GUIDELINES

Diagnosis and management of attention-deficit/hyperactivity disorder in children, young people, and adults: summary of NICE guidance

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Why read this summary?

Attention-deficit/hyperactivity disorder (ADHD) is a common childhood condition that may affect different areas of the child's life, seriously impairing academic achievement, peer relationships, and self care. Many affected children become socially isolated and develop conduct problems. Some 15% of children with ADHD will still have the condition in adulthood, and even more will develop a personality disorder and/or a substance misuse disorder in adulthood.[1-3]

Diagnosis rates for ADHD and prescriptions of stimulant medication have risen substantially in England during the past decade, with 220 000 prescriptions for stimulants (costing about £5m (€6.3m; $9.4m)) in 1998 and 418 300 (almost £13m) in 2004.[4] The prescription of stimulants, which are potential drugs of misuse, to children remains controversial, with concerns about their safety and the potential for misuse and diversion (where the drug is passed on to others for non-prescription use).This article summarises the most recent recommendations from the National Institute for Health and Clinical Excellence (NICE) on the diagnosis and management of ADHD in children, young people and adults.[5]

Recommendations

NICE recommendations are based on systematic reviews of best available evidence. When minimal evidence is available, recommendations are based on the guideline development group's opinion of what constitutes good practice. Evidence levels for the recommendations are in the full version of this article on bmj.com.

Service organisation and training of professionals

Specialist ADHD teams for children and young people and equivalent teams for adults should jointly develop training programmes on the diagnosis and management of ADHD for all mental health, paediatric, social care, educational, forensic, and primary care providers who have contact with people with ADHD.

Diagnosis and assessment

- Diagnose ADHD when all of the following three conditions apply:

  -The symptoms of hyperactivity, impulsivity, and inattention meet the criteria for ADHD in the DSM-IV (*Diagnostic and Statistical Manual of Mental Disorders*, fourth edition) or for hyperkinetic disorder in the ICD-10 (international classification of diseases, 10th revision)

  -Impairment is at least of moderate clinical and/or psychosocial significance. This means when the level appropriate to the child's chronological and mental age has not been reached in several domains (for example, achievement in schoolwork or homework, dealing with physical risks, and avoiding common hazards; forming positive relationships with family and peers; developing a positive self image; and avoiding criminal activity). The level of dysfunction could also be estimated from cut-offs on an overall adjustment scale (such as a score of <60 on the children's global assessment scale[6]).[7] Comparable impairments may be seen with adults.

  -The apparent symptoms of ADHD are pervasive (that is, occurring in two or more settings such as home, school, or workplace).
- Assess the person's needs, coexisting psychiatric conditions; social, familial, and educational, and/or occupational circumstances; physical health; and for children and young people, their parents' or carers' mental health.

Treatment in children and young people

Preschool children

- Offer parents or carers of children of preschool age a referral to a parent training or education programme as the first line treatment.
- Drug treatment is not recommended for children of preschool age with ADHD.

Teachers of school age children

- Teachers trained in ADHD and its management should provide behavioural interventions in the

classroom to help children with the condition. An example would be a token system whereby the teacher lists the child's responsibilities, assigns a value to each responsibility, and awards or removes points on the basis of good behaviour (and/or good attitude) and regulation of emotions. The teacher also removes points when the child exhibits specific serious, defiant behaviours.

School age children and young people with moderate ADHD

- Offer their parents or carers a referral to a parent training or education programme, either on its own or together with a group treatment programme for the child or young person (cognitive behavioural therapy and/or social skills training). Most parenting programmes combine elements of the two main approaches: behavioural programmes, which focus on teaching the parenting skills needed to reduce and cope with problem behaviour; and relationship programmes, which aim to help parents to understand both their own and their child's emotions and behaviour and to improve their communication with the child.
- When parents or carers attend parent training or education programmes, the professional delivering the sessions should consider contacting the school to provide the child's teacher with written information on the areas of behavioural management covered in these sessions.
- Offer drug treatment to those who have refused non-drug interventions or to those whose symptoms have not responded sufficiently after their parents or carers have attended parent training or education programmes or after group psychological treatment. Depending on a range of factors—such as the presence of coexisting conditions, side effects, and patient preference—the child or young person may be offered methylphenidate, atomoxetine, or dexamfetamine. Drug treatment should be started only by an appropriately qualified healthcare professional with expertise in ADHD and should be based on comprehensive assessment and diagnosis. Continued prescribing and monitoring of drug treatment may be performed by general practitioners, under shared care arrangements.

School age children and young people with severe ADHD

- Offer drug treatment (with methylphenidate or atomoxetine) to those with severe ADHD, for whom it should be first line treatment. At the same time offer families referral to a group based parent training or education programme. Drug treatment should be started only by an appropriately qualified healthcare professional with expertise in ADHD and should be based on a comprehensive assessment and diagnosis.
- Drug treatment should always be part of a comprehensive treatment plan that includes psychological, behavioural, and educational advice and interventions.

Dietary advice

- The elimination of artificial colouring and additives from the diet is not recommended as a generally applicable treatment for children with ADHD.

Treatment in adults

- Offer drug treatment (with methylphenidate as the first line treatment) unless the person would prefer a psychological approach. Drug treatment should be started only by an appropriately qualified healthcare professional with expertise in ADHD and should be based on a comprehensive assessment and diagnosis.
- Drug treatment should always form part of a comprehensive treatment programme, which should aim to meet psychological, behavioural, and educational and/or occupational needs.

Overcoming barriers

Attention-deficit/hyperactivity disorder has been increasingly recognised in the UK over recent years, although the provision of treatment has been variable.[8 9] Although drugs (stimulants and atomoxetine) have been the mainstay of treatment for children and young people, adults are less likely to receive these despite evidence of effectiveness[6]; moreover, geographical variation is considerable.[10]

The NICE guideline aims to correct this situation. For children and young people the care pathway and treatments offered in the guideline are comprehensive, including guidance for professionals in health, mental health, social care, and education. For adults, drug and psychological treatments are recommended. To tackle any challenges, oversee the implementation of this guideline, coordinate and introduce training programmes (including those for teachers), and oversee the development of parent training and education programmes, NICE recommends that every locality should form a multiagency group. This group should comprise professionals from specialist ADHD teams, paediatrics, mental health and learning disability trusts, forensic services, child and adolescent mental health services, the Children and Young People's Directorate, and parent support groups.

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Commentary: Controversies in NICE guidance on attention-deficit/hyperactivity disorder

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In 2002 both attention-deficit disorder and hyperactivity appeared in a list of human problems that *BMJ* readers believed were "non-diseases."[1] In producing its third and most comprehensive synthesis of research, clinical consensus, and economic analysis on attention-deficit/hyperactivity disorder (ADHD),[2] the National Institute for Health and Clinical Excellence (NICE) will no doubt fuel the controversy about the nature of ADHD.

The full report of the NICE guidance acknowledges the social scientific paradigm that casts doubt on the utility and appropriateness of ADHD as a diagnostic category. The report goes on to examine the diagnosis of ADHD and concludes it is a valid concept. Persistent sceptics will read sobering accounts from both research and personal testimony of the impairment experienced by hyperactive people, including the iatrogenic impairment resulting from professional ignorance and disbelief.

If those who purchase services fund fully NICE's recommendations for a stepped care approach to managing childhood ADHD and for ubiquitous mental health care for the estimated 3% of adults with ADHD, then clinical services will look quite different from the current inadequate and varied provision. NICE rightly says that full implementation of the guidance will take time. It sets out a bold vision of improvement in both child and adult services.

The guidance moves the recognition and initial management of childhood ADHD out of secondary care clinics and into the broad range of universal and targeted children's services slowly developing in NHS trusts and local authorities. It proposes that these services should offer evidence based parent training programmes to all families who have a child with ADHD. The use of this model of "comprehensive child and adolescent mental health services" as outlined in the Department of Health's document *The Mental Health and Psychological Wellbeing of Children and Young People*[3] will be a stiff challenge to joint purchasing processes, which are still in their infancy.[4]

Community paediatricians and child and adolescent psychiatrists will find that much of the information about the assessment and medical management of children with severe ADHD is similar to previous international guidelines.[5,6] The main positive impact of the guidance for children's specialists seems to be that children with suspected ADHD should arrive at secondary care clinics having already had some assessment and intervention. More controversially, the existing multimodal, multiprofessional interventions which are assumed to be good practice in comprehensive child and adolescent mental health services are not supported by NICE's economic evaluations. If guided by NICE's assertion that group based parent training for all affected children and medication for severely impaired children are the two best treatments, future purchasing decisions may eventually reduce the breadth of specialist service provision for children with ADHD.

The inclusion of adult ADHD in the guidance will be welcomed by adults whose ADHD has previously gone unrecognised, misdiagnosed, or untreated. The guidance outlines services for diagnosis, medication management, and psychological intervention for adults while recognising that the current lack of training and service provision in adult mental health services is a major impediment to implementation. NICE recommends prescribing methylphenidate as first line treatment, and many adult psychiatrists may be initially reluctant to do this. While psychostimulants have been used in children for 70 years, methylphenidate is not licensed for adults. Service purchasers keen to set up new provision for adult ADHD should be wary of taking headlines about the NICE recommendations at face value: the assertion that psychological

intervention for adults with ADHD is best delivered in a group format is based on a comparison of just two trials.

Interventions not endorsed for ADHD in the guidance are unlikely to cause much controversy: specifically not recommended are elimination diets, polyunsaturated fatty acid supplements, and antipsychotic drugs.

NICE charges local ADHD teams and multiagency groups with the task of training both health and education staff. Such training could usefully have been the subject of an economic analysis as it will be far from cost neutral. Changing professional thinking away from the "non-disease" model of ADHD will not be accomplished by single formal teaching sessions. Those who know ADHD, its morbidity, and the successes seen in treatment will need to work regularly alongside those who do not to bring about lasting changes in practice.

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UNCERTAINTIES PAGE

How effective are some common treatments for traumatic brain injury?

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This is a series of occasional articles that highlights areas of practice where management lacks convincing supporting evidence. The series advisers are David Tovey, editorial director, BMJ Knowledge, and Charles Young, editor of BMJ Clinical Evidence, and editor in chief, BMJ Point of Care.

Surveys show that mannitol, hyperventilation, cerebrospinal fluid drainage, and barbiturates are commonly used in the United Kingdom, Europe, and the United States to treat traumatic brain injury.[1-3] Yet the effects of such treatments are uncertain.

Traumatic brain injury is a major cause of death and disability worldwide. Every year at least 10 million people sustain a traumatic brain injury serious enough to result in death or admission to hospital.[4] Bearing in mind that almost half of all patients with traumatic brain injury experience long term disability[5 6] and that most injury occurs in young adults, the medical, social, and financial burden is clear.

What is the evidence of the uncertainty?

The Cochrane Injuries Group maintains a specialised register of randomised controlled trials of interventions for traumatic brain injury and has searched extensively for trials evaluating the effects of mannitol, hyperventilation, cerebrospinal fluid drainage, and barbiturates. The group has also prepared, and regularly updates, systematic reviews to assess the effects of barbiturates,[7] hyperventilation,[8] and mannitol.[9]

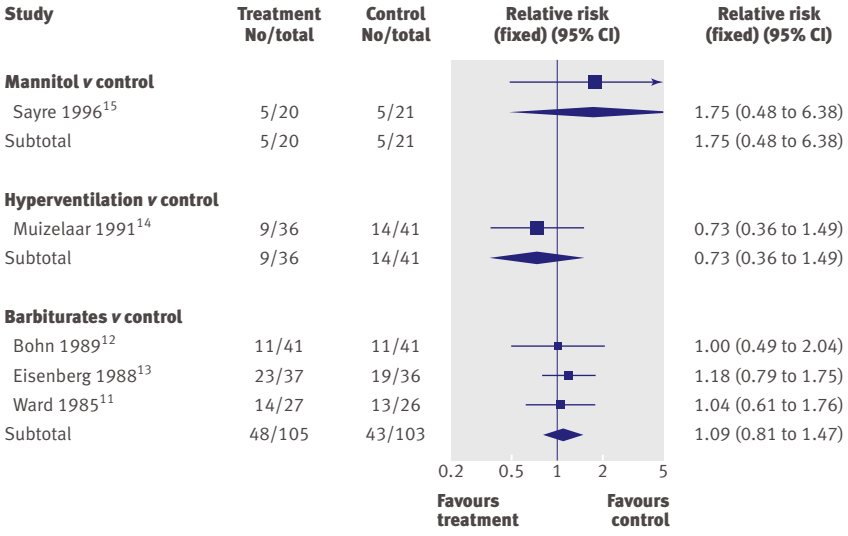
In 1998 the Cochrane Injuries Group highlighted the absence of reliable evidence for the effectiveness of these four treatments[10] when searches identified only three small trials of barbiturates,[11-13] none of cerebrospinal fluid drainage, one small trial of hyperventilation,[14] and one small trial of the use of mannitol.[15] Our latest searches, to January 2008, indicate that there remains a lack of adequately powered randomised controlled trials of these interventions, with no additional trials found. The uncertainty is evident in the meta-analyses presented in the figure. The relative risks of death for barbiturates, hyperventilation, and mannitol are compatible with both moderate decreases and moderate increases in the risk of death, and no estimate is available for cerebrospinal fluid drainage owing to the absence of any clinical trial data. The existing trials are far too small to detect clinically plausible treatment effects.

Evidence of improved clinical outcomes with high dose mannitol compared with low dose mannitol[16-18] provided indirect evidence that mannitol administration may be useful, but an investigation by the Cochrane Injuries Group could not confirm the validity of the trials in question,[19] which have now been withdrawn from the Cochrane Review.

The previous uncertainty surrounding the use of corticosteroids in traumatic brain injury, however, was resolved by the large scale (10 008 randomised patients) CRASH-1 trial in 2004 (www.crash.lshtm.ac.uk).[20] The current evidence shows that administration of corticosteroids after brain injury does more harm than good.

Is ongoing research likely to provide relevant evidence?

To the best of our knowledge no clinical trials are currently being conducted aimed at resolving these uncertainties. Searches by the Cochrane Injuries



Summary of relative risks for death at the end of studies on mannitol, hyperventilation, and barbiturates. No estimate is available for cerebrospinal fluid drainage as no clinical trials were found

Group covering all dates to January 2008 have not identified any further unpublished or ongoing trials that would contribute relevant evidence about the effectiveness of barbiturates, cerebrospinal fluid drainage, hyperventilation, and mannitol in the treatment of traumatic brain injury.

What should we do in the light of the uncertainty?

It is essential that clinicians and the public—as users and potential healthcare users—are fully informed of the uncertainties surrounding the efficacy of these commonly used treatments. Until these uncertainties are resolved, clinicians should continue to make treatment decisions based on their judgment and experience according to the best available evidence. However, influencing the research agenda in order to tackle these uncertainties is the key challenge.

Trauma is one of the most neglected areas of research.[21,22] The reasons for this are unclear, although the following may be contributing factors. Firstly, traumatic brain injury is an acute, unexpected condition with a high case-fatality rate. These patients, at the time of their need for treatment, are not in the position to consider the uncertainties of treatments and lobby for further trials.

Secondly, conducting clinical trials in the emergency setting is challenging, with a major obstacle being the failure to appreciate that unconscious patients in emergency situations are an exception to the general requirement for informed consent for medical research.[20]

Thirdly, although trauma is a leading cause of death and disability worldwide, it is largely a problem for low and middle income countries, where 90% of the deaths occur.[23] Most research infrastructure and funding, however, are found in high income countries, where although trauma is an important cause of premature mortality, it is not a leading cause, and thus may not be a research priority. Pharmaceutical companies also show less interest in examining uncertainties surrounding commonly used licensed treatments, such as those featured here, preferring to invest in research into new patentable treatments—for obvious commercial reasons.

Need for a different approach

Ten years after the Cochrane Injuries Group highlighted the uncertain effectiveness of these four treatments,[10] there has been little progress. A different approach is needed. Lessons could be learnt from research strategies for other neglected conditions, such as a neglected tropical disease initiative recently launched with support from governmental and non-governmental organisations.[24] Global partnerships, including such organisations as the new Carso Health Institute (which has recognised the huge health burden from injuries in Latin America and made injury research a priority), may raise the profile and generate the necessary financial resources to tackle these and other important uncertainties in trauma research.

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LESSON OF THE WEEK

Fatal reactivation of hepatitis B after chemotherapy for lymphoma

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Routine screening for hepatitis B in patients receiving chemotherapy or immunotherapy will save lives

One third of the world's population has evidence of previous infection with the hepatitis B virus (hepatitis B core antibodies), and 350 million people have chronic infection (hepatitis B surface antigen).[1] Global migration will change the prevalence of disease in the UK; currently 200 000 people are chronically infected, and around 1500 acute and 8000 chronic new infections are diagnosed annually (www.hpa.org.uk). Although intravenous drug users and homosexual men are at notable risk, most cases are in people coming from high prevalence areas, where vertical transmission is common.[2]

Patients receiving chemotherapy or immunomodulatory drugs who have been exposed to hepatitis B virus are at risk of viral reactivation.[3-5] In this context, and particularly when steroids are included in the treatment protocol, it is thought that immune mechanisms keeping viral replication under control are suppressed, allowing unchecked viraemia. This occurs in a large proportion of patients who have been infected with hepatitis B virus, and it can be fatal. However, screening for hepatitis B virus before starting immunosuppressive treatments or chemotherapy is not done throughout the United Kingdom.

We present a case of fatal hepatitis B virus reactivation in a young woman treated for lymphoma. With predictions of a rising prevalence of hepatitis B virus in the UK, clinicians prescribing chemotherapy or immunosuppressive treatments (including biological agents such as rituximab) should adopt strategies for screening for hepatitis B and give prophylaxis where required to prevent similar occurrences.

Case report

A 21 year old woman originally from West Africa presented to hospital with a two month history of pleuritic chest pain and weight loss, having previously been fit and well. Chest x ray confirmed a large anterior mediastinal mass and associated left sided pleural effusion. After computed tomography, mediastinoscopic biopsy was performed. Histology confirmed a high grade mediastinal large B cell lymphoma. Bone marrow was normal, as were results of routine blood tests, including liver function tests. An HIV test was negative.

Treatment with R-CHOP chemotherapy (rituximab, cyclophosphamide, doxorubicin, vincristine, and prednisolone) was started. After the fourth cycle of treatment, imaging showed that the mediastinal mass was smaller.

Two weeks later she presented with nausea and vomiting; although clinical examination was normal, liver function tests showed serum transaminase values more than 10 times the upper limit of normal. Further history confirmed no other risk factors for liver disease, but she mentioned transient childhood jaundice. In the next three days transaminase values continued to rise, and jaundice and coagulopathy developed. Doppler ultrasound examination of the liver was normal but hepatitis B testing confirmed ongoing infection (hepatitis B surface antigen was positive; e antigen was negative and e antibody was positive; hepatitis B core antibody was IgM negative and IgG positive; and hepatitis B DNA was 4.522×10^8 IU per ml). Alternative diagnoses were excluded by an extended hepatitis screen, along with a comprehensive biochemical liver screen. Testing of a stored serum sample taken before chemotherapy showed that before treatment she was positive for hepatitis B surface antigen and negative for e antigen, suggesting chronic infection with a precore stop-codon variant.

Lamivudine antiviral therapy was started, and her care was transferred to the regional liver unit. Despite this, fulminant hepatic failure developed, with associated encephalopathy, renal failure, sepsis, uncontrolled haemorrhage, and acute lung injury. She met the criteria for super urgent liver transplantation, but the aggressive,

partially treated lymphoma, which was confirmed by repeat imaging, precluded transplantation because it was felt she would not derive overall long term survival benefit from the procedure. Full supportive care was given on the intensive care unit, but a week later she died.

Comment

Reactivation of hepatitis B virus with immunosuppressive treatments is well described. It is most widely reported in connection with chemotherapy used to treat lymphoma. In a prospective study from China in which 27 patients positive for hepatitis B surface antigen underwent chemotherapy for lymphoma, 18 experienced viral reactivation, six became jaundiced, one developed non-fatal liver failure, and one died of liver failure.[3] Another study of 305 patients from Slovenia found that 78% of patients carrying hepatitis B surface antigen had viral reactivation, and death from liver failure occurred in 37% of these.[4] One study of the risk factors predicting reactivation found that using steroids, being hepatitis B virus DNA positive before chemotherapy, and having lymphoma or breast cancer all predicted viral reactivation.[6]

Although it is widely recognised that reactivation can occur with conventional immunosuppression, fewer clinicians are aware of the risk of hepatitis B reactivation with newer drugs, in particular rituximab, which has a higher risk of hepatitis than does conventional chemotherapy for lymphoma. Although lysis of hepatocytes infected with hepatitis B virus is mediated mainly by cytotoxic (CD8 positive) T cell immunity, B cells have important roles in priming the cytotoxic T cell responses in hepatitis B infection.[5] Reactivation of hepatitis B virus is also widely reported in patients undergoing immunosuppressive treatments for non-malignant disorders such as rheumatoid arthritis and inflammatory bowel disease.[7] The American Association for the Study of Liver Diseases has recommended that people at high risk of hepatitis B virus infection should be tested for hepatitis B surface antigen before they are given chemotherapy or immunotherapy.[2] However, reactivation can occur in people who are negative for hepatitis B surface antigen but positive for hepatitis B core antibody.[8]

Before antiviral agents were widely available, the only strategy for patients who tested positive for hepatitis B virus was to adjust their treatment, an approach that prejudices the chance of successfully treating the underlying disease. In our case, forgoing rituximab would have reduced the chance of developing viral reactivation, but the chance of five year survival would have been reduced by about 15%.[9] A study that omitted prednisolone from standard treatment for non-Hodgkin's lymphoma in patients positive for hepatitis B surface antigen found that overall survival at four years was lower—but jaundice occurred in only 4% of those not receiving steroids, compared with 28% of those who did.[10]

The antiviral drug lamivudine has been shown to be highly efficacious in preventing hepatitis B virus reactivation in these circumstances.[11] Although there are no randomised control trials, there are case-control studies. For example, Li et al compared a group of 40 patients positive for hepatitis B surface antigen who received lamivudine before starting and for eight weeks after finishing chemotherapy and a group, comparable in most characteristics, of 116 historical controls.[12] The lamivudine group had significantly less hepatitis (17.5% *v* 51.7%); hepatitis was of a lower grade in the lamivudine group (2.5% *v* 36.2% with grade 3 or 4 hepatitis), and death from hepatitis was less likely (0% *v* 5.2%). Data on mortality support the use of pre-emptive lamivudine, rather than waiting to see if hepatitis does occur: by the time of impending liver failure, lamivudine is less likely to be clinically efficacious.[13]

International guidelines recommend specific screening for hepatitis B core antibodies (or hepatitis B surface antigen) before chemotherapy or immunotherapy in people defined as high risk on the basis of geographic origin or behaviour patterns.[2] The group at increased risk includes anyone with multiple sexual partners or a history of a sexually transmitted disease. Since the assays are readily available and relatively inexpensive, and the intervention largely prevents disastrous consequences, the safest strategy is for all patients undergoing immunosuppressive chemotherapy to be tested for hepatitis B core antibodies.[14 15] Patients found to be positive for core antibodies should be tested for surface antigen and for hepatitis B virus DNA status. People positive for hepatitis B surface antigen are at risk of fulminant hepatitis and should receive prophylaxis with lamivudine or an equivalent antiviral agent throughout treatment and for at least six months afterwards.[16] Patients positive for hepatitis B core antibodies but negative for hepatitis B surface antigen are at lower risk of reactivation, and a reasonable approach may be to await hepatitis B virus

Managing the risk of hepatitis B virus reactivation in patients receiving chemotherapy or immunotherapy

- In treatment of haematological and solid organ malignancy, hepatitis B recurrence can be fatal; use of immunosuppressive agents such as rituximab has also been associated with recurrence
  - -Viral reactivation can occur during or after treatment, and can largely be avoided with appropriate antiviral therapy
- All patients from high risk areas or with a history of high risk practices should be screened for hepatitis B before treatment
  - -Since any patient with a history of multiple sexual partners or sexually transmitted disease is defined as high risk, where possible it is safest to screen ALL patients
- Screening consists initially of determining hepatitis B core antibody status; patients positive for antibodies should be tested for hepatitis B surface antigen and levels of hepatitis B virus DNA
  - -Patients positive for hepatitis B surface antigen or DNA should receive lamivudine (or an equivalent agent) for the duration of their treatment and for at least six months afterwards
  - -Patients positive for hepatitis B core antibodies but negative for DNA should be monitored throughout and for at least six months after treatment by determining alanine transaminase and hepatitis B virus DNA levels
  - -Patients negative for hepatitis B core antibodies, or positive with a low surface antibody titre, should be offered vaccination against hepatitis B
- Hepatology consultation is advised for patients receiving prophylaxis or if interpretation of results is in doubt

DNA status in such patients. If they are positive for hepatitis B virus DNA, prophylactic lamivudine is prudent; those who are negative can be observed, with liver function monitored regularly and hepatitis B virus DNA levels at least monthly, as the titre of hepatitis B virus DNA starts to rise several weeks before a rise in liver enzymes becomes evident.[17] If there is a significant rise in either, antiviral therapy can be considered (box).

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10-MINUTE CONSULTATION

Chronic diarrhoea in a teenager

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This is part of a series of occasional articles on common problems in primary care. The *BMJ* welcomes contributions from general practitioners to the series

A 14 year old boy has a three year history of loose stools with intermittent abdominal pain. He is the smallest boy in his class.

What issues you should cover

What is the risk of underlying disease?

Chronic diarrhoea is defined as >3 loose or liquid stools a day, lasting more than three weeks. This may indicate an underlying non-infectious diagnosis, such as coeliac or inflammatory bowel disease, particularly if accompanied by delayed development or growth retardation. A key consideration is whether the boy's gastrointestinal symptoms and growth are pathological, and if so, whether they are related. Since issues around puberty and diarrhoea may be embarrassing for him, it may be better to conduct the consultation without his mother present.

History

Ask about consistency and frequency of diarrhoea. Paleness and foul smell with residual fat droplets in the toilet water suggests fat malabsorption. Blood in the stool may occur with infection or inflammatory bowel disease. Mucus and pus are more likely with inflammatory bowel disease. A history of foreign travel in developing countries may indicate bacterial or amoebic dysentery.

- Pain associated with the diarrhoea suggests inflammatory bowel disease or irritable bowel disease. Irritable bowel syndrome can develop alone or after viral gastroenteritis, with persistent abdominal pain and change in bowel habit. There is no weight loss with irritable bowel syndrome
- Has diet changed and do any foodstuffs produce diarrhoea? Excessive fibre and misuse of laxatives will increase the frequency of stools. Ask specifically about change in appetite, including loss of appetite, energy levels, and weight
- A family history of gastrointestinal or autoimmune diseases, in particular coeliac disease, inflammatory bowel disease, and thyroid disease, may be important.

Important causes of chronic diarrhoea in children
Gastroenteritis
Viral (rotavirus, norovirus, adenovirus, etc)—usually acute diarrhoea
Bacterial (*Campylobacter*, *Shigella*, *Salmonella*, *E coli* spp, etc)—usually acute diarrhoea
Parasites (*Giardia lamblia*, *Cryptosporidium* spp, etc)
Inflammatory bowel disease
Crohn's disease
Ulcerative colitis
Coeliac disease (with or without selective IgA deficiency or hypogammaglobulinaemia)
Drugs (laxatives, for example)
Cows' milk or soya protein enteropathy; disaccharidase deficiencies
Endocrine causes (thyrotoxicosis, for example)
Irritable bowel syndrome

Examination

- Look for dehydration, weight loss (muscle wasting or loss of subcutaneous fat), rashes—for example, erythema nodosum (Crohn's disease), and thyroid enlargement
- Examine the mouth for features of gastrointestinal disease, including angular cheilitis of iron deficiency anaemia and mucosal ulcers in coeliac disease and inflammatory bowel disease. Palpate the abdomen for tenderness or masses—for example, a right iliac fossa mass suggests Crohn's disease. Rectal examination is unnecessary but perianal inspection is essential—for example, perianal skin tags suggest Crohn's disease
- Plot height, weight, and pubertal staging on a growth chart, with earlier observations if available to determine if the patient is "falling off his centiles." Tanner staging for pubertal development is essential to detect pubertal delay. Crohn's disease may present with growth failure with few or no gastrointestinal symptoms. Constitutional delay is delayed puberty and growth without underlying disease

What you should do

- Initially, send stool for microscopy and culture for *Giardia lamblia*, *Salmonella, Shigella* and *Campylobacter* and for virology (box) and reducing substances for disaccharidase deficiency. Viral carriage can persist in immune suppression.
- Blood tests indicating malabsorption include full blood count, serum B12, folate, ferritin, liver function tests, albumin, inflammatory markers (erythrocyte sedimentation rate, C reactive protein, and platelet count), coeliac serology (with total IgA to exclude IgA deficiency) including endomysial or tissue transglutaminase antibodies that are highly specific for coeliac disease, and thyroid function. Raised total IgE indicates atopy, and radioallergosorbent tests (RASTs) for cows' and soya milk protein will identify the most common food allergens
- Recommend a low fibre diet. Wheat should not be restricted until tests for coeliac disease have been performed. Irritable bowel syndrome is a diagnosis of exclusion, and in this age group the Rome criteria for irritable bowel syndrome in adults can be used (http://ibdcrohns.about.com/cs/ibs/a/romecriteria.htm)
- Refer to a paediatric gastroenterologist if there is evidence of weight loss or growth retardation or if investigations reveal malabsorption, inflammation (raised erythrocyte sedimentation rate, C reactive protein, or platelet count) or positive coeliac antibodies
- Review regularly and monitor height and weight. Measurements taken six months apart are required to confirm growth retardation.

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USEFUL READING

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Information for patients:

Coeliac UK (www.coeliac.co.uk)—website for people with coeliac disease

Crohn's in Childhood Research Association (www.cicra.org)—support for children and young adults with inflammatory bowel disease

Endpiece
Fear of unemployment

Running through the hospitals of Britain today, invisibly linking doctors from one end of the country to the other, are three closely woven strands of frustration, fear, and despondency. Highly skilled though these men and women often are, they nevertheless go about their daily tasks in the bitter and frustrating knowledge that the future holds little promise for them in their chosen field of work. Fear of unemployment—and worse, of being unemployable—haunts them as much as it did any factory worker of the depression years. Despondency grows steadily year by year while the authorities continue to discuss their problems without apparently getting any closer to a solution.

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Submitted by David Hamilton, *Glasgow, retired consultant surgeon*

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