and acyclovir. While these children and their parents may desire the increased protection of community immunity, the increased risks that such immunity entails for otherwise healthy children cannot be justified.

The costs of chickenpox infection are partly the medical expenses and partly the days of work lost among families. The medical expenses are generally low. Studies have shown that universal chickenpox immunisation is not cost effective in terms of health costs alone.[7,8] These studies may even underestimate the costs, because they do not account for the possible increase in costs if universal immunisation delays disease until adulthood.

The cost of days of work lost by parents because of their children's chickenpox is substantial, and universal chickenpox immunisation would probably be cost effective from this angle.[8,9] A large part of the cost, however, is due to policies of isolation. We believe that this cost is avoidable; if it was avoided this might tip the balance of the cost-benefit studies against universal chickenpox immunisation. Children should not have to stay home while asymptomatic but still capable of transmitting the disease. This policy, which is justifiable primarily on the basis of its benefit to immunocompromised children, in fact offers such children false security since they are still exposed to children who are presymptomatic but are capable of transmitting the disease. The best way to protect immunocompromised children is to immunise them, not all their peers.

A policy of mandatory universal immunisation would be justified only if the benefits of participation for each individual outweighed the risks and costs. Given the mild course of chickenpox in healthy children, such a policy is not justified. Chickenpox immunisation should be recommended only to families in which one or more members are at high risk of serious infection.

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The MacLean Center for Clinical Medical Ethics is supported by grants from the Dorothy J MacLean family, the Henry J Kaiser Family Foundation, the Andrew W Mellon Foundation, and the Pew Charitable Trusts.

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Thrombolysis in patients with diabetes

Withholding treatment is probably mistaken: patients should be given a choice

Any junior doctor treating a patient with diabetes mellitus and an acute myocardial infarction faces a dilemma. Lists of cautions and contraindications for thrombolytic treatment usually include diabetic retinopathy. The reasonable fear of precipitating a vitreous or retinal haemorrhage helps to explain why fewer diabetic than non-diabetic patients are given thrombolysis.[1,2] Funduscopy is not, however, easy in a brightly lit receiving room after the administration of opiates. Even after mydriatic drops are given it may not be possible definitely to exclude changes in the eye. The next hurdle to face after making the decision to give thrombolysis—or not—is to justify one's actions on the post-take ward round.

The *British National Formulary* states that diabetic retinopathy is a contraindication to thrombolysis, although this will be changed to a caution in future editions. The datasheets from drug manufacturers vary from making no mention of diabetes (anistreplase, Boehringer) through advising special caution in the presence of diabetic proliferative retinopathy (alteplase, Boehringer) to stating that thrombolysis is contraindicated in severe diabetes mellitus (streptokinase, Hoechst) or in diabetic retinopathy (streptokinase, Pharmacia). Junior doctors must find it difficult to give a drug when its use is directly contraindicated in the *British National Formulary*.

Against that background the lack of published case reports is surprising. We have been able to find one account of bleeding from retinopathy in a single diabetic patient after thrombolysis[3] and one other of ocular haemorrhage after streptokinase in a patient without diabetes.[4] In neither case was there any long term effect on vision. The Committee on Safety of Medicines has received one report of subconjunctival haemorrhage associated with streptokinase. In a published overview of fibrinolytic trials in patients with myocardial infarction the proportionate reduction in 35 day mortality was slightly, but not significantly, greater in diabetic patients (136/1000 *v* 173/1000; 21·7%) than in non-diabetic patients (87/1000 *v* 102/1000; 14·3%).[5] These figures imply that, for every 1000 diabetic patients treated, 37 patients survive who would otherwise have died. The overview of fibrinolysis found no evidence of excess bleeding or stroke in the diabetic patients. One small study suggested an excess risk of haemorrhagic complications in diabetic patients aged over 75,[6] but in an analysis of over 9000 patients treated with thrombolysis, of whom a tenth had diabetes, complication rates were similar in the diabetic and non-diabetic patients.[7]

Among the large trials of thrombolytic treatment only that conducted by the Gruppo Italiano per lo Studio della Streptochinasi nell'Infarto Miocardico included haemorrhagic diabetic retinopathy as a contraindication to treatment,[8] while the second[9] and third[10] international studies of infarct survival and the study by the Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto Miocardico[11] made no mention of diabetes, with or without retinopathy, in their exclusion criteria. In these trials alone more than 80 000 patients, of whom around 11% had diabetes, received thrombolytic treatment, without any reports of detrimental effects in their eyes. In a subgroup analysis of the thrombolysis and angio-

plasty in myocardial infarction trial no retinal haemorrhages were seen in 148 diabetic patients, 7% of whom had documented retinopathy.[12]

Patients with diabetes have a poor outcome after myocardial infarction, yet—as for some other groups with a poor prognosis—they are less likely to receive thrombolytic treatment.[2] We believe that in the light of the proved benefits of thrombolysis in patients with acute myocardial infarction the treatment should not be withheld on the basis of the existence of diabetic retinopathy.[4] Any increase in risk seems to be small. Those who support the empowerment of patients might also suggest that this is a decision in which it is appropriate for the patient to have a voice.

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Bronchiolitis

Tachypnoea (>50 breaths/min) warrants admission to hospital

Bronchiolitis is a pathological description that has come to be used as a clinical diagnosis. It is primarily a disease of the small airways, causing these to be obstructed by inflammatory exudate. More than 70% of cases are caused by respiratory syncytial virus, which in temperate climates results in a sharp winter epidemic lasting two to five months.[1] Bronchiolitis is a disease of infancy, characterised by cough, fever, tachypnoea, diffuse crackles, hyperinflation, and chest retraction. Wheezes are a less constant feature,[1-3] and bronchiolitis should be distinguishable clinically from infantile asthma by the presence of widespread crackles. Unfortunately, the diagnostic criteria for bronchiolitis have varied considerably, with consequent blurring of the distinction between it and asthma.[4]

Over 95% of infants have been infected with respiratory syncytial virus by the end of their second winter; 40% of the infections in infancy affect the lower respiratory tract,[1-5] although only about 1% of these children will need admission to hospital.[1] The overall mortality from primary infection in previously healthy infants is low and has been estimated at from 1 in 5000 to 1 in 20 000.[6] The mortality among children admitted to hospital with respiratory syncytial virus infection is about 1% and is about 3.5% for those with underlying cardiac or chronic lung disease.[7] Other high risk groups for severe infection are babies born before term[1 4] and children with congenital or acquired immunodeficiency.[8 9]

Transplacental maternal antibody confers at best partial immunity, so, although all adults have antibody to respiratory syncytial virus, babies can develop severe infection from birth. However, a "honeymoon period" exists up to 4 weeks of age, during which infection is relatively uncommon, perhaps because of some relative protection from maternal antibody or decreased exposure.[1] The peak incidence is 2-5 months of age. If babies become infected in the first month, and particularly if they were born before term, apnoea may be the first sign of illness.[10]

Reinfections with respiratory syncytial virus, of decreasing severity, occur throughout life.[11] Although reinfections virtually never cause bronchiolitis, they are epidemiologically important in forming a reservoir of infection so that infants are infected for the first time by a school age sibling or an adult with a cold.[6] The main mode of transmission of the virus to infants is probably through direct inoculation of nasal secretions on the hands of infected children or adults,[2] although spread by fomites may also be important.[12] Spread of infection through droplets seems to be less important.[1 13]

The risk of the lower respiratory tract being affected in respiratory syncytial viral infection is increased by overcrowding, day care, and parental smoking and is reduced by breast feeding for longer than one month.[4 14] Children admitted to hospital with bronchiolitis due to respiratory syncytial virus have about a 1 in 2 risk of later recurrent wheezing[1 4 15]: some develop classic asthma, while others have bronchial hyperreactivity even after symptoms have resolved.[16] It is unclear whether pre-existing atopy predisposes to severe bronchiolitis and later asthma or whether infection with respiratory syncytial virus damages the bronchial mucosa; allows the entry of, and sensitisation to, inhaled allergens; and thus "causes" asthma. Children infected with respiratory syncytial virus who are not admitted to hospital are not at increased risk of asthma.[1 4]

The treatment of bronchiolitis is largely supportive. In severe cases treatment with oxygen can be lifesaving. Most studies, including some recent ones, have suggested that bronchodilators have no role and might even be deleterious.[17 18] However, a trial of nebulised salbutamol for wheezy infants with a diagnosis of bronchiolitis, half of whom were positive for respiratory syncytial virus, showed improvement, even in those under 6 months old.[19] These varying results may be due to different diagnostic criteria, but they suggest that when wheeze predominates, a trial of nebulised salbutamol is indicated. Corticosteroids are of no benefit in bronchiolitis.[4] Systemic bacterial superinfection is rare, even in severe infections with respiratory syncytial virus[20]; and antibiotics are not routinely indicated.

In general practice, assessment of severity is critical. Mild