Figure 1 The patient was instructed to monitor his peak expiratory flow (PEF) twice a day. The first 2 days after the third adalimumab administration he had a stable PEF of approximately 450 l/min. On the third day he developed dyspnoea, wheezing and a reduction in his PEF to 290 l/min. His PEF went back to baseline after 2 weeks.

volume in 1 s (FEV₁) reduced to 1.7 litres (46.8% of predicted) and FEV₁/forced vital capacity (FVC) ratio 55.9%. Since his RA responded favourably on adalimumab and there was not enough evidence to ascribe his respiratory complaints to this drug, it was decided to rechallenge him with adalimumab. During the third administration of adalimumab he still used prednisolone 20 mg/day and inhaled budesonide/formoterol twice daily. On the third day following adalimumab administration he developed dyspnoea, wheezing and a reduction in his peak expiratory flow (PEF) from 450 to 290 l/min (fig 1). These symptoms were less severe than at admission, probably because of concomitant treatment with corticosteroids. The prednisolone was increased to 50 mg/day and his PEF went back to baseline after 2 weeks. He resumed treatment with infliximab. His RA remained controlled and he did not suffer from bronchospasm again.

Bennett *et al* reported a patient with presumed adalimumab-induced asthma.¹ They hypothesised that once the tumour necrosis factor (TNF) α blocking adalimumab was introduced the T helper cell (Th)1 response characteristic for RA was suppressed, allowing the Th2-activated pathway to express itself as asthma.¹ However, patients with asthma have upregulation of the TNF α axis so one would expect suppression of asthmatic inflammation.^{2,3} Since Bennett and colleagues assumed that the pathophysiological mechanism causing adalimumab was a direct effect of the TNF α blockade, they suggested a class effect and decided not to treat their patient with other TNF α blocking agents.¹ Our patient used etanercept, adalimumab and infliximab within a short time frame and only reacted to adalimumab. This case therefore refutes the hypothesis that the asthmatic response is caused by blockage of TNF α , as well as the existence of a class effect. Symptoms

started 3 days after drug administration which is not compatible with anaphylaxis. A delayed-type T-cell mediated hypersensitivity reaction would be more likely.

At present it is unclear how often adalimumab has induced asthma-like symptoms. Because the use of adalimumab is increasing, this adverse event may become more prevalent in the future. We are the first to report adalimumab-induced bronchospasm with a positive rechallenge on the agent itself and negative challenges on etanercept and infliximab. We believe it is justified to make a switch to a different TNF α blocker under strict medical observation.

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Renal impairment in cystic fibrosis

We read with interest the paper by Bertenshaw *et al*¹ on the incidence of acute renal failure (ARF) in patients with cystic fibrosis (CF) and would like to supplement these results with our findings. With increasing survival and therefore consideration for lung transplantation, monitoring adult patients with CF for renal impairment is assuming increasing importance (creatinine clearance <50 mg/ml/min is a contraindication to lung transplantation).²

We examined the prevalence of renal impairment in our stable adult CF population³ in Birmingham using the Cockcroft-Gault formula⁴ for estimating glomerular filtration rate (GFR). Between 1 January 2004 and 9 February 2005, 207 of our 273 adult patients with CF had at least one serum creatinine measurement; eight were excluded from analysis owing to established renal failure, one had subnormal creatinine precluding analysis and in six cases the data were incomplete, leaving 192 patients for analysis. Using the Cockcroft-Gault formula and the United Kingdom National Kidney

Foundation definition of renal impairment (GFR <90 ml/min), 50 of the 192 patients analysed had renal impairment; 26 of these had a current infective exacerbation of which 12 were receiving intravenous antibiotics. Thus, excluding those in a current exacerbation and those with established renal failure, 24/192 (12.5%) of our patients with stable CF had renal impairment. This is probably a conservative estimate as we know from a study in Liverpool in a *Pseudomonas*-colonised CF population that 24-hour urine collection diagnoses more renal impairment than the Cockcroft-Gault formula.⁵ Only one of the 50 patients identified as having renal impairment by the Cockcroft-Gault formula had a raised serum creatinine level, which is often the only test that first draws our attention to the problem.

Our findings support the need for regular renal assessment in adult patients with CF using measures superior to raised serum creatinine levels alone (eg, at least using the Cockcroft-Gault GFR formula for all routine annual reviews) to enable early diagnosis while we await “firmer inferences to be drawn regarding the causation of ARF in CF and allow avoidable precipitating factors to be identified”.

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Is bronchodilation required routinely before diagnostic sputum induction? Evidence from studies with tuberculosis

Sputum induction (SI) by inhalation of nebulised hypertonic saline is an efficient