The BMA's guidance is not a consensus document. However, it follows a wide ranging consultation exercise, during which over 2000 responses were considered and were instrumental in deciding its scope and contents. The list of factors that should be taken into account in making decisions to withdraw or withhold treatment and the recommended extra safeguards in relation to withdrawing or withholding artificial hydration or nutrition from patients who are not in a persistent vegetative state fill longstanding lacunas. These include, for the former, the quality of the evidence about the condition; the need for a proper assessment of the benefits, risks, and burdens of treatment; the level of awareness the patient has of his or her existence or surroundings; and the views of people close to the patient about what the patient is likely to see as beneficial; and, for the latter, the need for an independent formal clinical review. Nevertheless, like many reports giving guidance on ethical aspects of medical practice, the report also raises many new issues. There can be no doubt that both legal and ethical thinking relating to this difficult area of medical practice will continue to evolve over the next decade.

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Gluten sensitivity: a many headed hydra

Heightened responsiveness to gluten is not confined to the gut

In a lecture entitled "On the coeliac affection"[1] given in London in 1887 Dr Samuel Gee first described the condition we now refer to as coeliac disease or gluten sensitive enteropathy. With clinical manifestations confined to the gastrointestinal tract or attributable to malabsorption, it was logical to assume that the key to the pathogenesis of this disease resided in the gut. However, focusing diagnostic criteria on the gut (as most physicians still do) has delayed the appreciation of the wider spectrum of gluten sensitivity.

The treatment of coeliac disease remained empirical until 1940-50, when the Dutch paediatrician Willem Dicke noted the deleterious effect of wheat flour on individuals with coeliac disease.[2] Removal of all dietary products containing wheat resulted in complete resolution of the gastrointestinal symptoms and a resumption of normal health. The introduction of the small bowel biopsy in 1950-60 confirmed the gut as the target organ in coeliac disease. The characteristic features of villous flattening, crypt hyperplasia, and increase in intraepithelial lymphocytes with improvement on gluten free diet became the mainstays of the diagnosis of coeliac disease.

However, in 1966 Marks et al showed an enteropathy with a striking similarity to coeliac disease in 9 out of 12 patients with dermatitis herpetiformis.[3] The enteropathy and the rash were gluten dependent and the skin disease could occur even without histological evidence of gut involvement. This discovery started to shift the emphasis from the gut as the sole protagonist in this disease. With dermatitis herpetiformis too came the concept of "latent" gluten sensitivity. The term is now used to describe people with a histologically normal small bowel while on a normal diet who at some stage of their lives have had or will have an abnormal small bowel that responds to a gluten free diet.[4]

Also in 1966 Cooke and Thomas-Smith published a paper on neurological disorders associated with adult coeliac disease.[5] Further case reports have since been published, but most are based on patients with coeliac disease who later develop neurological dysfunction, implying that gut disease is a prerequisite. We have, however, shown that neurological dysfunction can not only precede coeliac disease but can also be its only manifestation.[6] Of even more interest is the demonstration of a high prevalence of circulating antigliadin antibodies (IgG, IgA, or both) in patients with neurological dysfunction of obscure aetiology (57% *v* 5% in neurological controls and 12% in normal controls).[7] Only 35% of these patients had histological evidence of coeliac disease. The remaining 65% have gluten sensitivity where the target organ is the cerebellum or the peripheral nerves, a situation analogous to that of the skin in dermatitis herpetiformis.

In the light of these findings the specificity of antigliadin antibodies has been questioned yet again. When the histological criteria for coeliac disease are used as the gold standard, IgG antigliadin antibody has low specificity. Nevertheless, IgG antigliadin antibodies have a high sensitivity not only for patients with coeliac disease but also for those with minimal or no bowel damage where the principal target organ is the cerebellum or peripheral nervous system. Supportive evidence for this contention comes from the HLA genotype of patients with neurological disorders associated with gluten sensitivity. As coeliac disease has one of the strongest HLA associations of any immune disease (HLA DQ2 in more than 90% of patients) one would expect that patients positive for antigliadin antibodies and with normal duodenal mucosa should have a similar HLA genotype if they are truly gluten sensitive. In fact 85% of our patients with neurological disorders associated with gluten sensitivity have an HLA genotype in keeping with coeliac disease compared with 25% of the normal population.[8]

Unlike antiendomysium or antireticulin antibodies, antigliadin antibodies are antibodies against the extrinsic causal factor for gluten sensitivity. Antiendomysium antibodies may be more specific for coeliac disease, but no large scale data are available as yet on their specificity or sensitivity in patients with gluten sensitivity where the immunological target organ may be other than the gut.

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The typical clinical expression of a patient with gluten sensitivity where the sole manifestation is neurological is cerebellar ataxia, often with a peripheral neuropathy.[9] Most of these patients will have histologically normal mucosa on biopsy and few or no gastrointestinal symptoms. Both the ataxia and the neuropathy may be reversible with adherence to a gluten free diet.[9]

Marsh's "modern" definition of gluten sensitivity is to be recommended: "a state of heightened immunological responsiveness to ingested gluten in genetically susceptible individuals."[10] Such responsiveness may find expression in organs other than the gut. Gastroenterologists, dermatologists, neurologists, and other physicians need to be aware of these developments if the diagnosis and treatment of the diverse manifestations of gluten sensitivity are to be advanced. The aetiology of such diverse manifestations presents the next challenge.

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Helping parents identify severe illnesses in their children

Baby Check may not do this but it can improve the quality of a consultation

General practice p 1740

The management of sick children is both a microcosm of primary care and a test of its success—if we cannot get this right we cannot justify any claims to excellence. General practitioners see lots of children, and most of them have minor, self-limiting illness. The fourth national morbidity survey reports a consulting rate of 4.97 for children aged 4 and under, a rate exceeded only by those aged 75 and over. When the illnesses are classified as minor, intermediate, or serious the consulting rate for minor illness is the highest of any age group.[1] General practitioners have the task of dealing with this high volume of work quickly and efficiently, without overtreating the children or making parents feel they have been the victims of perfunctory or, worse, incompetent care. At the same time, and most important of all, they must be able to identify the small numbers of children with serious illness.

For many general practitioners the amount of time that work with minor illnesses takes up, among adults as well as children, is a source of frustration. We know we can do next to nothing to alter the course of the illness and often it is hard so see what either doctor or patient gets out of the encounter. If patients could be encouraged to deal with minor illness without professional help their confidence in their health and their sense of independence would be enhanced, and professionals' time could be liberated to address other tasks where our skills are more effectively deployed. Any intervention that supports parents' ability to identify minor illness correctly and professionals' ability to identify serious illness correctly is welcome.

In this week's issue Thomson and colleagues report a randomised controlled trial using Baby Check to improve parents' assessment of their babies' health (p 1740).[2] Baby Check is a scoring system using 19 symptoms and signs, designed to enable parents and professionals to assess the seriousness of illness in babies aged 6 months and under. It grades infantile illness into four levels of severity.[3] The study by Thomson et al confirms the prediction of the original Baby Check authors, that its use would not increase the numbers of mothers seeking medical care.[4] Neither, however, did Baby Check reduce the demand for care. Nor did it influence the demand for out of hours services or the patterns of prescribing that took place in the subsequent consultations.

Enthusiasts for Baby Check should not be disappointed with this result. At one end of the range of illness severity it does have the potential to empower parents with better information. In the original field trials up to 81% of mothers found it useful and 96% would recommend it to others.[4] In a subsequent qualitative study Kai has reported on the use of Baby Check in a group of socioeconomically disadvantaged families: the parents found that it was helpful, reducing anxiety and increasing their confidence in coping with illness and dealing with doctors.[5] General practitioners both trusted it and reported that they would want mothers, health visitors, and midwives to use it.[6]

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