LETTERS TO THE EDITOR

Trends in respiratory mortality in England and Wales

Mr P N Lee and colleagues (September 1990;45:657–65) show that mortality rates for chronic obstructive lung disease are characterised by a cohort related peak (occurring 20–30 years later in women than in men) superimposed on a downward drift (in both sexes). The drift component has previously been interpreted as a cohort effect due to the declining incidence or severity of respiratory illness in early childhood, despite being presented as a period effect.[1] However, as Mr Lee and his colleagues point out, linear drifts cannot be reliably assigned to period or cohort, and only non-linear trends can be partitioned in this way.[2]

Inspection of earlier mortality data for England and Wales during 1861–1930[3] suggests that deaths attributed to bronchitis (acute, chronic, and unspecified) began to decline simultaneously in all age groups around 1880 (figure), suggesting an influence of period of death rather than a cohort effect. This decline occurred in both sexes (not shown) and preceded the decline in mortality due to bronchitis and pneumonia in early childhood by some 20 years (figure). Furthermore, there is no evidence of a downward inflection around 1900 in the cohort curves for chronic obstructive lung disease (figs 3 and 4 in Lee *et al*), as would be expected if childhood respiratory infection influenced subsequent respiratory death rates in the same cohorts.

Cause specific mortality rates for the first year of life are not available for the nineteenth century, and there must be considerable uncertainty about the interpretation of bronchitis death rates from a period when pulmonary tuberculosis was the leading cause of death. Nevertheless, these observations offer no support for the hypothesis that the remarkable underlying decline in mortality from chronic obstructive airways disease since the war is a result of changes in the incidence or severity of respiratory infection during infancy in the earlier part of this century.[1]

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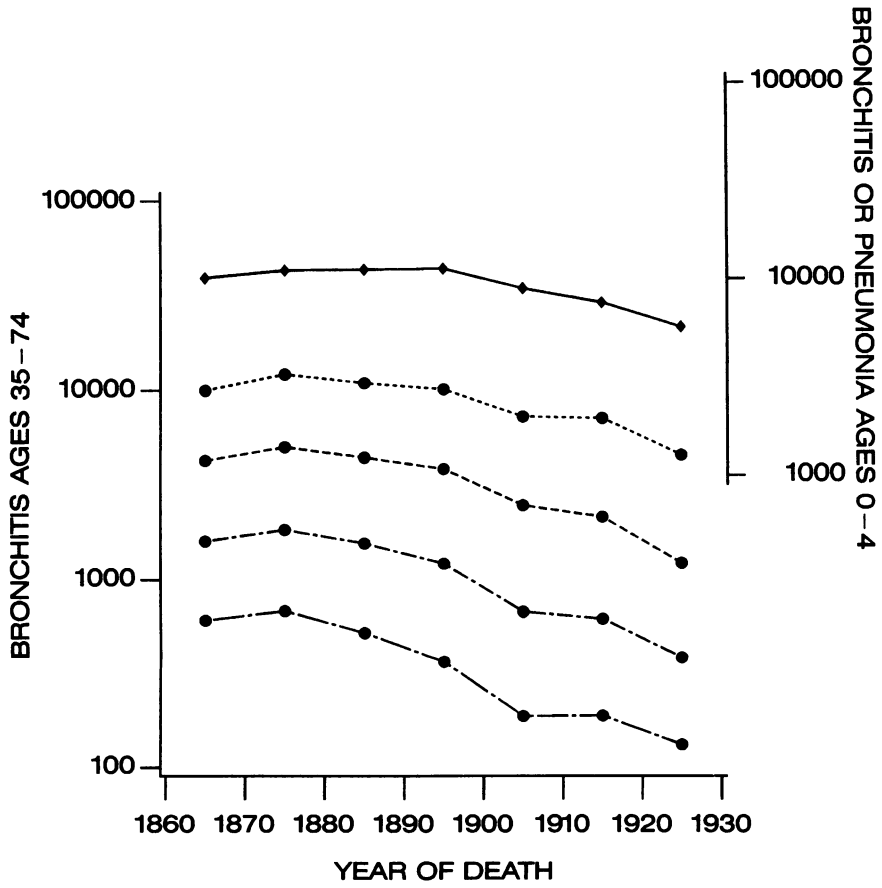
1 Barker DJP, Osmond C. Childhood respiratory infection and adult chronic bronchitis in England and Wales. *BMJ* 1986;293:1271–5.
2 Clayton D, Schifflers E. Models for temporal variation in cancer rates. II: Age-period-cohort models. *Stats Med* 1987;6:469–81.
3 Registrar General for England and Wales. *Decennial supplement 1931*. Part III, table 12. London: HMSO, 1952.



Mortality rates (per million per year) for males and females combined, England and Wales 1861–1930. Bronchitis (all forms) at ages 35–44 ——●——; 45–54 — - ● — -; 55–64 - - ● - -; 65–74 - - ● - -. Bronchitis or pneumonia at ages 0–4 —— ◆ ——. Note that the vertical scales are logarithmic.

Pericardial effusion after open heart surgery

In their review (September 1990;45:655–6) Drs A J Bryan and G D Angelini rightly emphasise the important diagnostic role of two dimensional echocardiography. They seem, however, to have overlooked certain relevant papers,[1–3] which, in brief, show the following. (*a*) Large postcardiac surgery pericardial effusions causing tamponade are often loculated posteriorly, the anterior pericardial sac being virtually obliterated by intrapericardial adhesions. Tamponade resulting from such effusions are in fact more common in our experience[1–3] than tamponade resulting from circumcardiac effusions in the postcardiac surgery setting. (*b*) Large posterior loculated effusions causing postcardiac surgery tamponade may present with certain specific echocardiographic manifestations: (i) pronounced distension of the oblique pericardial sinus[3]; (ii) stretching of left pulmonary veins[3]; (iii) paradoxical motion of the left ventricular posteroinferior wall.[1] (*c*) Tamponade tends to occur with somewhat smaller amounts of pericardial fluid in posterior loculated pericardial effusions than is the rule with large "medical" circumcardiac effusions.[1] (*d*) In patients with large post-surgical pericardial effusions but little or no clinical or echo evidence of tamponade serial echocardiography has a useful role in management.[2] If the effusion does not increase progressively in size and/or new intrapericardial spoke like fibrous bands develop, tamponade is very unlikely to supervene and conservative management (without surgical intervention) can be advised with confidence. Such effusions eventually disappear, though some may do so in several weeks or months.

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1 D'Cruz IA, Kensey K, Campbell C, Replogle R, Jain M. Two-dimensional echocardiography in cardiac tamponade occurring after cardiac surgery. *J Am Coll Cardiol* 1985;5:1250–2.
2 D'Cruz IA, Dick A, Pai GM, Kamath MV. Large pericardial effusion after cardiac surgery: Role of echocardiography in diagnosis and management. *South Med J* 1989;82:287–91.
3 D'Cruz IA, Macander PJ, Gross CM, Pai GM. Distension of the oblique pericardial sinus in tamponade due to loculated posterior effusion. *Am J Cardial* 1990;65:1520–1.

AUTHORS' REPLY We welcome the interest in our editorial shown by Drs D'Cruz and Pai. We are aware of their work in this area; it was impossible, however, to make reference to all published work in a short editorial.

We agree that posterior effusions are much more common after surgery and more often cause tamponade within this setting. We acknowledge the echocardiographic features they have identified that suggest tamponade. The possibility that tamponade tends to occur with smaller amounts of fluid in this context is speculation, which may be true but at present is unsupported by scientific data (including their reference 1). Serial echocardiography may be used to follow the natural tendency to resolution of post-operative effusions, though in the absence of clinical symptoms it does not alter management, which is conservative.

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