Clinical audit in primary health care: towards quality assurance

Let 100 flowers blossom seems to be the message of the Clinical Outcomes Group

What should replace medical audit advisory groups in just over a year's time? New developments, such as the emergence of commissions, and new information on the results of audit, due out soon from the National Audit Office and other bodies, will have to be taken into account in the decision.

Last month's report from the working group of the Clinical Outcomes Group clearly defines the agenda for the debate about the future of clinical audit in primary health care.¹ The group wants every practice unit (whether in general or dental practice, community health services, or community pharmacy) to have an integral quality assurance system. Such systems would use practice development plans, guidelines, protocols, clinical audit, and other tools and would relate to external quality assurance mechanisms such as accreditation schemes.

To attain this goal the group recommends two steps to improve the effectiveness of the separate organisations currently supporting quality assurance in primary health care. Firstly, the body that evolves from or replaces the medical audit advisory groups must encompass all sectors of primary health care, including community health services. Secondly, developments in audit and service should be linked through family health services authorities. This would avoid duplication and concentrate resources more effectively.

The working group considers that nationally prescribed structures are no longer necessary—instead, a variety of arrangements, tailored to suit local circumstances, should support audit and service development. One option would be for the health commission to provide the support; another would be for the health commission to coordinate its own service development activities with those of the community trust and clinical audit. But, as failure of coordination is one of the main weaknesses of the present arrangements, this approach may be overoptimistic.

A third option is to contract support for audit and service development to a separate agency that might, for example, be developed through substantial restructuring of the medical audit advisory groups or be based within existing units such as community trusts. The functions, accountability, and

funding of such an agency would be agreed in an annual contract. The agency would not, however, monitor performance—the working group does not want audit to get mixed up with monitoring of clinical contracts. The working group also highlights the need for better links between audit and continuing education, a centrally managed research and development programme for quality assurance in primary health care, and a consistent approach towards allocating resources to all professional groups.

Promoters of clinical audit in primary health care will welcome much in this report. Unifying the functions of audit and service development into a single system for all professional groups in primary health care would be an important step, given the longstanding divisions within the management of the service. A policy that enables clinical audit to develop into comprehensive quality assurance systems within practices offers an important opportunity to improve care.

For the new proposals to succeed, managers and health professionals will have to share the same views about clinical audit. The confidence of health care staff in the system is critical if they are to participate without reservation and if future developments are to benefit from their experiences of audit. At the same time health commissions that are contracting for audit need a voice in the selection of topics and enough information about the findings to convince them that their investment is worth while. The coming debate about both the working group report and the future of clinical audit in primary health care will be largely concerned with achieving a balance between the potentially competing demands of health professionals and managers.

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Injuries to child pedestrians

The key to prevention is a change in transport policy

Injury to child pedestrians is a leading cause of death and an important cause of severe brain injury. In 1991 in England and Wales 185 child pedestrians were killed and 3965 were seriously injured in road accidents.¹

Injuries to child pedestrians have held the rank of "captain of the kings of death" for the past 20 years, yet only recently have epidemiological methods been applied to their prevention. In the past preventive strategies were based on assumptions about causation drawn from case series, but such an approach was bound to fail. Some crucial questions need to be addressed. Firstly, why do rates of injury among child pedestrians change over time, and why do some populations have higher rates than others? Secondly, within populations,

why are some children injured and not others?² Children are exposed to the risk of pedestrian injury only while walking, so measures of frequency should take account of exposure. For example, child pedestrians are rarely injured on motorways, but that does not mean that motorways are safe places to walk. Any judgment about the safety of walking must take pedestrian exposure into account.

Denominators for the incidence of many diseases are conveniently measured at each census, but such figures cannot be used to provide denominators for measures of pedestrian safety.³ Some recent studies of pedestrian exposure have provided important insights.⁴ The results suggest that the most likely explanation for the fall in death rates in child

pedestrians over the past two decades is a reduction in the numbers of pedestrians.⁵ The rapid increase in the volume of traffic over this period makes it unlikely that walking has become any safer. The death rates have fallen because children walk less than they used to. Exposure also helps to explain the high injury rates among poor children. Children from poor families and children from families without a car walk more than affluent children.⁶ Whether Sweden's low death rate in child pedestrians is due to the importance given to environmental approaches to prevention or to differences in exposure has yet to be answered.

The reasons why some children are injured and not others have been investigated in cohort and case-control studies. The most striking finding in these studies is that hardly any personal characteristics differentiate between children who are and are not injured.⁷ Studies that have examined the contribution of the traffic environment have shown that children from neighbourhoods with high traffic volumes and speeds are at greatly increased risk.^{8,9}

This distinction between personal and environmental factors is also seen in intervention studies. Programmes to educate pedestrians attempt to teach children safer ways to cross roads. Such programmes may be evaluated by assessing how much knowledge about traffic has improved, by studying the way children cross roads, or by looking at any reduction in injuries. In practice, several evaluation studies have shown improvements in knowledge, much less often has road crossing behaviour been seen to improve, and few studies in any countries have shown reduced injury rates. Perhaps this should come as no surprise. The physical and developmental abilities of young children have long since been outstripped by the complexity of the traffic environment.¹⁰ The disappointing results of education of pedestrians should be contrasted with the established track record of efficacy of area wide traffic management schemes.¹¹ Targeting schemes on areas with the highest injury rates makes them more cost-effective and, because these areas are also the most disadvantaged, would go some way to addressing socioeconomic differentials in injury rates.

The most radical strategy has yet to be tested. This entails intervention at the level of land use and transport policy to reduce the volume of traffic throughout the road network by encouraging a shift from private cars to bus, rail, or air travel

for long journeys and cycling and walking for short journeys.¹² The potential of this approach was shown during the energy crisis, when reductions in traffic volume were accompanied by a sharp decline in death rates in child pedestrians.¹³ There would be other benefits, too. Twenty times more passenger deaths, per kilometre, occur with car travel than with rail or air travel, and seven times more deaths than with bus travel. Less car travel would also mean less urban air pollution and a more pleasant environment.¹¹

For most health professionals transport policy is unfamiliar. Many seem to believe it is best left to the experts. Yet policy is about politics; and for too long transport policy has been dictated by the powerful vested interests of the road lobbies. Unless child health professionals enter the fray the interests of children are likely to come low on the transport agenda.

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A one day conference, organised jointly by the BMJ Publishing Group and *Injury Prevention*, a new quarterly journal that will be launched at the conference, will be held in London on 9 March. Details from BMA Conference Unit (tel 0171 383 6605).

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Hepatitis E

Large outbreaks, faecal-oral transmission, and mainly young adults affected

Hepatitis E virus is the latest addition to the list of viruses causing hepatitis. Recently Reyes's group cloned and sequenced the virus¹ previously referred to as non-A or epidemic hepatitis because of its propensity to cause large outbreaks.²

A faecally transmitted form of viral hepatitis that was distinct from hepatitis A had long been suspected, and its existence was confirmed in the late 1970s when specific serological tests for hepatitis A virus failed to diagnose many cases of "infectious hepatitis."³ Virus-like particles were observed in stool samples from infected patients, and the infection was transmissible to experimental animals.⁴ Lack of supply of antigen hampered the development of convenient serological assays until the viral genome was cloned by Reyes's group.

Only one type of hepatitis E virus exists, although several strains, including those derived from Mexican and Burmese isolates, have been recognised. Recombinant antigens representing various regions of these isolates have been used in the development of diagnostic assays.⁵ These assays have already been used to establish that hepatitis E virus causes waterborne outbreaks of hepatitis⁶ and also a substantial proportion of sporadic cases in endemic areas.⁷

Infection with hepatitis E virus closely resembles that with hepatitis A virus, both in its pattern of transmission and because it usually causes an acute, self limiting illness with no chronic sequelae. But important differences exist between the two infections. In particular, hepatitis E affects mainly young adults and seems to be responsible for high mortality in pregnant women, especially when it occurs in the third