decided to apply categorisation of some continuous variables in our study. On the other hand, we should mention that this transformation of variables did not modify the results, as both age and the number of exacerbations behaved as independent prognostic factors on inclusion in the model as continuous variables. Specifically, in this predictive equation, age proved to be an independent prognostic variable with an OR of 1.06 (95% CI 1.01 to 1.11). The same applies to the number of exacerbations with an OR of 1.20 (95% CI 1.03 to 1.39).

Secondly, with regard to the role of age as a predictor of mortality, different studies involving both stable patients[2] and acute cases[3 4] have also found age to be an adverse prognostic factor. Despite such evidence, we consider the hypothesis suggested by Yohannes—that other age related and potentially modifiable variables would determine the prognostic effect attributed to age—to be very interesting. Unfortunately, in our analysis we did not include measures such as social support, physical disability, depression, or quality of life so we are unable to assess their specific weight. Almagro *et al*,[5] in a study that also explored mortality predictors after hospitalisation and which considered variables of this kind, found age to have a predictive value in the univariate analysis, but this effect disappeared in the multivariate study. Therefore, as suggested by Yohannes, it is probable that the effect of age may be minimised when other predictors that condition or define such an effect are included in the model.

In conclusion, age dichotomisation did not substantially change the results and conclusions drawn in our study. Re-analysis of the data using continuous (non-dichotomised) variables continues to suggest that severe exacerbations are independent predictors of mortality.

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Diaphragm paralysis after nephrectomy

We read with interest the case report by Moore *et al*[1] on the diaphragm weakness of two patients after anatomically distant surgery.

We are currently following a patient who had bilateral paralysis of the diaphragm after a nephrectomy for renal cancer. The patient, a 60 year old male non-smoker without any concomitant cardiac or lung disease, underwent surgery in August 2004 and immediately after the operation he complained of orthopnoea. Chest radiographs showed the elevation of both hemidiaphragms, which was not present preoperatively, along with a restrictive ventilatory defect detected by spirometry (TLC 61% predicted, VC 72% predicted, FEV_1 67% predicted, FEV_1/VC 70%). The diagnosis of bilateral paralysis was confirmed by electromyography and respiratory muscle strength assessment in October 2004. Because of a nocturnal oxygen desaturation, he started with nightly non-invasive ventilation. Up to now he has also undergone periodic courses of respiratory muscle training. In 2004 and 2005 he was checked regularly and an improvement in VC was found, but not in Pımax nor in TwPdi. Moreover, at the December 2005 check up the nocturnal oxygen desaturation had significantly improved and the patient had stopped the ventilation support.

Diaphragm paralysis is associated with renal cancer and is considered to be a paraneoplastic syndrome.[2 3] In our patient, however, the temporal link between the surgical operation and paralysis is evident. Moreover, during the operation and after the perioperative period our patient did not undergo central venous cannulation, nor did he experience any electrolyte disturbance. Postoperatively, the patient also underwent magnetic resonance imaging which excluded any injury to his spinal cord. The similarity between the case histories presented by Moore *et al*[1] and our patient therefore appears to be evident.

In addition, we think the patient's follow up is of interest. So far, the patient's VC has recovered 0.48 l, being 4.2 l and 86% of predicted value in orthostatism. Furthermore, VC now accounts for 2.3 l and 47% of predicted in clinostatism and can assure a normal oxygen saturation during sleep. However, the patient's diaphragm is still paralysed, since the TwPdi value is extremely low (3 cm H_2O) and the fall in VC from orthostatism to clinostatism is significant (45%). The recovery in VC might be due only to the increase in strength of the accessory inspiratory muscles, probably due to the respiratory muscle training courses. This finding further supports the recommendation by Moore *et al*[1] to measure the diaphragm strength separately from global inspiratory muscle strength in patients with raised hemidiaphragms after surgery.

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