BMJ. Aug 8, 1998; 317(7155): 359–360. PMCID: PMC1113665

The changing classification and diagnosis of diabetes

New classification is based on pathogenesis, not insulin dependence

Nicholas J Wareham, MRC clinician scientist fellow

Department of Community Medicine, University of Cambridge, Cambridge CB2 2SR (Email: njw1004@medschl.cam.ac.uk)

Stephen O'Rahilly, Professor of metabolic medicine

University of Cambridge Departments of Medicine and Clinical Biochemistry, Addenbrooke's Hospital, Cambridge CB2 2QR (Email: sorahill@hgmp.mrc.ac.uk)

See the article "Will new diagnostic criteria for diabetes mellitus change phenotype of patients with diabetes? Reanalysis of European epidemiological data" on page 371.
See the article "Diabetes care in general practice: meta-analysis of randomised control trials" on page 390.
This article has been cited by other articles in PMC.

At its annual meeting in June 1997 the American Diabetes Association announced the conclusions of an expert committee, which recommended changes to the way that diabetes is classified and to the choice of diagnostic method and cut off value that should be used to define the disease.[1] A provisional report from a World Health Organisation consultation group, with some overlap in members with the American committee, has recently been published.[2] These recommendations could have important epidemiological implications, but they will also affect individual patients.

The previous classification of diabetes was based on the extent to which a patient was dependent on insulin.[3] Although this was a logical distinction that separated the two main forms of diabetes, it gave rise to clumsy and sometimes confusing subcategories. Both the reports of the American Diabetes Association and the WHO recommend altering the classification to define four main subtypes of diabetes. Type 1 includes immune mediated and idiopathic forms of β cell dysfunction which lead to absolute insulin deficiency. Type 2 diabetes is disease of adult onset, which may originate from insulin resistance and relative insulin deficiency or from a secretory defect. Type 3 disease covers a wide range of specific types of diabetes including the various genetic defects of β cell function, genetic defects in insulin action, and diseases of the exocrine pancreas. Type 4 disease is gestational diabetes.

The move to a classification that allows for subgrouping by pathogenesis is an explicit recognition of the heterogeneity of processes that lead to diabetes. It is forward looking as it creates a framework that can accommodate the increasing number of specific causes for diabetes which are likely to be discovered.[4] The hope is that better subclassification will lead to more precise targeting of specific treatments and eventually to better outcomes.

The American report also considers how to define diabetes when the diagnosis is in doubt. Clinically, there is no difficulty when there are symptoms and unequivocal hyperglycaemia, but there is much greater complexity in asymptomatic patients with lesser degrees of glucose intolerance. In part, both committees were reacting to criticisms that the previous definition relied too strongly on the oral glucose tolerance test, which, although widely used in epidemiological studies, is rarely performed in clinical practice.[5] The 1985 WHO definition of diabetes,[3]

based on the 75 g oral glucose tolerance test, defined diabetes either by a fasting plasma glucose concentration of ⩾7.8 mmol/l or by a glucose concentration of ⩾11.1 mmol/l at 2 hours after the glucose challenge. These diagnostic thresholds were selected because in certain high prevalence populations glucose concentration at 2 hours after glucose challenge has a bimodal distribution that can be separated into two distinct subgroups, and also because of the link between glucose concentration at 2 hours and future risk of the specific microvascular complications of diabetes. It is clear from longitudinal studies, however, that other tests such as fasting glucose concentration or glycated haemoglobin could equally well predict future microvascular risk, and that appropriate and equivalent thresholds could be set for any of these tests.[6] Because of its simplicity and availability, the American Diabetes Association's report recommends basing the diagnosis of diabetes on the fasting glucose concentration.

A change is also proposed to the diagnostic cut off point for fasting glucose concentration, reducing it from 7.8 mmol/l to 7.0 mmol/l. This change introduces a new intermediate category, impaired fasting glucose, defined as a fasting glucose concentration of 6.1-<7.0 mmol/l. There is evidence that these changes will have little effect on the true prevalence of diabetes, as described by Borch Johnsen et al on behalf of the DECODE group in this issue (p 371).[7] Nevertheless, there will be considerable reclassification of individuals when these new criteria are compared with the previous WHO definition, as the diagnostic emphasis is on fasting hyperglycaemia rather than the dynamic response to an oral glucose load. The DECODE group also show that this reclassification is not random but depends on age and obesity. Therefore the proposed changes will have an impact on the phenotype of people classified as having diabetes, as the new criteria are more likely to identify middle aged obese individuals. Perhaps most importantly, these changes are likely to lead to an increase in the prevalence of diagnosed diabetes as it would become practically much easier to detect the large number of people whose disease is currently undiagnosed.[8]

The most contentious part of the American Diabetes Association report, and one not considered by the WHO, is the recommendation that testing for diabetes should be considered for everyone aged 45 or over and should be repeated every three years. Testing is also recommended for younger people with a variety of risk factors such as obesity (liberally defined as a body mass index of ⩾27 kg/m^2) or a family history of diabetes. Although the prevalence of undiagnosed disease is high[8] and many patients have evidence of complications at diagnosis,[9] the recommendations for screening are not backed by evidence that earlier detection leads to fewer adverse outcomes or that such a programme would be cost effective.

Overall, the practical implications of these reports for clinical practice are that the diagnosis of diabetes in people with classic symptoms should be established with a random plasma glucose concentration of ⩾11.1 mmol/l, preferably repeated or confirmed by a raised fasting glucose value on a subsequent day. In less clear cases the diagnosis can be established with a fasting plasma glucose of ⩾7.0 mmol/l, again repeated on a different occasion. Although the American Diabetes Association report was published as the final findings of its expert committee, the paper from the WHO is labelled as a provisional report. Individuals or groups who want to make comments and suggest modifications should write to the cochairmen by the end of September 1998.[2]

Notes

Go to:

Papers p 371, *General practice* p 390

References

Go to:

1. Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. Report of the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. Diabetes Care. 1997;20:1183–1197. [PubMed]

2. Alberti KGMM, Zimmet PZ.for the WHO consultation. Definition, diagnosis, and classification of diabetes mellitus and its complications. Partiagnosis and classification of diabetes mellitus. Provisional report of a WHO consultation Diabetic Med 1998. 15539–553.553 [PubMed]

3. World Health Organisation. Diabetes mellitus: report of a WHO Study Group. Geneva: WHO; 1985. (Technical Report Series 727.)

4. O'Rahilly S. Science, medicine, and the future: non-insulin dependent diabetes mellitus: the gathering storm. BMJ. 1997;314:955–959. [PMC free article] [PubMed]

5. Drash AL. Is the oral glucose tolerance test obsolete? Diabetes Care. 1995;18:1072–1073. [PubMed]

6. McCance DR, Hanson RL, Charles M-A, Jacobsson TH, Pettitt DJ, Bennett PH, et al. Comparison of tests for glycated haemoglobin and fasting and two hour plasma glucose concentrations as diagnostic methods for diabetes. BMJ. 1994;308:1323–1328. [PMC free article] [PubMed]

7. Borch-Johnsen K., for DECODE study groupon behalf of the European Diabetes Epidemiology Study Group. Will new diagnostic criteria for diabetes mellitus change phenotype of patients with diabetes? Reanalysis of European epidemiological data BMJ 1998. 317371–375.375 [PMC free article] [PubMed]

8. Williams DRR, Wareham NJ, Brown DC, Byrne CD, Clark PMS, Cox BD, et al. Undiagnosed glucose intolerance in the community: the Isle of Ely diabetes project. Diabetic Med. 1995;12:30–35. [PubMed]

9. Harris MI, Klein RE, Welborn TA, Knuiman MW. Onset of NIDDM occurs at least 4-7 years before clinical diagnosis. Diabetes Care. 1992;15:815–819. [PubMed]