To the Editor:

We are thankful for the letter from Drs. Clausen and Neuman that provides some meaningful supplements for the discussion about the value of lung function testing in the diving medical examination.

In our article,1 we showed medical history findings, preinjury and postinjury lung function, as well as radiologic findings of 15 consecutive patients who had suffered from diving-related pulmonary barotrauma. We found a considerable number of lung blebs when assessing CT of the chest that did not emerge from plain film radiology. Moreover, in some of our patients, a decrease of end-expiratory flow was apparent in spirometric performed preinjury. A comparison of preinjury and postinjury spirometric values between the 15 patients who suffered from pulmonary barotrauma, and another sample of 15 patients who had suffered from decompression sickness, revealed a significant difference with respect to the end-expiratory flow rates.

Drs. Clausen and Neuman criticize the lack of a Bonferroni correction that would drop the significances. When evaluating the patients’ records, a decrease of MEF25 was evident in some patients that stimulated us to further investigate lung function parameters in those patients with a clinical diagnosis of pulmonary barotrauma and in patients with decompression illness related to other causes. We assumed that a small airway dysfunction might have precipitated air trapping, and in consequence, burst lung during ascent. With respect to this particular hypothesis, it is justifiable to do statistics without a conservative Bonferroni correction.2 The relatively low overall incidence of pulmonary barotrauma, however, may limit prospective studies; our 15 patients were referred to us within a 6-year interval. In the meantime, another case of pulmonary barotrauma occurred at our institute where an end-expiratory flow limitation was apparent as well.3

Another major point of criticism is the conclusion that we drew from our lung function results. We are aware of the relatively great standard deviation of the MEF25. The demand for a value of > 80% of predicted may be somewhat overstated. However, it was our intention to raise the discussion about the meaning of the MEF25. We were not the first to report on small airway dysfunction in divers who had suffered from pulmonary barotrauma,4 and the importance of the assessment of small airways function for divers’ fitness has been stressed by outstanding specialists.5 Drs. Clausen and Neuman correctly state the factors that may limit the MEF25 as a screening test for diving candidates. However, renunciation of recommended values for MEF25 in the assessment for fitness to dive may bear the risk of not looking at small airway function, because most family physicians, who predominantly do the examinations, are not necessarily familiar with the interpretation of the shape of the flow-volume loop, and thus, may overlook a decrease of end-expiratory flows.

Finally, we agree that it may be premature to make a definitive decision about the meaning of expiratory flow rates in the assessment of fitness to dive. The question of whether asthmatic divers are at increased risk of pulmonary barotrauma is still unanswered, but these divers are somewhat overrepresented in diving fatalities6 and therefore, it seems worthwhile to carefully evaluate lung function when screening divers. Hopefully, more data will be provided in the near future that may ascertain the tendencies obtained from our study and those cited.

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REFERENCES

Tracheal Lacerations After Intubation

To the Editor:

We read with great interest the report by Kaloud and colleagues3 concerning iatrogenic ruptures of the tracheobronchial tree. We agree that it is an extremely rare condition, much more frequent in middle-age women. We agree about its posterior longitudinal localization on the membranous tracheal wall, and about the need for immediate fiberobronchoscopic evaluation once the clinical diagnosis has been made. We also agree with the cuff–related cause of injury and the inappropriate use of a stylet during endotracheal intubation as the main cause of tracheal laceration. We would suggest that a little cough in a patient with moderate sedation at the time of the intubation is also an easy way to cause a linear longitudinal tear in the posterior membranous part of the trachea.

We cannot support their statement that “an absolute indication for surgical repair is present whenever a transmural tear with a length exceeding 1 cm causes pneumothorax and/or pneumomediastinum.” We have recently published a case report4 of two female patients and a recent third case who presented with subcutaneous emphysema, pneumomediastinum, and pneumothorax shortly after single-lumen endotracheal intubation; bronchoscopic examination revealed a posterior linear tear of 2.5 cm, 4.5 cm, and 5 cm in each case. After evaluation of a stable patient status, we treated the first two conservatively without intubation and with placement of the tube distal to the lesion in the last case. There was no progression of the clinical and physical signs in the following days and the outcomes were excellent, without complications. A 2-month fiberobronchoscopic control revealed the lesions to be completely healed with a little granuloma in one patient that disappeared in a subsequent control.

Adding the 12 patients reported by Kaloud and colleagues to a review of the literature,3 of 48 cases of tracheal laceration after intubation, 40 were repaired surgically (83.3%) with a 20% mortality (eight patients). The eight patients published as conservatively treated all survived. We assume that this data is not from a homogeneous group, but our and others’ experience4–6 support the criteria to be used as guidelines in deciding on nonoperative management of postintubation tracheal lacerations proposed by Ross and colleagues3:

1. Stable vital signs.
2. No difficulty ventilating the patient while intubated or respiratory distress while extubated.
3. No evidence of esophageal injury.