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The health of travellers

A recent report from Kent suggests that travellers (nomadic people who are not necessarily of Romany origin) have serious health problems.¹ In particular, their perinatal mortality rate is a fifth higher than the average national rate and their infant mortality rate 40% higher. The conditions in which they live are often very poor, and the question arises whether health authorities should take some specific action.

Counting travellers is difficult, but the Department of the Environment found 9600 traveller caravans with an estimated total population of between 30 000 and 50 000 in July 1984.² Travellers are concentrated in Kent and East Anglia, where temporary seasonal agricultural employment is most available.

Evidence on the health of travellers was thin until the recent study from the University of Kent's Health Services Research Unit,¹ although a study by the Save the Children Fund in 1983 described a large population of travellers in East Anglia as "disadvantaged in health and health care."³ In the Kent study health visitors questioned mothers with children aged under 15 on all traveller sites in Kent, and the degree of cooperation was high. Many families lacked running water, lavatories, and electricity. A third of families did not have access to a lavatory on site, whereas in the 1981 national census 4% of all British households lacked or shared an inside lavatory. In 1982 7% of social class V households lacked a bath and shower, but 14% of traveller families in the study not only did not have a bath or shower but also lacked access to running water on site. Conditions on private

caravan sites were least primitive, and those on illegal sites were the worst. Conditions on some local authority sites were as bad as on illegal sites.

The most important health findings of the study relate to mother and child health. The average number of children was 3.1 in each family compared with the national average of under two. Some 16% of mothers interviewed had had five or more children compared with less than 2% in the population as a whole.⁴ The women did, however, have access to reliable contraceptive methods. As well as perinatal and infant mortality rates being higher than the national average, the prevalence of low birthweight babies (less than 2500 g) was about double the national rate.⁵ Prolonged breast feeding was uncommon. Child surveillance was substandard, and rates of child immunisation were only about a third of the national average. Sadly the study did not include a settled control group matched for social class. The effects of social class thus cannot be disentangled from the effects of a "nomadic habit of life."

What should be done in response to these findings? There is clearly a need for local authorities to provide more and better appointed sites for traveller families and for district health authorities to take action to improve standards of child surveillance and childhood immunisation. The poor reproductive performance of traveller women also needs attention, but as we do not understand the reasons for this poor performance health authorities can do little except perhaps promote research. To improve standards of child surveillance and immunisation rates traveller families in each district should first be identified. Services might then be taken to sites. Travellers may not use traditional clinic or general practitioner based services. Responsibility for improving the health care of traveller families must be allocated to a named individual—a health visitor might be the best person. Some health authorities have already done this.

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Urinary incontinence after stroke

Urinary incontinence after stroke is common, uncomfortable, and undignified and often constrains rehabilitation and discharge. It carries a bad prognosis for rehabilitation^{1,2} and may be of more specific prognostic importance than a history of depression of consciousness.³ Although detrusor instability, with consequent urge incontinence, is associated with certain cerebral lesions,⁴ other more peripheral or, indeed, external factors may be important. A mobile alert patient fumbling left handed with his fly buttons after 60 years of dexterity and continence may wet himself. A more dependent patient who needs help to urinate into a bedpan or bottle

but who cannot communicate to summon it will also be incontinent—perhaps despite awareness of a full bladder and reasonable sphincter control.

Urinary incontinence after stroke, an unglamorous complication of a mundane disease, does not routinely attract much attention from doctors. Management may often be left to nursing staff prepared to tackle sympathetically the component problems of communication, dependency, and intimate personal care. Doctors should probably be more interested, and a recent prospective study from a group of geriatricians in New Zealand gives grounds for optimism.⁵ Of 151 stroke patients who required admission to hospital 17% had been previously incontinent, and incontinence rates among survivors were 60%, 42%, and 29% at one, four, and 12 weeks respectively. In patients with moderate or severe persistent incontinence the commonest cystometric finding was detrusor instability.

Urinary incontinence at four weeks was associated with mental impairment, earlier moderate or severe motor deficit, and current dependency in mobility. Early dysphasia in those with right hemiparesis was associated with incontinence, but sensory impairment in those with left hemiparesis did not appear to make incontinence more likely. At no time were more than a quarter of the patients catheterised (and those mainly in the early weeks), and only 9% required catheters 12 weeks after their strokes. Continence might be expected to go on improving after three months. Brocklehurst *et al* reported 85% urinary continence in 92 patients one year after a stroke. At two and three years' follow up incontinence rates had increased again, but at four years they had fallen once more to 14%—a figure comparable to that for the elderly population at large.

Continence is a complex feat of awareness, control, mobility, and dexterity and is vulnerable at many points to the direct or indirect effects of cerebrovascular disease. In patients with strokes, as in others, faecal impaction, urinary retention, and urinary infection may contribute to urinary incontinence. Managing urinary incontinence after stroke is therefore complex, demanding awareness not only of the possibility of constipation and urinary tract infection, but also of how it might feel to be unable to move or speak.

If confusion can be minimised and mobility restored continence too will often return. Regular toileting and charting of continence is essential because progress is thereby both encouraged and recorded. The doctor looking after a patient with stroke should not ask simply "Is she continent?" but, if she is, go on to ask himself which of several predictable factors may be contributing. Some will be specifically and easily remediable. Others will diminish only as orientation, mobility, and self care improve. A few survivors of stroke, but only a few (mainly the very demented and dependent), have permanently lost normal continence and may need to be considered for permanent catheterisation.

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Regular Review

Markers for insulin dependent diabetes: towards early detection

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Type I insulin dependent diabetes mellitus is probably due to autoimmune disease of the pancreatic β cells.¹ The insulinitis leads to insulin secreting cells being gradually destroyed and islet cell failure and clinical diabetes.² The prediabetic phase, which is thought to last for many years, is symptomless.

Whether this account is correct is important for two reasons. Firstly, if clinical diabetes reflects the complete and irreversible destruction of the functioning islet cell mass, only replacement treatment—that is, insulin—can be offered. Secondly, treatment in the lengthy prediabetic phase might be able to halt immune destruction. Such a possibility would require not only a suitable treatment but also a way of identifying those who needed the treatment—and the markers that might be used are the subject of this review.

Islet cell antibodies

Islet cells have many antigens distributed on their surface and in their cytoplasm, but these antigens have not been purified, isolated, or even identified—so that whole tissue must be used for islet cell antibody assays. The variants of islet cell antibodies—known as cytoplasmic^{3,4} complement fixing⁵ and surface antibody⁶⁻⁹—mostly reflect differences in methods of preparing the substrate and detecting the signal.

Understanding these differences in assay technique is important for interpreting the data they provide. Only the β cells are destroyed in the insulinitis of diabetes, yet the cytoplasmic islet cell antibodies react with all the islet cells. Complement fixing islet cell antibodies and islet cell surface antibodies^{10,11} may be selective for β cells, but this is not