alternative treatments (i.e. increase in dosage, another DMARD, combinations of DMARDs and corticosteroids) when treatment does not have the desired effect. The comparator treatment in a cost-effectiveness study should reflect this.

Our second concern relates to the merging of data from all active treatment arms from the ATTRACT trial for estimating the effectiveness of infliximab and the use of only the treatment arm with the lowest dose, 3 mg/kg, for estimating the costs. Although no statistically significant difference between the active treatment arms in the ATTRACT trial could be demonstrated, there might be a clinically relevant difference. In this regard, absence of evidence is not equal to evidence of absence. Both the costs as well as the effects should preferably be based on the same population. Also the transition probabilities and the cost and utility values of the health states in a Markov model should be as context specific as possible.

The final concern relates to the assumption regarding the duration of the infliximab treatment and the assumptions made on the influence of stopping this treatment on the disease course in the basic model. It is not plausible that patients stop infliximab treatment after 1 yr, especially when the treatment has a beneficial effect. In this analysis it is also assumed that after stopping the infliximab treatment no ‘relapse’ occurs, but rather over a period of 9 yr equilibrium is formed in both treatment arms with a comparable distribution over the health states (as far as this can be concluded from the mean values in Figs 5 and 6). This also seems an unrealistic assumption, since, as the authors state, the HAQ is influenced by disease activity and by joint damage, and probably only the influence of the progression of joint damage on the HAQ might (partly) remain, and the disease activity might flare up after stopping treatment.

To incorporate such an effect loss the authors present an alternative model. However, in this alternative model the ICERs only marginally increased and using all costs the cost-effectiveness ratios were even more favourable (cost saving)—this result is counterintuitive. Apparently, in the alternative model, not only an effect loss was modelled but also the way of calculating the model was changed explaining the counterintuitive result. When it is intended to investigate the influence of an effect loss on the results of the study, one should not also change the calculation method. A figure of the mean HAQ scores using this approach (like Figs 4 and 5) would be helpful.

Making assumptions in modelling studies is unavoidable [3, 5]. However, for the results of (cost-effectiveness) modelling studies to be credible, assumptions should be made realistic or even conservative towards the experimental treatment and should be investigated transparently using univariate and multivariate sensitivity analyses to study robustness of the findings. This is an important approach to reduce the likelihood of bias in this kind of analysis [6].

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Dysphagia associated with lower thoracic spondylisis

Sir. A 73-yr-old man presented with several weeks of severe mid-thoracic back pain and dysphagia, which occurred only on assuming a supine position. The pain was likened to ‘being thumped by a truckload of watermelons’, and was associated with a sensation of struggling to swallow. Both symptoms were prominent in the recumbent position and settled promptly with change of posture.

His past medical history included seropositive rheumatoid arthritis diagnosed at age 50 and currently well controlled, left elbow prosthesis, inactive peptic ulcer disease, mild gastro-oesophageal reflux symptoms relieved with antacids, hypertension, hypercholesterolaemia, ischaemic heart disease and cerebral vascular disease. He was taking hydroxychloroquine and sulphalazine, was a non-smoker and did not drink alcohol nor use non-steroidal anti-inflammatories. Examination revealed a symmetrical deforming polyarthropathy with no active synovitis, and reduced movements of the cervical spine. Examination was otherwise unremarkable.

Imaging studies revealed thoracic spondylisis and diffuse idiopathic skeletal hyperostosis (DISH), with large osteophytes projecting anteriorly from the ninth and tenth thoracic vertebrae (Fig. 1A). In the supine position these osteophytes caused extrinsic oesophageal compression (Fig. 1B, C). We suggest that in this patient with spondylisis and DISH, the weight of the diaphragm...
and other thoracic organs during recumbence caused compression of the oesophagus against the large anterior osteophytes, causing pain and dysphagia. Symptoms settled following the initiation of a proton pump inhibitor and avoidance of lying flat, thus surgical treatment was avoided.

Anterior vertebral osteophytes of the mid- to lower cervical spine have previously been reported as a cause of extrinsic oesophageal compression and dysphagia in patients with spondylitis and/or DISH [1–3], with one case reported of oesophageal compression by a thoracic osteophyte at the level of the fourth thoracic vertebra (T4) [4]. It has been postulated that fixation of the oesophagus is a prerequisite for its extrinsic mechanical obstruction; in the cervical spine the cricoid cartilage prohibits forward movement of the oesophagus [5], whereas at the level of T4 the aortic arch lies anterior to the oesophagus [4]. The oesophagus is also potentially fixed where it penetrates the diaphragm at approximately T10, although this level can alter with posture and respiration. We hypothesize that in our patient, in whom radiological imaging demonstrated extrinsic oesophageal compression at this level, localized mucosal inflammation was likely to have exacerbated his symptoms, which were readily amenable to anti-reflux treatment.

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Methotrexate for rheumatoid arthritis: what should the patient be told?

Sir, Methotrexate (MTX) has become the disease-modifying anti-rheumatic drug (DMARD) of choice in treating rheumatoid arthritis (RA). Side-effects, however, are the major reason for cessation of therapy. The most serious complication is MTX pneumonitis (MTX-p), which is both unpredictable and poorly understood. We present a case highlighting the need for vigilance in all patients treated with MTX and explore the degree to which patients should be informed of possible adverse events.

A previously fit, 72-yr-old retired air force officer suffered from progressive seropositive erosive RA for a period of 7 months despite non-steroidal anti-inflammatory drugs (NSAIDs) and prednisolone at 10 mg daily. Owing to disabling disease the patient was given the Arthritis Research Council leaflet on MTX and was counselled regarding the risks and benefits of the medication. He was then commenced on MTX at 7.5 mg weekly with excellent symptomatic relief. Three months after the introduction of MTX he presented with a 3-day history of shortness of breath, dry cough and night sweats. On examination he had a respiratory rate of 24 breaths per minute, was cyanosed and febrile (38°C) with coarse crackles bilaterally. Full blood examination was normal, inflammatory markers were significantly raised and arterial blood gas analysis revealed hypoxaemia and hypocapnoea. A septic work-up prior to antibiotics was negative, as were viral antibody titres. Chest radiography showed a diffuse interstitial and alveolar infiltrate as did high-resolution computed tomography, with no evidence of granulomas or micronodules (Fig. 1). A baseline chest radiograph was normal. Transbronchial lung biopsy showed a mixed inflamma-