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CORRESPONDENCE

Neither questions nor answers, just original data

To the Editor:

We have read with interest the letter by EWIG [1] in relation to our article [2]. According to his opinion, our study raises an unproductive debate about the usefulness of bronchoscopic sampling techniques in ventilator-associated pneumonia (VAP).

As it is well known, most of the studies dealing with the diagnostic efficacy of bronchoscopic sampling techniques in VAP include a large percentage of patients already on antibiotics when the procedure is carried out. Owing to the different nature of the antibiotic treatment used in these series, the variable "prior antibiotics" suppose an important bias for the interpretation of the microbiological results. Furthermore, only a few studies provide adequate information concerning the nature of the previous antibiotic regimens and, consequently, as it has been recently emphasised [3] in this setting, the interpretation of the microbiological data is usually complex.

We designed our study in order to obtain prospective data that could demonstrate the effect of an adequate antibiotic regimen on susceptible strains obtained by protected specimen brush technique, before antibiotic treatment and at different periods of time after the introduction of the antibiotic. So far, very few studies have used a similar approach [4]. Our results demonstrate that some bacterial species appear to be highly vulnerable to antibiotics (*Streptococcus pneumoniae, Haemophilus influenzae*), whereas other organisms (*Pseudomonas aeruginosa, Acinetobacter baumanii, Staphylococcus aureus*) are still viable 48–72 h after starting an active antibiotic treatment.

We do not believe that our data can be taken as an argument in the controversy about the usefulness of bronchoscopic sampling techniques in ventilator-associated pneumonia. They just demonstrate that in some cases, basically in the early onset ventilator-associated pneumonia, a very short course of an appropriate antibiotic can provide false-negative results, and this has to be taken into account when defining the therapeutic strategy. In addition, it is possible that a similar antibiotic effect can be observed when using samples obtained by more simple sampling methods, such as endotracheal aspirates, but this, of course, has to be confirmed.

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Muscle weakness after short course of steroids

To the Editor:

NAVA *et al.* [1] have shown acute weakness of respiratory and skeletal muscles after a short course of methylprednisolone given for acute lung rejection after transplantation. As many chest physicians commonly use other corticosteroids, it is interesting to compare equivalent doses. Over a 5-day course of methylprednisolone [1] a 70 kg male would have received ~3.9 g. This is approximately equivalent to 4.9 g prednisolone or 19.5 g hydrocortisone [2].

I have previously suggested there is a dose effect with hydrocortisone in causing myopathy in ventilated asthmatics, with weakness more likely if >5 g hydrocortisone was used [3]. Those patients were paralysed with neuromuscular blocking agents (which might predispose to myopathy), but severe weakness has also been described with 10.0 g hydrocortisone over 10 days in a nonparalysed ventilated asthmatic [4]. Methylprednisolone is often given in doses of 1.0 g and theoretically even 2 days treatment (equivalent to 10.0 g hydrocortisone) might be enough to cause weakness. NAVA *et al.* [1] have usefully highlighted the need to be aware of acute muscle weakness following high-dose steroids.

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From the author:

First of all we would like to thank C.D. Shee for the very useful comments. We agree with most of his conclusions except with the sentence "as many chest physicians commonly use other corticosteroids" (*i.e.* than methylprednisolone), since the large majority (three quarters) of the randomised controlled trials performed during an acute exacerbation of chronic obstructive pulmonary disease have employed methylprednisolone, though using different dosages [1, 2, 3].

As correctly stated by C.D. Shee, the daily dose at which respiratory muscle weakness and eventually myopathy occurs is critical, but this still needs to be clearly identified for the different kinds of corticosteroids (*i.e.* fluorinated and nonfluorinated) that may have different effects on the muscles when chronically administered at moderate doses [4]. However, when given acutely at massive doses, both fluorinated and nonfluorinated steroids may have similar effects on the contractile and histopathological properties of the diaphragm [5]. A difference between the deleterious clinical effects of low *versus* high doses of steroids is therefore likely.

Massive doses of steroids (*i.e.* methylprednisolone >500 mg·day^{-1}) are usually employed, apart from cases of acute lung rejection after transplantation, in severe asthma requiring mechanical ventilation. These patients, as highlighted by C.D. Shee, also receive a neuromuscular blocking agent (NMBA), and consequently it is difficult to assess the independent effect of each drug, and the interactive effect of concurrent use, even though it has been suggested that muscle weakness is limited to patients who had received both steroids and NMBA, rather than steroids alone [6]. Our recent study however, seems to partly contradict this statement, since none of our patients were taking NMBA [7].

From a clinical point of view we agree with C.D. Shee that we should be "aware of the acute muscle weakness problem" that has been shown to occur in 28% [7], 30% [8] and 46% [6] of the patients treated with massive doses of steroids.

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