damage, those consequences will also be permanent. This is faulty logic—on the same basis no one would treat the epilepsy of children with cerebral palsy. Nevertheless, gloomy prognoses can be self-fulfilling if they stand in the way of initiating treatment. In many cases, children with hemiplegia and psychiatric problems are more responsive to treatment than children whose psychiatric problems stem from chronic psychosocial adversity. With highly motivated parents, a relatively small amount of informed advice can go a long way. In addition, specific disorders seem likely to respond to the same sorts of specific treatments—such as medication, behavioral therapy, and family therapy—used for other children with comparable psychiatric presentations.

Problems with peer relationships

Even though most children with hemiplegia are integrated in mainstream schools, problems getting on with other children are common. While most hemiplegic children do have some playmates, they have fewer close reciprocal friendships than most of their classmates, and they are substantially more likely to be teased or bullied. Prejudice may play a part, but there is increasing evidence for constitutional impairments in social understanding. There is growing interest in children’s abilities to understand other people’s perspectives, beliefs and intentions; the development of these abilities appears to be delayed in children with hemiplegia, contributing to their emotional and social immaturity.

Practical help

As a child health professional, what can you do to prevent and treat the psychological complications of your patients with hemiplegia? First, put the issue on the agenda: show through your attitude and questions that you are concerned about psychological as well as physical development. Parents are often greatly reassured to find that their child’s problems are common consequences of hemiplegia; the energy previously locked up in self-blame can then be diverted into more profitable channels. By telling the family about Hemi-Help, the nationwide parents’ support group, you can further reduce the family’s sense of isolation and powerlessness by providing them with access to regular newsletters, meetings, and advice (inquiries and help line: 0181-672-3179). Ensuring appropriate educational provision may also depend, at least in part, on health service input. Adequate neuropsychological assessment helps delineate the child’s needs and the best ways of meeting those needs; in some areas this assessment is most appropriately carried out by clinical rather than educational psychologists. Reassessment is advisable if a child is running into psychological problems, particularly if the child is becoming hopelessly demoralised by lack of success in all areas of school life. Finally, if the child’s psychological problems seem too complicated to assess or treat locally, refer on.

ROBERT GOODMAN

Department of Child and Adolescent Psychiatry,
Institute of Psychiatry,
De Crespigny Park,
London SE5 8AF

Preventative strategies on meningococcal disease

The dramatic occurrence of meningococcal disease challenges doctors and attracts intense public interest. It is treatable and preventable, so every death raises the question: Could this have been avoided? Last winter’s experience of clusters in schools was the latest round in a question: Could this have been avoided? Last winter’s treatable and preventable, so every death raises the prevention strategy depends on a reliable count of cases. Notifications are an enduring source of statistics, but there has been under-reporting in the past. In the last three years there have been fewer laboratory isolates than notifications, which produced problems in the autumn of 1995 when the public were aware of clusters and an increase in notifications, which was not substantiated by laboratory reports. The only reliable way to enhance surveillance is to combine sources of data. The laboratory reporting system is accurate for diagnosis, but is an underestimate of incidence since the widespread use of preadmission penicillin, which reduces the proportion of laboratory confirmed cases. Caution in performing lumbar punctures on children may also reduce the numbers confirmed by microbiology. The gap can be reduced by serology, culturing throat swabs from patients on admission and by antigen detection (for example the polymerase chain reaction).

Microscopy of fluid from purpuric lesions is a technique useful in understanding social impairment in children with hemiplegic cerebral palsy. (Clin Psy D thesis.) Norwich: University of East Anglia, 1996.


downloaded from group.bmj.com on June 15, 2017 - Published by group.bmj.com
Meningococci are all members of one species, Neisseria meningitidis, and can be divided into groups according to polysaccharides in the cell wall: serogroups A, B, C, W135, and Y are well recognised causes of human disease. Group A meningococci are the cause of extensive epidemics in Africa which have spread to the Middle East and the Indian subcontinent. Group B meningococci are the commonest cause of disease in the Americas, Europe, and Australia; the group is further subdivided by type and subtype antigens. Group C meningococci have caused about a third of the cases in Europe and North America in recent years, including some outbreaks among teenagers and young adults.

**Risk factors and special risk groups**

Besides the obvious factors of contact with someone carrying meningococci and a lack of immunity to the strains that are circulating in the community, other risk factors for meningitis are imprecise. Pre-existing respiratory disease, for example influenza, is a factor that could explain the seasonal variation. Overcrowding has been a factor known since the first world war. Smoking in household members increases the carriage rate and the risk of disease. International travel to areas where epidemics are occurring has increased the risk and spread of infection due to group A meningococci. Smoking and overcrowding are worth prevention strategies in their own right. Hygiene and vaccines have a part in the control of other factors, like influenza A and travel to epidemic areas, although the scale at which meningococcal A disease is epidemic and occasional for the control of meningococcal C outbreaks, as described below under secondary prevention.

The development of group B vaccines has been more difficult. The capsule of the group B meningococci is poorly immunogenic and the polysaccharide is antigenically similar to a human epitope. Linking the native group B polysaccharide to tetanus toxoid does not improve immunogenicity. A non-capsular vaccine that has shown some promise against group B disease is based on class 1 outer membrane proteins. Moderate efficacy was demonstrated in older children in Cuba, Brazil, and Norway, but again protection was not given to children under 2 years of age. If the development of the Norwegian and other outer membrane protein group B vaccines is successful, the decisions on whom to vaccinate will be as difficult as for the conjugated meningococcal C vaccine.

**Secondary prevention**

The prevention of secondary cases attracts most public interest. As a strategy for prevention, it has serious limitations. As most of the cases are sporadic, secondary prevention can make a relatively small reduction to the incidence and mortality of meningococcal infection. The principle of secondary prevention is based on the observation that a second case occurs within a month in the same household or family of 1% of primary cases. The risk is highest in the first week after the first case, but because this risk extends beyond the incubation period, it is thought that secondary cases are often due to transmission within families after the primary case has been admitted to hospital. Treatment of the family is supposed to reduce this risk. It is also recommended that patients with meningococcal disease have chemoprophylaxis before going home. Doctors and the public often misunderstand that chemoprophylaxis is intended to stop spread from carriers, and not to treat early infection. Carriers are likely to be immune, and the most commonly used antibiotic, rifampicin, is not reliable for treating incubating infections. Penicillin should be used for treating patients with co-primary infection (that is those with active disease at the time when the index case presents). Such cases are uncommon. The clinical features of early infection maybe unimpressive and vigilance is required. Some have taken this further and recommend a combination of chemoprophylaxis and early treatment for high risk contacts. There is no good evidence of the effectiveness of this approach, and guidelines should be as simple as possible.

It is clear from the scale of the risk that one would have to do a very large trial to test the efficacy of secondary prevention. It is difficult to see how a controlled trial could now be conducted, but the question may become pressing. Uncontrolled use of antibiotics promotes antibiotic resistance. If rifampicin resistance should increase, following the

---

**Annotations**

- Haemophilus influenzae type b vaccine has shown how successful this strategy might be. If the clinical trials have the expected result of showing that conjugated meningococcal C vaccine is safe and effective, there will be difficult strategic decisions to make. Assuming that conjugated meningococcal C vaccine will be included in the routine infant schedule, should there be a large 'catch-up' programme to vaccinate older children, and even university students? Should we ask for a combined conjugated meningococcal A and C vaccine, which would have more international appeal, but perhaps a higher cost? Will there be enough vaccine, the money and the staff available in the school health services and general practices to give the millions of doses that will be demanded? The vaccine that is now available in Britain is a mixture of unconjugated A and C polysaccharides. It is used for travellers abroad to countries where meningococcal A disease is epidemic and occasional for the control of meningococcal C outbreaks, as described below under secondary prevention.

The prevention of secondary cases attracts most public interest. As a strategy for prevention, it has serious limitations. As most of the cases are sporadic, secondary prevention can make a relatively small reduction to the incidence and mortality of meningococcal infection. The principle of secondary prevention is based on the observation that a second case occurs within a month in the same household or family of 1% of primary cases. The risk is highest in the first week after the first case, but because this risk extends beyond the incubation period, it is thought that secondary cases are often due to transmission within families after the primary case has been admitted to hospital. Treatment of the family is supposed to reduce this risk. It is also recommended that patients with meningococcal disease have chemoprophylaxis before going home. Doctors and the public often misunderstand that chemoprophylaxis is intended to stop spread from carriers, and not to treat early infection. Carriers are likely to be immune, and the most commonly used antibiotic, rifampicin, is not reliable for treating incubating infections. Penicillin should be used for treating patients with co-primary infection (that is those with active disease at the time when the index case presents). Such cases are uncommon. The clinical features of early infection maybe unimpressive and vigilance is required. Some have taken this further and recommend a combination of chemoprophylaxis and early treatment for high risk contacts. There is no good evidence of the effectiveness of this approach, and guidelines should be as simple as possible.

It is clear from the scale of the risk that one would have to do a very large trial to test the efficacy of secondary prevention. It is difficult to see how a controlled trial could now be conducted, but the question may become pressing. Uncontrolled use of antibiotics promotes antibiotic resistance. If rifampicin resistance should increase, following the
pattern that occurred with sulphonamides, then trials may be necessary to show that drugs that are unlicensed for paediatric use, like ciprofloxacin, have benefits that outweigh the risks. Although the public demand that something should be done is understandable, mass prophylaxis is difficult to do well and increases expectations for similar action on the next occasion. There is substantial doubt about the value of wider use of prophylactic antibiotics. For example, the guidance that nursery school contacts should have chemoprophylaxis has been changed to advice that school and nursery contacts should be considered for prophylaxis only when there are two or more cases (a ‘cluster’) in a school or similar community within four weeks. If the second case is not the same serogroup or type, or if the diagnosis in one of the cases is unconfirmed, the cases should be treated as sporadic cases and not as a cluster. It must be emphasised that chemoprophylaxis is not foolproof and may fail because meningococci are not eradicated or carriers are not recognised. Failure to prevent secondary cases in families and a nursery have been recorded. Therefore chemoprophylaxis must be accompanied by clear advice on the signs of meningococcal disease, preferably using the leaflets produced by the National Meningitis Trust and the Meningitis Research Fund. When a single case occurs in a school or nursery, the other parents should be told about the diagnosis and the signs of meningitis and septicaemia, usually by a letter taken home at the end of school.

Tertiary prevention

Tertiary prevention refers to the prevention of death or complications in children with clinical infection. It is recommended in Britain that benzylpenicillin (or chloramphenicol if there is an allergy to penicillin) is given as soon as meningococcal infection is suspected, preferably before admission. The National Meningitis Trust and others have campaigned to teach parents and doctors to recognise meningococcal infection early. The emphasis on early diagnosis may heighten public anxiety. Publicity about selected cases of meningitis may imply that some places have a high risk, and miss the point that most cases are sporadic. The leaflets and stories in the press have alerted parents in some cases, but the case fatality rate has been slow to fall from 11%. Publicity can mislead by emphasising meningitis and neglecting the rash (haemorrhagic or maculopapular) of septicaemia. Less well described is the mildness of some cases who have symptoms and a rash untreated with antibiotics for several days and still make an uncomplicated recovery. If parents and doctors are to act quickly in order to prevent death and neurological damage, they must recognise that meningococcal disease can start as a mild non-specific febrile illness, without a diagnostic rash or the classical meningeval signs.

Conclusion

The necessity to ask the question ‘Could this be meningococcal disease?’ for all patients reveals the limitations of current meningococcal disease control. There is a real prospect of ‘bacterial meningitis’ vaccines for infants, using conjugate vaccines to cover H influenzae type b, group C meningococci, group A meningococci in epidemic areas, and the commoner types of pneumococci. After these vaccines are introduced, the unitidy strategies of secondary prevention will still be needed until there is an effective group B meningococcal vaccine. Only then will we realise the dream of meningococcal disease prevention.

RT MAYON-WHITE
PT HEATH

Oxfordshire Department of Public Health,
Oxfordshire Health Authority,
Old Road, Headington,
Oxford OX3 7LG

Preventative strategies on meningococcal disease

R T MAYON-WHITE and P T HEATH

Arch Dis Child 1997 76: 178-181
doi: 10.1136/adc.76.3.178

Updated information and services can be found at:
http://adc.bmj.com/content/76/3/178

These include:

References
This article cites 28 articles, 7 of which you can access for free at:
http://adc.bmj.com/content/76/3/178#BIBL

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/