AUTHORS' REPLY Dr Thurlbeck objects to our conclusion that the severity of the lesions of the bronchioles and parenchyma assessed by grading techniques in resected lung specimens do not correlate with lung function measurements obtained in the same subjects. We agree that this conclusion is at variance with much that is in the literature and, indeed, reported it for that reason. The data we presented make two basic points: (1) that rather extensive emphysematous destruction of the lung can be present in patients who have a normal FEV_1, and (2) that a reduction in FEV_1 did not correlate with increased inflammatory changes in the peripheral airways assessed with current grading systems. This does not mean that we think emphysematous destruction is not important, only that its presence does not explain the decline in FEV_1. Similarly, we did not mean to imply that the abnormal peripheral airway function may not eventually be explained by the effects of the inflammatory process in these airways – only that current morphological grading systems do not identify the lesions responsible for the abnormalities in function. We agree with his closing statement that airflow obstruction is a complicated matter in which the different lesions of the bronchi (not just chronic bronchitis), bronchioles (not just the Cosio score), and parenchyma (not just emphysema) contribute to the decline in function. Specific responses to the other points raised in his letter follow.

The criticism concerning our failure to report data on tumour size and stage does not recognise that we have previously reported these data on the first 143 cases entered into our study.[1] Tumour size, involvement of lymph node by tumour, and staging were included in that report. A more recent analysis of staging also showed no correlation between tumour size, stage, and function. We are well aware that centrally placed tumours might interfere with lung function and did our best to exclude this possibility before a case was entered into the study.

His objection to the fact that most of our cases were lobectomy specimens is irrelevant because the manuscript clearly states that the same result was obtained when the data from patients undergoing pneumonectomy were examined separately. The percentage of cases in each FEV_1 category that were pneumonectomies (table 2) was not different between groups. Furthermore, we have previously shown that the problem of emphysema distribution can be taken into account when single lobes are evaluated.[2] His conclusion that the emphysema shown in the specimen in the figure is subpleural is interesting. The magnification bar which is included allows the reader to confirm that the emphysematous destruction extends to 4–8 cm below the pleural surface in the mid sagittal plane. In our view this constitutes far more than subpleural disease.

The criticism that we have tried to mislead the readers in table 2 is unfounded. Line 2 of the table shows the prevalence of emphysema which increases from 2 in 24 (9%) of those with an FEV_1 >110 to 11 of 22 (50%) of those with an FEV_1 <50% of the predicted value. Line 3 shows the mean emphysema score for the cases in each category where the lesion was present. Simple arithmetic using the data in lines 2 and 3 allows the mean score to be calculated for all the cases. Our point is that the effect of emphysema on the decline in FEV_1 is a direct result of an increase in prevalence (line 2). While this may be of some interest to those interested in population studies, it does not relate emphysematous destruction to a decline in FEV_1. When the FEV_1 is compared with the severity of the disease present in the specimens with emphysema, lung destruction did not increase as FEV_1 declined. We interpret this to mean that an increase in the grossly visible emphysematous lesions does not explain the decline in FEV_1. By exclusion, we suggest that the contribution of the decrease in elastic recoil (Pimax) to the decline in FEV_1 documented in table 1 might be related to microscopic lung destruction.

Dr Thurlbeck extends the interpretation of the measurements of L_M beyond our intentions. Our data show (table 2) that the L_M values were similar as FEV_1 ranged from 50% to 100% of the predicted value, and increased only in the group whose FEV_1 values are less than 50% predicted. It is true that the values for L_M which we obtained are smaller than those reported by others and we do not understand the reason for this result. However, as the same person made all the measurements, it is reasonable to compare the values between groups. We estimate that L_M was measured at approximately 40% of TLC by comparing the volume of fixative added to the lung with the volume of air present at TLC. Table 1 shows that elastic recoil decreased as FEV_1 declined at TLC but not at FRC. This makes it hazardous to interpret L_M values measured at volumes that were well below where the loss of elastic recoil was measured. Reduced lung elastic recoil could be based on either a loss of surface area or decreased elastic properties of the tissue. Both of these factors could increase airspace size at TLC in a way that might not be apparent at lower lung volumes. We speculate that the measured elastic recoil at TLC might be due to increased airspace size even though we were unable to measure it using L_M.

In summary, we reported a 10 year study of more than 400 cases where lung function was measured in close proximity to obtaining lung tissue by resection. Previously reported grading systems were used to evaluate airway and parenchymal structures in relation to the decline in lung function. The data show that the presently used grading system for peripheral airway pathology does not provide data that adequately explain the decline in lung function. The results also strongly suggest that significant emphysematous destruction of the lung can be present when the FEV_1 is normal. This does not mean that the observed airway and parenchymal lesions are harmless, only that they do not explain the decline in FEV_1. We trust that our report will cause those interested in the condition to seek new and better ways of investigating lung structure in relation to function because, in our view, those that have been used for the past 30 years need updating.

JAMES C HOGG
UBC Pulmonary Research Laboratory, St Paul's Hospital, Vancouver, British Columbia, Canada V6Z 1Y6

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Chronic respiratory questionnaire

The recent article on reliability and validity of the chronic respiratory questionnaire (CRQ) by Wijkstra *et al* (May 1994;**49**:465–7) highlights important aspects of the research process. It is imperative that new and old measuring tools – either questionnaires or physical measurements – are assessed in terms of validity and reliability. It is only through such rigid tests that comparative measurements may be made to further knowledge but, unfortunately, in this particular research, inappropriate methodologies were used to measure validity and reliability.

Test-retest is a common means for measuring reliability but the time between tests should be short enough for change not to occur, yet long enough to exclude high agreement due to remembrance.[1] This time is suggested by de Vaus[2] to be between two and four weeks. The time gap of one day used in this particular study may result in the high correlation scores obtained for fatigue, emotion, and mastery. Secondly, the authors described the testing of the CRQ with the SCL-90 as content validity when in fact it was criterion validity that was being measured – that is, the correlation between a new measuring tool against an already accepted criterion – in this case the SCL-90. Content validity should have been qualitatively measured by a panel of experts in the field of chronic respiratory disease. It is noted that construct validity was not measured, and hence the range of tests to validate this questionnaire and measure its reliability were not completed.

It is imperative to ensure reliability and validity of new measuring tools, but even more important to carry out the correct procedures to ensure valid results.

U JONES
Institute of Health Care Studies, University Hospital of Wales, Cardiff CF4 4XW, UK

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AUTHORS' REPLY We appreciate the comments of Mrs Jones regarding the methodologies that were used to measure reliability and validity of the chronic respiratory questionnaire (CRQ) but we do not entirely share her opinion.

Firstly, we disagree with her remark concerning the test-retest reliability – that is, the time between both assessments of the CRQ. The CRQ assesses quality of life with reference to the two weeks preceding the test. The suggestion of Mrs Jones to have at least two weeks between both assessments would mean that quality of life would be measured over two different periods. Differences in quality of life in patients with COPD are likely to occur when measured over a different period. This means that any difference between the two assessments would have nothing to do with test-retest reliability. Secondly, if remembrance is as important as Mrs Jones suggests, it is remarkable that scores on the dimension dyspnoea during activities chosen by the patients themselves have only a moderate test-retest reliability compared with the

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other three dimensions. We believe that a gap of one day between both measurements is justifiable to obtain test-retest reliability of the CRQ in this particular group of patients.

Perhaps the most fundamental misunderstanding is that our research was focused on the prediction of a specific criterion (criterion validity) based on the scores of the CRQ. In our research we investigated the validity of the four dimensions of the CRQ by relating these dimensions to comparable dimensions of the SCL-90. Thus, the principal focus was not on the prediction of a criterion (according to Mrs Jones, the scores of the SCL-90), but on the ability of the CRQ to measure the four dimensions. From a methodological point of view it should be noted that the scores on the CRQ are based on psychological properties of individuals. As a consequence it is often impossible to find a single satisfactory real world criterion available for the evaluation of its validity. The SCL-90 can never serve as such a naturally occurring criterion. Content validity and construct validity are powerful tools to establish our understanding of what dimensions of specific measurement instruments intend to measure, and that was, indeed, the core issue of our research.

P J WIJKSTRA
E TENVERGERT
Asthma Centre,
Beatrixoord Hospital,
9751ND Haren,
The Netherlands

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Impact of HIV on tuberculosis in developing countries

The advent of HIV has considerably affected tuberculosis control programmes in developing countries as outlined by Drs Nunn, Eliott, and McAdam (May 1994; **49**:511–8). In Blantyre, Malawi, for example, the number of notified new cases of tuberculosis has increased fourfold between 1986 and 1993. The brunt of this increase has been borne by young adults and children.

Nunn and colleagues comment on the disproportionate increase in smear negative pulmonary tuberculosis in Malawi. Although in 1986 90% of new cases of pulmonary tuberculosis in adults aged between 15 and 44 in Blantyre were AAFB smear positive, this fell to 33% in 1991. Possible reasons for this change included the association of HIV with an increase in smear negative pulmonary tuberculosis, a breakdown in the sputum microscopy service or, alternatively, the occurrence of other HIV associated pulmonary pathogens mimicking tuberculosis.[1]

We investigated 82 patients with sputum smear negative, but clinically suspected, pulmonary tuberculosis by the method of sputum induction.[2] The induced sputum was examined for *Mycobacterium tuberculosis* and other significant pathogens. Tuberculosis was confirmed by culture in 30. The only other significant pathogens detected were *Pneumocystis carinii* in one patient and *Strongyloides stercoralis* in a second. Both patients were HIV seropositive. We concluded that, although other HIV associated pathogens do occur in Malawi, they appear to be currently uncommon.

During 1992 intensive efforts were made to ensure that all patients had a proper sputum examination for AAFB. By the end of 1992 the proportion of young adults with smear positive pulmonary tuberculosis had risen from 33 to 60. This re-emphasises the vital importance of a good sputum microscopy service in a tuberculosis control programme, even in areas with a high incidence of HIV infection.

This work was supported in part by the Scadding-Morriston Davies Fellowship.

C M PARRY
Centre for Tropical Microbiology,
Liverpool University,
Liverpool L69 3BX, UK
O KAMOTO
A D HARRIES
Department of Medicine,
Queen Elizabeth Central Hospital,
PO Box 95,
Blantyre, Malawi
L NYONG'ONYA MBEWE
D S NYANGULU
Malawi National Tuberculosis
Control Programme,
Llongwe, Malawi

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Ultrasound assessment of diaphragmatic movement

I read the paper by Houston and colleagues (May 1994:**49**:500–3) with interest. A similar technique was used to answer the question "how does the diaphragm move in association with the induction of anaesthesia?". This question was stimulated by a study of three volunteers using lateral radiographs.[1] The reproducibility of the ultrasound measurements that were made was assessed by asking the operator to identify the precise time when the diaphragm passed through a particular position.[2] Volunteers were asked to breathe while holding their rib cage dimensions constant, so that the diaphragm position could be measured exactly from a spirometer. The ultrasound operator was able to judge diaphragmatic position with a standard deviation of 15, 39, and 56 ml in three volunteer subjects. I believe that these values, although obtained in a single operator, compare well with the reproducibility values in the more recent study.

In contrast to previous radiological observations in three subjects, the study using ultrasound indicated that motion of the diaphragm dome in a cranial direction, associated with the induction of anaesthesia, was very small. In eight out of 20 subjects no cranial motion was detected. Because the dimensions of the rib cage may also change at the induction of anaesthesia, exact inferences regarding the contribution of diaphragmatic movement to changes in lung volume in these circumstances cannot be made with certainty. It is possible that ultrasound, by allowing measurement of a specific part of the diaphragm, is a better method than radiography, where the image consists of several overlapping shadows.

In the study of Houston and colleagues the reduction in diaphragmatic movement per change in lung volume, observed when the subject moved from the supine to sitting position, presumably indicates the greater action of the diaphragm on the rib cage in that position.

GORDON DRUMMOND
Department of Anaesthetics,
Royal Infirmary,
Edinburgh EH3 9YW, UK

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Bordetella bronchiseptica pneumonia

De la Fuente *et al* (July 1994;**49**:719–20) report a case of pneumonia caused by *Bordetella bronchiseptica* in a patient with AIDS. Human disease, although rare, was reported as early as 1911 in a laboratory worker at the Royal College of Physicians, Edinburgh with an upper respiratory tract infection.[1] However, infection in association with AIDS may become a subject of increasing importance. The authors suggest exposure to ill animals should raise clinical suspicion. Although clinical disease has been reported in various animals from cats to skunks and racoons,[2] it should be noted that outwardly asymptomatic carriage occurs.

Unusual lung permeability measurements and evidence of minor foci of haemorrhage and inflammation in the lungs of rabbits supplied from a large commercial source led me to search for infection. All such animals grew *B bronchiseptica* from bronchoalveolar lavage fluid, though none showed clinical disease before death or evidence of other infection. Discussion with the suppliers revealed that screening of their stocks showed a 45% carriage rate although all appeared clinically healthy. Other laboratories using the animals had not experienced difficulties with the animals' overall health, suggesting that changes were local and subtle. The suppliers believe that carriage is easily transmitted and that its eradication would require complete restocking of their rabbits so they had no plans to attempt its elimination.

In patients with AIDS and other causes of immunosuppression with pneumonia a history of close contact with any mammal without regard to its health should be sufficient to raise suspicion.

R A LAWSON
Rayne Laboratory,
City Hospital,
Greenbank Drive,
Edinburgh EH10 5SB,
UK

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