should be more open.[8] But most managers would want new and explicit criteria, with more emphasis on efficiency and productivity and less on academic or medicopolitical merit. Furthermore, they would expect to have a greater hand in setting the criteria and in the review procedure. The payments would almost certainly have to be one off, with no expectation of renewal, thereby raising serious questions about the costs of administering the system. Forward thinking managers may also be considering the merits of rewarding teams instead of individual doctors and including other professionals in the teams. Where teams are stable this is feasible; where team members work for several different teams administration would be difficult. Finally, the reward offered need not necessarily be money: time off in lieu might be an attractive alternative, especially where staff can earn at far higher rates outside the NHS. Is such major reform feasible or will a separate system of performance related pay be necessary (instead of or as well as distinction awards)? The debate is about to begin.

Recognising and rewarding differences in contribution, where they can be fairly shown, has much to recommend it. But where there is doubt about the fairness, performance related pay may be divisive and demotivating. Even if it is demonstrably fair, it requires considerable skill to evaluate performance accurately, courage to advise poor performers that they will be penalised, and experience to advise them how to improve poor performance. Unless such conditions can be met performance related pay for doctors may be an expensive and unproductive mistake.

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Campylobacter: epidemiological paradoxes

The vehicles for most cases of infection remain unknown

Campylobacter has been the commonest reported bacterial cause of diarrhoea in Britain since 1981.[1] *Campylobacter jejuni* has been estimated to account for over 95% of human campylobacter infections in England and *C coli* for nearly 5%.[2] These two species share many clinical and epidemiological characteristics.

The Public Health Laboratory Service Communicable Disease Surveillance Centre first recorded cases of campylobacter infection in 1977 after Skirrow's description of a selective stool culture medium for *C jejuni*.[3] The numbers of cases reported rose every year to reach over 34 500 in 1990 but fell in 1991 to about 32 600.[4] About 10% of people found to be infected have acquired the infection abroad. The early rise in reports throughout the 1980s probably resulted from more frequent identification rather than a true increase in incidence. Nevertheless, campylobacter infection is undoubtedly an important public health problem in Britain and elsewhere.[5]

Fewer than 10 deaths in people with campylobacter infection have been reported in England and Wales since 1981 (Public Health Laboratory Service, Communicable Disease Surveillance Centre, unpublished data), and most of these were in patients with predisposing conditions; but the disease causes considerable morbidity and may occasionally have serious sequelae, including the Guillain-Barré syndrome.[6] Campylobacter infection is also expensive. A study in 1986 estimated that each identified case in England cost £587—a total of £14 million in that year.[7] Not all cases are identified, so the total cost to the community is probably much greater.

Campylobacters are common in sewage and have been cultured from untreated water.[8] They are found frequently in the intestines of animals of many species; on raw meats, especially poultry meat[9]; and in raw milk.[10] Despite its widespread distribution the organism does not multiply below 30°C[11] and is therefore unlikely to grow on food at room temperature.

Many vehicles of infection have been identified in outbreaks, both from epidemiological and microbiological evidence. These include untreated water[12] and water from storage tanks, which may have been contaminated after treatment[13]; raw milk[10] and milk that may have been inadequately pasteurised[14]; and undercooked meats, including poultry meat.[15] Cross contamination also occurs, allowing foods as diverse as salads and cake icing to be vehicles.[16] Asymptomatic excretion of campylobacter is unusual,[17] and infected food handlers do not seem to present a risk.

Indeed an odd feature of campylobacter diarrhoea is that although isolated cases are common, outbreaks are rarely reported. Fewer than 1% of the cases reported to the Communicable Disease Surveillance Centre in 1991 were part of known outbreaks. Furthermore, even though some causes of outbreaks have been shown to be responsible for some sporadic infections as well—for example, consumption of raw milk[18] and undercooked chicken[19] and handling of raw chicken[20]—the vehicles are not necessarily the same. Other risk factors more specific to sporadic infections have been identified and include contact with pets, particularly puppies with diarrhoea,[21] and, in some parts of Britain in the spring, drinking milk that has been delivered to the door and has been pecked by birds such as magpies and jackdaws.[22] This last finding may explain, at least partially, why reports rise in the spring—one of the enigmas of campylobacter infection. It does not, however, explain the autumn rise. Nor is there any known explanation for most of the cases which occur throughout the year.

Person to person spread seems to be unusual. Family clusters are seldom seen, and secondary transmission after point source outbreaks is also rare. This is puzzling—the infective dose may be as small as 500 organisms,[23] and diarrhoeic faeces can contain as many as 10^6-10^9 organisms per gram.[24]

Why, then, are outbreaks, clusters, and secondary infections so infrequent, and what are the routes of infection in the many patients who do not seem to have been exposed to known risk factors? The first question may be answered by the widespread existence of immunity in the population resulting from repeated exposure to the organism. There is

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.

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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 available[2]—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 unfounded[11] 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 nasopharygeal 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]