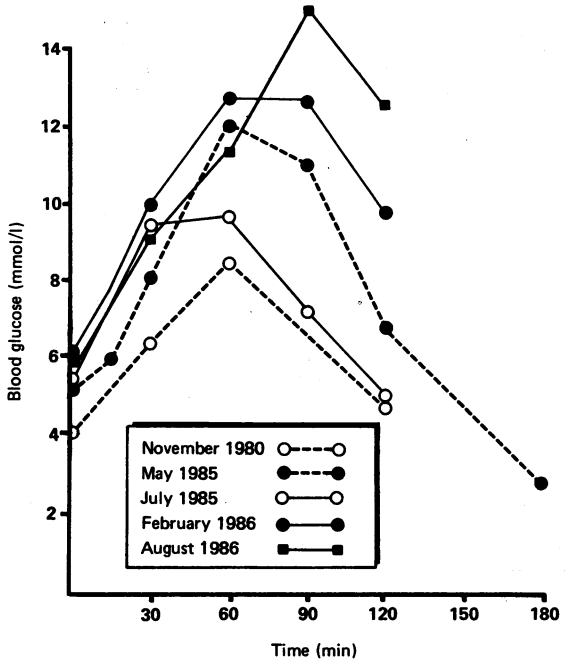
Characteristics of subjects developing insulin dependent diabetes

Case No	Sex and age (years) at start of insulin treatment	HLA-DR type	Duration of islet cell antibody before insulin treatment (months)	Random blood glucose concentration (RBG) >97·5th centile and result of oral glucose tolerance test (OGTT)	Weeks before insulin treatment	Symptoms	Weeks before insulin treatment
1	M 9	1,4	36	—	—	Tiredness Polyuria	36 1 day
2	F 23	3,3	50	RBG 7·7 mmol/l OGTT "diabetic"	52 84	Vaginal candidiasis	28
3	M 24	3,3	84	RBG 7·3 mmol/l OGTT "diabetic"	74 16	Asymptomatic	
4	M 21	3,4	78	RBG 6·4 mmol/l OGTT normal 4 years before	36	Tiredness Polyuria	16 1
5	M 13	1,4	86	RBG 15·4 mmol/l OGTT "impaired"	58 30	Tiredness Polyuria	58 1
6	F 18	4,8	6	RBG 6·7 mmol/l OGTT "impaired"	26 16	Polyuria	4
7	M 5	4,X	30	RBG 6·4 mmol/l	28	Tiredness Polyuria	26 4 days
8	F 45	3,4	57	RBG 7·2 mmol/l	52	Polyuria Failed oral agents	8 26
9	M 20	3,4	23*	—	—	Weight loss	8
10	M 61	4,4	—	RBG 6·4 mmol/l	157	Weight loss Failed oral agents	16 104
11	F 12	—	80	—	—	Weight loss Polyuria	12 4
12	F 16	3,8	—	RBG 29·4 mmol/l	144	Weight loss	52
13	F 46	1,4	23	OGTT "diabetic"	26	Thirst, polyuria	12
14†	M 22	3,4	96	RBG 10·0 mmol/l OGTT "impaired"	364 78	Tiredness	84
15†	M 43	3,4	62	RBG 9·4 mmol/l OGTT "impaired"	64 56	Asymptomatic	

*Islet cell antibody = complement fixing except for case 9.
†Not needed insulin up to November 1986.

diabetic brother. He was positive for islet cell antibody and for thyroglobulin and thyroid microsomal antibodies (thyroid function remained normal). An oral glucose tolerance test in February 1981 gave a normal result, though the insulin response was slightly reduced. A random blood glucose estimation in April 1984 was 7·3 mmol/l and the following April 8·7 mmol/l; at the second estimation his glycosylated haemoglobin concentration was 6·3%. A 25 g intravenous glucose tolerance test showed complete loss of first phase insulin response to glucose, and an oral glucose tolerance test showed him to be diabetic. Fasting blood glucose values were in the diabetic range on three other occasions (7·8, 11·0, and 10·1 mmol/l) but he remained asymptomatic. Glibenclamide failed to reduce his hyperglycaemia and in September 1985 he started to lose weight. He began insulin 17 months after the first recording of a high random blood glucose concentration.

Case 14—A 22 year old man who was haploidentical with his diabetic younger brother had the following random blood glucose concentrations: October 1979, 10·0 mmol/l; January 1980, 8·2 mmol/l; and March 1980, 9·8 mmol/l. In November 1980 an oral glucose tolerance test gave a normal result by WHO and National Diabetes Data Group criteria (figure). In



Case 14. Serial blood glucose responses to oral glucose tolerance test (75 g).

March 1985 he gave a six month history of tiredness; a random blood glucose concentration was 8·8 mmol/l but the glycosylated haemoglobin value was normal (5·4%). An intravenous glucose tolerance test showed a virtually absent first phase insulin response; the result of an oral glucose tolerance test was still normal by WHO criteria but showed impaired glucose tolerance by National Diabetes Data Group criteria. Further oral glucose tolerance tests were carried out in July 1985 and February 1986. By both criteria the first of these gave a normal result but the second showed impaired glucose tolerance. In August 1986 his glucose response was diabetic and his glycosylated haemoglobin concentration had risen to 7·4%; he remained, however, without any specific symptoms.

Discussion

Symptomatic insulin dependent diabetes is simple to diagnose, though in practice recognition is often delayed. In the survey of Hamilton *et al* of 66 newly presenting children 31 had attended their general practitioners more than once before diabetes was recognised; this included 11 of 17 children admitted with keto-acidosis.[2] Childhood diabetes is uncommon in Britain (reported incidence 7·7-13·8 cases per 100 000 a year), though the incidence may be rising.[8] Hence a general practitioner with an average list might see a new case only once in 14 years. In addition, the diagnosis may not be obvious in the early stages; in our study it was overlooked even by parents who already had one child with diabetes. Increased awareness of the possibility of diabetes and routine urine screening would undoubtedly prevent episodes of ketoacidosis and the occasional tragedy. For example, screening the urine of 38 000 schoolchildren identified 18 new cases of diabetes.[9] Even so, glycosuria may not be present with mild hyperglycaemia.

Random blood glucose concentrations may be more useful; Lind and Anderson showed that the normal range for these is surprisingly low.[10] In pregnant women the 99% cut off values were 6·1 mmol/l within two hours of a meal and 5·6 mmol/l more than two hours after. In our series, which included non-diabetic parents as well as siblings, the 97·5% value was 6·3 mmol/l, well below the normal renal threshold for glucose.

Several of our subjects appeared to have had intermittent hyperglycaemia long before symptoms developed. One (case 14) continued to have normal oral glucose tolerance until five years later, though unfortunately no tests were performed at times of the initial high random glucose values. This would be consistent with the concept of recurrent attacks on pancreatic β cells with fluctuating immune destruction and regeneration.[11]

The belief that the onset of glucose intolerance may be slow in type I diabetes is not new and there are isolated case reports of gradual deterioration in glucose tolerance.[12,13] Several studies have shown that testing glucose tolerance in children and adolescents will identify a subgroup with impaired tolerance, but the rate of progression to insulin dependent diabetes is low. The groups tested have been heterogeneous and almost certainly included subjects with maturity onset diabetes of the young.[14] Rosenbloom *et al* examined the 10 year prognosis of impaired glucose tolerance in siblings of children with diabetes and found that five of 19 such children had begun to receive insulin within the next seven years compared with one of 86 with a normal screening test result.[15] Within the Pittsburgh study six subjects (two siblings and four parents) developed diabetes, of whom four needed insulin. Three of these had impaired glucose tolerance or a reduced insulin response 18, 22, and 17 months before they began insulin.[16]

Indirect evidence that the onset may be slow comes from observed differences in height at diagnosis between diabetic and non-diabetic identical twins.[17] There was no difference when diabetes was diagnosed over the age of 19, but the diabetic twin was shorter by a mean of 3·5 cm in eight of 16 presenting before this age. The average period of delayed growth before diagnosis was 35 weeks compared with a duration of symptoms of six weeks.

Not all cases of insulin dependent diabetes have such a prolonged onset, and the histories described reflect the wide clinical range within the disease.[18] Only 10-15% of new patients have a first degree relative with insulin dependent diabetes, so our familial group may not be wholly representative. Nevertheless, there is little to suggest that they differ substantially from sporadic cases. It might be argued that our older subjects were not truly insulin dependent, as definition in this group is difficult.[19] None, however, were obese (all were <110% of ideal body weight); all but two were positive for islet cell antibody; and all possessed the HLA-DR antigens associated with insulin dependent diabetes. It has been suggested that patients possessing HLA-DR3 without DR4 have a less abrupt clinical course, though only three of our subjects fell into this category.[20] Five patients had other autoantibodies and therefore might be considered to have had a primary autoimmune type of diabetes reported to have a slower onset.[21]

The search for possible causes of insulin dependent diabetes continues and interest has again focused on the Coxsackie B4 and B5 viruses. Raised titres of IgM antibodies to these have been detected in some 30% of newly diagnosed children.[22] In view of the lengthy prodrome recorded in our and other studies it seems likely that viral illnesses may act by precipitating rather than initiating the process of β cell destruction.[23] This might explain the apparent clustering of new cases presenting during viral epidemics and the seasonal variation in onset of the disease.[24]

If insulin dependent diabetes typically has a slow onset, as in our series, early diagnosis would give the impression of a more benign course over the first year. The remission or "honeymoon" phase reported in one third to a half of new cases[25] may simply reflect

earlier diagnosis and treatment. Reports of clinical remission induced by immunosuppression have not taken this into account and future trials may need to be evaluated in this light.[26] Early detection of metabolic abnormalities would improve the chances for success of intervention, but prediction at a much earlier stage (even before impaired glucose tolerance is found) would offer the best prospect of preserving viable numbers of functioning β cells.

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Comprehensive care of patients with head injuries

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Abstract

The comprehensive head injury service run by the neurosurgeons at the Hull Royal Infirmary for the surrounding population of one million was analysed. The analysis showed that all patients with either a fractured skull or a lowered level of consciousness should be admitted to a district general hospital because the associated risk of their having a major head injury is over 20%. Those patients with both a fractured skull and a lowered level of consciousness have a 60% likelihood of a major head injury and should be transferred immediately to the neurosurgical unit. Patients with compound or complicated fractures of the skull and those without fractured skulls but with neurological impairment persisting for four hours or more, should also be transferred to the neurosurgical unit.

If these guidelines are followed about 200 patients/million population will be referred to the neurosurgical centre. Patients with a minor head injury and none of the clinical risk factors may safely be sent home. This should reduce the rate of admissions to hospital for head injuries by 60%.

Introduction

When the Royal Infirmary in Hull was opened in 1967 it was decided that all patients who required admission to hospital after a head injury would come directly under the care of the neurosurgeons. In addition to patients with obvious major head injuries, those with minor head injuries were admitted if they had the following signs: loss of consciousness (however brief); headache; and vomiting and unsteadiness, particularly in children. Skull radiographs were obtained for all patients with any kind of head injury, and radiological proof of a fractured skull was a further criterion for admission.

The neurosurgeons at this hospital have thus provided a clinical service for all patients with minor or major head injuries for the population on the north bank of the Humber (about 500 000). During this time the neurosurgeons have also accepted all patients with head injuries referred to them from the two district general hospitals south of the Humber and other smaller hospitals around Hull. The total population served by these referring hospitals is also about 500 000 (see figure). This combined service is part of a neurosurgical practice that has grown in recent years (see table I) with satisfactory results.

In the past two years the identification of several factors correlated with the risks of intracranial haematoma in patients with

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