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

cases, which do not necessitate admission to hospital, will generally occur in babies aged 3 months or more, born at term, who are feeding well and whose respiratory rate is <50 breaths/min. Moderate cases necessitate admission to hospital or at least management at home with small, frequent feeds and frequent observations. In these cases the babies are tachypnoeic (50-70 breaths/min), with only slight difficulty with feeds. In severe cases the babies are highly tachypnoeic (>70 breaths/min) or have apnoea, do not feed well, and need urgent admission. Clinical predictors of severity are tachypnoea (>70 breaths/min), age less than 3 months, preterm delivery (particularly before 34 weeks), and an "ill" or "toxic" appearance.[21] Babies with difficulty in feeding or apnoea should be admitted, and it is probably wise to admit babies with underlying heart, lung, or immune problems who develop bronchiolitis. Cyanosis is a late and sinister sign.

Pulse oximetry, if available, is the best predictor of the severity of the disease and need for oxygen.[21 22] Given its valuable role in assessing children with bronchiolitis and, to a lesser extent, other respiratory diseases, general practitioners should strongly consider buying a pulse oximeter. The mortality associated with bronchiolitis is low, but babies can deteriorate rapidly and clinical skills are necessary to reduce the mortality and morbidity still further.

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Growing pressure on *BMJ*'s obituaries

Something will have to give

Our obituary pages are experiencing growing pressure for the simple reason that the number of British doctors has more than doubled in the past 45 years. In 1950 there were just under 70 000; now there are more than 140 000. Unlike in any other part of the *BMJ*, in the obituaries section we accept everything that is submitted to us—provided that it arrives within our time limit of three months and we have not received any other submission on the person who has died. But we are unlikely to be able to sustain this policy indefinitely, and we welcome readers' ideas on how we might change.

We publish obituaries on about 400 doctors annually, which must be less than half of the British doctors who die each year. We commission obituaries of the famous, which amount to one or two a month; we simply wait for obituaries on other doctors to arrive. The form of the obituary (whether or not the biographical details appear in italic after the main body of the text) is determined simply by the amount of information we are given: with many obituaries almost nothing would be left if we extracted the straightforward biographical details for the final paragraph. Most obituaries are published in the order we receive them, and sadly there is a delay of about three months (meaning that obituaries appear on average about five months after the death).

The pressure on our obituary pages has been increasing for many years, and in 1992 we undertook a survey of 1080 readers to ask how we should respond. Almost half (49%) responded, and they were strongly against us selecting obituaries for publication on the grounds of fame or interest and rejecting the rest. Most didn't want us to increase the number of pages we devote to obituaries, but they did recognise our problem and suggested that we shorten obituaries further and reduce our time limit. We adopted both these suggestions but have a more generous time limit—three months—than our respondents recommended.

But still our obituaries present us with problems. One of the most unpleasant parts of our job is to have to talk or write to bereaved people who have submitted obituaries after the three month limit. About 45 obituaries a year are submitted late, and about a third of authors appeal against our decision not to publish. We always say "no" because to say "yes" would be unfair on those who missed the time limit and therefore did not submit anything and on those who accepted our decision not to publish without appealing. We would find it invidious to have to decide which deaths were the saddest and which excuses for late submission the best.

A second problem with our obituaries is that many are dull, reflecting only the successes in the lives of those who have died. Most of the doctors whose obituaries appear in our pages seem to have been saints, whereas all doctors have light and shade in their lives. Sadly, medicine does not any more have a culture of "telling it like it is" in obituaries—but it would be good to start one. One strategy we adopted in the hope it would lead to more interesting obituaries was to ask people to write their own[1]: people would surely, we thought, have insight into their own deficiencies and failures. Sometimes, but not often, this strategy has been successful. Archie Cochrane, the famous epidemiologist, wrote probably our best self written obituary: "In 1957 he survived a professor of