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Effectiveness of cycling-off during pressure support ventilation: a reply

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Sir: We would like to thank Dr. Mojoli and Dr. Braschi for their valuable comments regarding our paper [1]. We totally agree with them that expiratory asynchrony is the rule during pressure support ventilation, as predicted by mathematical models [2]. However, the purpose of our study [1] was to examine patient-ventilator interaction during pressure support ventilation in critically ill patients when they were ventilated by two different methods of triggering and cycling and not to study the phenomenon of expiratory asynchrony. Recently, we have extensively examined expiratory asynchrony during pressure support ventilation [3, 4]. These studies showed that expiratory asynchrony increased progressively with increasing pressure support levels [3, 4]. It is of interest to note that expiratory asynchrony influenced—via a reflex feedback—the patient's neural expiratory time and thus breathing frequency; increasing expiratory asynchrony lengthens neural expiratory time and decreases breathing frequency [3, 4]. The authors correctly observed (using indirect data from our paper) that in the study of Prinianakis et al. [1] expiratory asynchrony increased progressively with increasing pressure support. Indeed, we calculated the actual time that mechanical inspiration extended into neural expiration (Text, an index of expiratory asynchrony), and found that independent of the method of cycling Text increased progressively with increasing PS, averaging 210 ms, 290 ms, and 680 ms with the traditional method of cycling (25% of peak inspiratory flow) and 200 ms, 290 ms, and 710 ms with the flow waveform method of cycling, respectively, during low, moderate and high levels of pressure support. The method of cycling did not influence the magnitude of expiratory asynchrony. Notwithstanding that in the study of Prinianakis et al. [1] chemical feedback was not taken into account these data reconfirmed our previous findings [3, 4].

We certainly agree with the mechanism of expiratory asynchrony discussed by Dr. Mojoli and Dr. Braschi. Indeed, we observed that at least in sedated critically ill patients increasing pressure support is not associated with increased expiratory muscle activity [1, 3, 4], exaggerating the phenomenon of expiratory asynchrony as Dr. Mojoli and Dr. Braschi correctly stated. Nevertheless, another mechanism may be of importance. We have shown that increasing pressure support by augmenting inspiratory flow decreases neural inspiratory time, thus lengthening Text [1, 3, 4]. It follows that increasing pressure support may increase expiratory asynchrony by affecting neural inspiratory time.

Collectively, these observations indicate that expiratory asynchrony during pressure support ventilation is due to multiple factors related both to patient and ventilator. We agree, however, with Dr. Mojoli and Dr. Braschi that the clinical impact of this expiratory asynchrony needs further study.

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The letter by Mojoli and Braschi can be found under 10.1007/s00134-004-2274-9

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