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# Gastroenteritis from Cheese

Even those with palates for very ripe cheese do not apparently risk their health in eating it. The occasional illness such as brucellosis from cheese[1 2] is usually traced to some brought privately from abroad and not to cheese sold commercially in Britain. Reports[3] from the U.S.A. of a widespread outbreak of gastroenteritis in adults due to imported cheese are therefore of some interest not only because of the food concerned but also because the offending organism, an enteropathogenic strain of *Escherichia coli*, is usually associated with illness in infants.

The outbreak[4] was known to have affected at least 387 of 409 persons at risk (95%) in 13 states in the space of four weeks at the end of 1971. Many of those affected developed a dysentery-like syndrome, associated in a few with the passage of blood. Six patients required hospital admission, but no deaths are known to have occurred.

From epidemiological investigations camembert and brie cheeses imported from France were strongly suspected to be the source of the outbreak; this was confirmed when *E. coli* 0124: B17 was isolated from the stools of several patients and from samples of the implicated cheeses. The same serotype was also isolated from a sample of coulommiers cheese produced by another factory in France but imported into the U.S.A. by the same firm, though no documented cases of gastroenteritis were attributed to that cheese. All the cheeses that had already been distributed were recalled by the importers. The recall was monitored by the U.S. Food and Drug Administration, and no further cases of gastroenteritis from French cheese have been reported since.

Though three brands of brie and camembert cheeses were implicated they had all been imported by one firm in New York and had been produced in one factory in France—and indeed were identical except for their shapes.[3] All the cheese had been manufactured during two days, and *E. coli* 0124 was isolated in the factory from the curdling tank and from samples of cheese. The probable source of infection was river water used in cleaning equipment: the filtration system at the factory had not been working efficiently at the time.[5] Further details of the treatment of river water for use in the factory were not available, but a filtration system alone is commonly regarded as insufficient to guarantee the purity of water for use in food manufacturing premises—chlorination is usually required. Fine filters capable of removing bacteria and even viruses from water are available, but these are impracticable with the large volumes required in manufacturing processes, especially with river water as a source. A coarse filter was most probably used, and this would remove large particles and organisms such as algae only. Hyperchlorination of the filtered water would then be necessary followed by dechlorination using a carbon filter.

Gastroenteritis in infants from enteropathogenic serotypes of *E. coli* is well recognized, and outbreaks are commonly reported,[6-8] and symptoms may also be produced in volunteer adults and children.[9-13] Reports of outbreaks in adults are, however, few. In 1949 Hobbs, Thomas, and Taylor[14] described a school outbreak of gastroenteritis associated with a pathogenic paracolon bacillus (now known as *E. coli* 0124) and were able to reproduce symptoms in volunteer experiments. A year later Stevenson[15] reported the occurrence of *E. coli* D433 (*E. coli* 0111) in the stools of adult patients with diarrhoea. More recently, in an investigation of travellers' diarrhoea in a group of British soldiers in Aden[16] a new *E. coli* serotype, 0148, was found in as many as 54% of those who had diarrhoea but not in any of those without diarrhoea. Last year 87 of 714 adults developed diarrhoea less than 24 hours after eating ham and egg pie,[17] and *E. coli* 0127 was isolated from all 15 patients investigated and from samples of pie remains and unused pies. It seems probable that outbreaks of *E. coli* gastroenteritis in adults may be more common than hitherto recognized.

The cheese associated with the outbreak in the U.S.A. was stated to have been consumed in France and the U.S.A. only, though there were no reports of gastroenteritis from this source in France. There have also been no reports to date of gastroenteritis from French cheese in Britain and, indeed, the moral to be drawn from this outbreak is not to beware of French cheese. The real lessons are much broader. Firstly, in the investigation of an outbreak of foodborne gastroenteritis the bacteriologist's search must not be considered to be complete if salmonellas, *Staphylococcus aureus*, and *Clostridium welchii* are not identified. In recent years, *Vibrio parahaemolyticus*,[18 19] non-agglutinating vibrios,[20] (*Bacillus cereus*,[21 22] and now *E. coli* have also been incriminated in outbreaks of food-poisoning and must be looked for in appropriate instances. Secondly, it is important not to assume that a particular food is innocent because it has never been known to cause gastroenteritis.

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[18] Public Health Laboratory Service, *British Medical Journal*, 1972, 1, 701.
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# Cars or Chairs?

For some time pressure has been mounting to replace three-wheeler invalid cars by specially converted conventional motor cars. Last week there appeared a critical examination by Baroness Sharp of the consequences of such a change.[1]

Lady Sharp's report was prepared without knowledge of any official verdict on the safety of three-wheelers; but, as

she points out, that did not matter since she was already convinced that there is an overwhelming case for their replacement. Apparently a small car adapted for driving by a disabled person is now cheaper than a three-wheeler and likely to become progressively more so. Add to this the noisiness, lack of comfort, and unreliability of the three-wheelers, and the fact that they cannot take passengers, and it is clear that the converted motor car is a far better vehicle for most disabled drivers.

Unfortunately this presents a problem. Lady Sharp argues—and many would agree—that "there can be no justification for insisting that a disabled person otherwise eligible cannot have a car if he or she is unable or does not want to drive, and can nominate an appropriate person to be the driver." One advantage of a change to cars would, she claims, be that disabled people who really should not be driving would no longer feel under any compulsion to try. But once it was agreed that disabled people could be supplied with cars to be driven either by them or by someone else the demand would be vast—perhaps 200,000 cars—and restrictions on eligibility would be needed. Lady Sharp recommends a test based on both physical handicap and on social need, by which she means a need for a car to get to and from a full-time job, to get to a place of further education, or to look after a household or keep a family together. What could not be offered would be cars to give disabled persons simply social mobility—travel to and from church and tea with friends—so that the change could mean loss of the right to a vehicle for some who have them at present. Furthermore there are quite a number of disabled people for whom the three-wheeler is preferable on practical grounds. It gives more headroom; it can accommodate a wheelchair; its tiller steering is easier for some disabled to manage; and it is small, tucking out of the way when parked. Lady Sharp was told that if cars replace three-wheelers for many people then the three-wheelers will go out of production, and again some disabled would suffer.

At this point it becomes clear that the wheel has turned a full circle. The three-wheelers were first introduced as a development of the motorized Bath chair, itself a vehicle provided to overcome the disabled person's inability to walk—an extension of the artificial limb. As such it was clearly a personal aid. Progressive improvements to the powered chair made it in effect a small three-wheeled car. Only then was it compared with a car—and it compares very badly. However, as an all-weather self-propelled wheel-chair it does quite well.

The proposed switch from a powered wheel-chair to a family car changes the whole concept from a medical aid to a social service. Almost every family wants a car; and, as Lady Sharp points out, it is not only the physically disabled whose lives would be transformed by one. There are those who are virtually housebound by the need to look after a mentally handicapped child or an ageing relative. Should cars really be the first priority in Government spending on the disabled? A disablement income as of right,[2] which is already given to the disabled in several E.E.C. countries, might well be seen as more equitable. Clearly if cars are made available there will need to be a stringent controls on their issue, and this will lead to feelings of unfairness among those who do not get them, who will be in the majority.

Lady Sharp suggests that it would take four or five years to provide the 40,000 cars she estimates would be needed if the policy were changed, and that may explain Government reluctance to declare the three-wheelers unsafe, since such an announcement would logically mean that they should be taken off the road at once. Before any final decision is taken some clear thinking is needed on the purpose for which vehicles should be provided for the disabled and on their place in the hierarchy of priorities.

[1] Lady Sharp, *Mobility of Physically Disabled People*. London, H.M.S.O., 1974, price 42p.
[2] *Creating a National Disability Income*, Occasional paper no. 12. Godalming, Disablement Income Group, 1972.

# Adverse Reactions to Beta-Adrenergic Blockade

Beta-adrenergic blocking drugs isolate the heart from the effects of sympathetic stimulation. They slow the heart rate, reduce the velocity of contraction, and delay atrioventricular conduction. Inhibition of beta-receptor stimulation in the bronchial tree and peripheral circulation enhances bronchoconstrictor and vasoconstrictor mechanisms, while inhibition of the sympathetic transmitters increases the influence of the parasympathetic system. The contraindications to the use of beta blockers result directly from their anti-beta-adrenergic action. In general they should not be prescribed in congestive heart failure after acute myocardial infarction, in states of impaired atrioventricular conduction, or to asthmatics. In conditions of vagotonia—as is common after myocardial infarction—unopposed vagal action may lead to profound falls in cardiac rate and output.

Since the introduction[1] of propranolol in 1964 beta-adrenergic blocking drugs have been widely used. Three drugs are generally available in Britain; propranolol and oxprenolol,[2] which are nonselective and short acting, and practolol,[3] which is longer acting, blocks predominantly excitatory receptors, and is therefore relatively cardioselective. Neither the membrane stabilizing effects demonstrable in the D-isomer of propranolol and in oxprenolol nor the mild agonist activity demonstrable before beta-inhibition in oxprenolol and practolol have any clinical relevance. All the therapeutic and toxic effects and most if not all of the side effects are attributable to the beta-blocking action. These drugs find their most important application in the treatment of angina. Their other principal uses include the treatment of high blood pressure, thyrotoxicosis, hypertrophic cardiomyopathy, and cardiac dysrhythmias.

In a report from the Boston Collaborative Drug Surveillance Program Greenblat and Koch-Weser[4] have reported on the adverse reactions to propranolol found in 268 medical patients in hospital with a wide variety of cardiovascular disorders. No deaths were attributed to the use of the drug. Eight patients suffered life threatening reactions, and in each case the adverse effects could be attributed to the pharmacological action of the drugs. Extreme bradycardia occurred in a 70-year-old with thyrotoxicosis on a dose of 120 mg per day and pulmonary oedema or extreme bradycardia developed in two patients after acute myocardial infarction. Three patients with heart disease, all elderly, developed pulmonary oedema or bradycardia, and two other patients with ischaemic heart disease developed hypotension and shock. Non-life threatening reactions occurred in 15 patients; again most were directly attributable to a beta-adrenergic blocking action detrimental to the patient. The Boston report concluded that the use of propranolol in patients in hospital is associated with appreciable risks, but that adverse reactions can be predicted in patients with severely compromized cardiac function.