good evidence that such immunity occurs and may reduce symptoms or even prevent colonisation.²⁵ The relation of serum concentrations of antibodies and immunity has yet to be established, though immunity may be mediated by IgG.²⁶ The value of epidemiological studies would be greatly enhanced if investigators documented the serological antibody status of cases and controls and if more reliable and convenient methods of identifying immunity were available.

The answer to the second question may lie in a combination of cross contamination and the low infectious dose in the susceptible. Some sources of the organism may have been identified, but other vehicles may be so numerous and each one in itself so uncommon that studies of sporadic cases fail to identify them. Or there may be an as yet unidentified source. More widely available typing schemes would help greatly not only in analytical epidemiological investigations but also in the environmental and human microbiological studies that are vital to tracing the possible routes by which campylobacter may move from the environment, through food and domestic animals, ultimately to cause illness in humans.

JOHN COWDEN

Consultant Epidemiologist,
Public Health Laboratory Service, London NW9 5EQ

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Reducing mortality from meningococcal disease

Give antibiotics before admission

Meningococcal disease remains an important cause of childhood mortality. Of 170 deaths last year from meningococcal infection in England and Wales, 110 were of children under 15. The case fatality rate of 5-10% has changed little in the past 30 years.¹ As two thirds of infections are due to group B strains—for which no vaccine is available²—the best prospect for reducing mortality in the short term is improved treatment.

The role of early antibiotic treatment in reducing mortality from bacterial meningitis has been recognised for many years.³ In 1988 the chief medical officer wrote to all doctors advising them to consider giving parenteral benzylpenicillin in all cases of suspected meningococcal disease before transfer to hospital,⁴ and this advice has subsequently been repeated several times.⁵⁻⁷

A survey carried out immediately after the chief medical officer's letter found that fewer than half of general practitioners carried parenteral penicillin in their emergency bags.⁸ Two papers in this week's journal show that the advice is still not being followed. Cartwright and colleagues found that despite regular reminders to general practitioners over many years the use of antibiotics in suspected cases of meningococcal disease before admission did not exceed 40% (p 143).⁹ In Strang and Pugh's study the rate was only 28% (p 141).¹⁰ In both studies mortality was lower in patients receiving antibiotics before admission than in those who did not receive such treatment, although the difference just failed to reach significance.

Good reasons exist, however, for believing that these

findings are clinically important. Fewer deaths were associated with antibiotics given before admission in all four study districts. The greatest reduction in mortality was observed in patients with the worst prognosis (those with a haemorrhagic rash or with disseminated intravascular coagulation). Finally, the findings are consistent with those of earlier studies.³

Why are general practitioners still reluctant to give antibiotics before admission? One reason may be fear of an anaphylactic reaction in patients with a history of allergy to penicillin. A history of such allergy is, however, usually unfounded¹¹ and is not a sufficient contraindication for a potentially life saving intervention. For patients with a proved history of hypersensitivity to penicillin chloramphenicol is a suitable alternative.

A second reason is that general practitioners have been taught not to give antibiotics before microbiological specimens have been obtained because this hinders the chances of obtaining a positive culture. Antibiotics usually render blood cultures and (to a lesser extent) cerebrospinal fluid samples sterile in meningococcal infections, although the rate of positive nasopharyngeal swabs in Cartwright and colleagues' study was unaffected by giving antibiotics before admission. Even if the diagnosis of meningococcal infection cannot be confirmed because an antibiotic has been given little harm is likely to result, and the consequences are likely to be insignificant compared with those of failing to initiate prompt treatment. Finally, concerns that giving antibiotic could result in release of harmful endotoxin and cytokines have proved unfounded.¹²

The case for early administration of parenteral antibiotics to patients with suspected meningococcal disease is now stronger than ever. General practitioners and casualty officers should treat such patients immediately with parenteral (preferably intravenous) benzylpenicillin. (The dose is 1200 mg for adults and children aged 10 and above, 500 mg for children aged 1-9, and 300 mg for children under 1.) Before reconstitution both penicillin and chloramphenicol are stable for at least two years in an emergency bag. Frequent reminders of this simple, cheap measure are needed to reinforce the message.

NORMAN BEGG

Consultant Epidemiologist,
PHLS Communicable Disease Surveillance Centre,
London NW9 5EQ

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Anhedonia: exclusion from the pleasure dome

A useful marker of biological depression

The concept of anhedonia, or loss of the experience of pleasure, has fallen in and out of psychiatric fashion over the past century. As psychiatrists are now recognising again the importance of symptoms, the concept of anhedonia is proving useful in identifying certain forms of depression—and their ability to respond to treatment.

In 1897 Ribot introduced the term anhedonia and accused psychologists of paying little attention to the study of the experience of pleasure.¹ Nevertheless, a review of psychiatric textbooks in the late nineteenth century revealed that the loss of the pleasure response was recognised as an early and important symptom of depressive illness.^{2,4} After the turn of the century psychiatric interest in anhedonia faded, and Jaspers's influential text does not mention it except as an aspect of the more severe, pervasive loss of all emotional response.⁵ Attention then focused on depressed mood as the pathognomonic feature of depressive illness. For instance, the *International Classification of Diseases, 9th revision*, still in use today, does not mention anhedonia in its definition of the depressive phase of manic-depressive psychosis but defines the disorder in terms of "a widespread depressed mood of gloom and wretchedness with some degree of anxiety."⁶

In the 1960s interest in anhedonia resurfaced. Meehl considered that low hedonic capacity, or joylessness, was a heritable trait predisposing to the development of schizophrenia and depression.⁷ For most psychiatrists, however, it was Klein's concept of endogenomorphic depression which revived interest in the notion.⁸ In the search for a reliable clinical marker which would distinguish a state responsive to antidepressant drugs from all the other states of misery variously called depression, depressed mood seemed too vague a concept, retardation difficult to assess, and the so called "biological" symptoms of depression such as appetite and sleep disturbance too common in most somatic illness.⁹

In devising a brief self assessment questionnaire to screen for depressive illness in patients suffering from physical illness—and who would respond to antidepressants—we constructed a depression subscale that concentrated on the experience of loss of pleasure.¹⁰

The loss of the ability to experience pleasure in what was once enjoyed is a fairly distinct experience. The American *Diagnostic and Statistical Manual of Mental Disorders* recognised this in its 1980 revision, when the concept of anhedonia was promoted to the position of one of the two pathognomonic

features of major depressive disorder. Klein's definition: "a sharp, unreactive, pervasive impairment of the capacity to experience pleasure, or to respond affectively, to the anticipation of pleasure" was slightly modified to "a loss of interest or pleasure in all or almost all usual activities and pastimes." Moreover, for the subtype of major depression, for which the term melancholia was resurrected, the anhedonic experience became essential to the definition. Interestingly, a construct of "somatic" depression appears in the third draft for the 10th revision of the *International Classification of Diseases*, which moves away from the "pervasive gloom and wretchedness" of the present version and produces a symptom based definition dominated by anhedonia.

Research in psychiatry has traditionally proceeded on a categorical basis. Attention is now turning to the study of symptoms.¹¹⁻¹³ Disturbances in neurobiological systems cause particular symptoms, and successful therapeutics requires clinicians to pay attention to the symptom as well as to the categorical diagnosis. Thus the anhedonic state which so often follows recovery from the florid phase of an acute schizophrenic illness may well respond to antidepressant drugs. We need now to work on defining symptoms better and on devising means of assessing them. For instance, the present tendency to combine "loss of interest" with "lack of pleasure" may be misleading. A gardener may retain an interest in the flowers he grows but no longer experience pleasure at their sight or their smell.¹⁴

PHILIP SNAITH

Consultant Psychiatrist,
Senior Lecturer in Psychiatry,
St James's University Hospital,
Leeds LS9 7TF

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