

Indeed, the degree of the patient's suffering is such that she finds herself hoping his death will be considerably foreshortened as a result of her non-treatment decision. Her decision that she must continue treatment places greater emphasis on the moral quality of her intent than on what all who are involved agree are the best interests of her patient. The current state of the law unfortunately appears to encourage such prioritisation and should be questioned.

Even within the context of palliative care itself, there may still be difficulties with the doctrine of double effect. The moral justifiability of a potentially fatal regimen of morphine to relieve suffering will not depend only on the good intent of the palliative clinician. Suppose, for example, that a doctor administers potentially lethal palliation to a patient who is not terminally ill but is in pain from a recoverable injury which is very difficult to control. Given her duty to protect life (subject to the exceptions which have been outlined), the doctor's professed intent to relieve suffering is likely to cut little ice with the General Medical Council or the courts. Again, the issue at stake is not moral character. It is whether or not the doctor compromised the patient's best interests through placing him at unjustifiable risk. It will be this consequence of her behaviour on which she will ultimately be judged and by which her action will take on its moral and legal identity.

These examples show some of the problems of the double effect argument. In particular they indicate that

too much emphasis can be placed on professed intent and not enough on the conformity of foreseeable consequences to independent standards relating to when life sustaining treatment is not in the best interests of patients. These standards constitute the broader professional environment within which non-treatment decisions and potentially lethal palliation take on their specific moral and legal meanings. Intent in itself, therefore, cannot determine the rightness or wrongness of such decisions.

Of course, one reason for placing so much emphasis on individual intent may be a concern about slippery slopes to euthanasia. Once it is accepted that the interests of patients rather than the character of clinicians should dominate discussion of the moral and legal acceptability of non-treatment decisions or potentially lethal palliation then it is just a short step to argue for active euthanasia. If the most dramatic foreseeable consequence of non-treatment decisions or potentially lethal palliation can be in the best interests of patients then why can the same not be said of more active intervention which will have the same result? It is time we stopped avoiding real debate on the possible legalisation of active euthanasia by pretending that the double effect argument will somehow resolve it for us. It will not.

Len Doyal *Professor of medical ethics*

St Bartholomew's and the Royal London School of Medicine and Dentistry, London E1 2AD

Foodborne viral infections

Most are caused by Norwalk-like viruses, but we need to know more about these

Media attention on outbreaks of infection caused by salmonella species and *Escherichia coli* 0157 and more recently on new variant Creutzfeldt-Jakob disease have highlighted the importance of foodborne transmission of infectious agents, but what of the role of viruses? A recent report from the Advisory Committee on the Microbiological Safety of Food (ACMSF) provides a timely reminder of the importance of these agents, points to gaps in our knowledge, and offers advice for their control.¹

Although the list of viruses causing intestinal disease in humans is long, the epidemiology of foodborne outbreaks in the United Kingdom reveals the predominance of a single group, the Norwalk-like viruses.² Also known as small round structured viruses, these are an antigenically diverse group of caliciviruses which have similar morphology under the electron microscope and appear to cause an identical clinical picture of projectile vomiting and diarrhoea. They include Hawaii, Snow Mountain, Taunton, Mexico, and Grimsby viruses.^{3, 4}

Norwalk-like viruses can cause illness at any age, possibly due to their antigenic diversity but also because infection appears to induce only short term immunity.⁵ Although other enteric viruses such as rotaviruses, astroviruses, and Sapporo-like viruses (caliciviruses which are morphologically and genetically distinct from Norwalk-like viruses) have also been

associated with foodborne outbreaks, most of us are protected by long term immunity acquired during childhood. Hepatitis A virus is a rare cause of foodborne outbreaks in the United Kingdom but is noteworthy because the level of immunity to this virus is falling in the population and infection can be life threatening. Indeed, a recent report from Finland of two outbreaks of hepatitis A virus infection linked to centrally prepared food emphasises the potential risks posed by this virus.⁶

There appear to be two important means of transmission of viruses to food. The first is the contamination of bivalve shellfish harvested from inshore coastal waters. In filtering large volumes of seawater during feeding the shellfish sequester viruses.⁷ Although depuration reduces the load of pathogenic bacteria, it does little to remove viruses.⁸ These viruses will be inactivated during cooking, but shellfish that are not sufficiently cooked or eaten raw present risks. One such shellfish, the oyster, is usually preferred uncooked and consequently causes most outbreaks of viral infection associated with shellfish. The second, numerically more important route of transmission, is the contamination of food during preparation by infected food handlers. Any type of food may be contaminated by this means, although more frequently handled foodstuffs, such as salads, are more commonly implicated as a vehicle of transmission. Breakdown in

good hygienic practice is the cause of these incidents. Food workers with infection with Norwalk-like viruses should not be handling food until 48 hours after becoming symptom free. The situation is not so straightforward for hepatitis A infections as individuals are infectious before the onset of symptoms.

At first glance viruses appear to be an uncommon cause of foodborne infection. An analysis of outbreaks of infectious intestinal disease reported to the Communicable Disease Surveillance Centre in 1995-6 showed Norwalk-like viruses to be associated with only 6% of foodborne outbreaks, whereas these viruses caused 60% of outbreaks of gastroenteritis, where the mode of transmission was mainly from person to person.⁹ Pathogenic bacteria and toxins were more commonly associated with foodborne outbreaks, although no agent was identified in 12% of incidents. However, the available data are limited and probably seriously underestimate the importance of foodborne virus infections.

Norwalk-like viruses are difficult to detect.¹⁰ Electron microscopy of faecal specimens has been the mainstay of diagnosis in the United Kingdom, but virus is shed in relatively small numbers and only for a short time after the onset of symptoms. Until very recently it has been impossible to identify Norwalk-like viruses in contaminated food as these viruses do not grow in tissue culture. In addition, reported outbreaks of Norwalk-like virus infections are likely to represent only a small proportion of community acquired Norwalk-like virus infections. Much less is known about the burden of sporadic Norwalk-like virus disease, in particular the proportion due to foodborne transmission. We should soon have an answer to this important question when a government commissioned study of intestinal infectious disease in the community is published. Thus, although Norwalk-like viruses are not likely to be as important as enteropathogenic bacteria as a cause of foodborne illness, the total number of people affected each year is probably high.

The report of the Advisory Committee on the Microbiological Safety of Food clearly lays out the many

problems in assessing and controlling foodborne viral infections and makes 17 recommendations which fall into four broad areas. These are: (a) improved surveillance and diagnosis of foodborne outbreaks; (b) a reduction in environmental contamination with sewage, particularly of shellfish harvesting areas; (c) increased investment into the use of new molecular methods for identifying Norwalk-like viruses in food and for assessing measures for viral inactivation; and (d) an improvement in hygiene in the food industry, an important point which cannot be overstated.¹¹ These measures are to be welcomed as they will improve our understanding of the importance of these agents and, if implemented fully, should lead not only to a reduction in foodborne viral illness but also to an overall reduction in foodborne disease.

Antony Hale *Senior registrar in virology*

Enteric and Respiratory Virus Laboratory, Central Public Health Laboratory, London NW9 5HT (ahale@hgmpr.mrc.ac.uk)

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Public health, civil liberties, and tuberculosis

How society encourages compliance reflects society's approach to the vulnerable

Drug resistant tuberculosis is a global health threat. Perhaps because of the size and urgency of the threat and the fact that vulnerable populations are most affected by the disease, some control programmes include coercion. The responses to this threat reflect how society views those on the margins, who are vulnerable—perhaps homeless, stateless, or psychologically disturbed. When treatment compliance is required for public health reasons (to prevent the development of drug resistant strains) how society encourages compliance reflects as much on society itself as it does on the irresponsible, poorly compliant individual.

A tension has always existed between the protection of individual civil liberties and the protection of public health. In the liberal era of the

1960s and 1970s somewhat draconian approaches to the mentally ill, for example, were questioned. Legislation was amended to put individual patients at the centre, to emphasise their rights, and to provide them with greater legal protection. Detention of the mentally ill became dependent on a determination of the threat they posed to themselves or others. Historically a similar approach has been taken to isolating those with communicable diseases, so that detention of individuals with notifiable diseases has depended on an assessment of the threat they pose to public health. People with tuberculosis who do not adhere to treatment are at risk of both relapse and developing drug resistant tuberculosis, but the risks are unpredictable.¹

In London tuberculosis notification rates have increased over the past decade, and so have rates of

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