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P0.1, breathing pattern and pressure support ventilation

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Sir: We read with interest the paper by Alberti et al. on the response of the breathing pattern, P0.1 and work of breathing (WOB) to a progressive reduction in pressure support ventilation (PSV) level [1]. The paper shows that changes in patient load of ventilation are well reflected by changes in P0.1, as previously reported [2–4]. The authors explain this result with a good relationship between P0.1 and WOB, as previously reported [4, 5]. In contrast with much literature, including our own reports [2, 3], the authors also support the thesis that changes in the patient load of ventilation due to changes in PSV level are poorly reflected by changes in breathing pattern. We wonder whether this contrast may just be a matter of interpretation of data.

The results section of the paper states that the only significant change in breathing pattern was found when the PSV level was decreased from the baseline (PS 100) to 85% of the baseline (PS 85), and that it was limited to a decrease in tidal volume (Vt) and to an increase both in respiratory rate (f) and in f/Vt. No further significant change was observed for lower PSV levels. However, the tabulated ANOVA does not support these statements, and suggests that the role of the breathing pattern was even less significant. Indeed, a significant difference between groups is found only for f and f/Vt, but for the latter parameter the comparison between PS 100 and PS 85 indicates no difference. As regards Vt and minute ventilation (V'e), no significant difference between the groups is found, so that the comparisons between couples of groups lack meaning. In the discussion the argument takes yet another turn: "f, Vt, V'e, Ti/Ttot and f/Vt changed significantly from PS 100 and PS 85 without further significant changes with lower PSV levels".

In contrast with the breathing pattern, P0.1 is claimed to change progressively with decreasing PSV levels. This statement, too, lacks support in the statistic analysis, which indicates no significant change with the two lower PSV levels.

More and more confused, but still interested, we analyzed the figures. From scattergrams, we read f, Vt, P0.1 and WOB data for each patient and group, and then calculated V'e and f/Vt. We treated data with two-way ANOVA, as indicated by the authors. Our probability values for difference between all groups (f: 0.0001; Vt: 0.0074; f/Vt: 0.0001; V'e: 0.0187; P0.1: 0.0001; WOB: 0.0001) differed greatly from those in the paper. Although our data collection from the scattergrams was necessarily approximate, the statistical significance for all parameters was so strong as to be hardly affected by imperfect point location. With our analysis, the picture of what happened when the PSV level was withdrawn clarifies, and becomes consonant with the literature. Simply, Vt decreased, while f, f/Vt, P0.1 and WOB increased. Interestingly, as previously reported [2], V'e significantly increased too. This is explained by the increase in f, which caused an increase in dead space ventilation that required compensation.

We agree that P0.1 is very promising: it is simple to measure and correlates with patient WOB. However, this already documented finding should not lead the reader to conclude that Vt, and particularly f and f/Vt, lack high relevance for the setting of PSV.

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Reply

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Sir: We appreciated the profound interest with which Drs. Iotti, Braschi and Galbusera went through our paper in great detail [1], although we must disagree with most of their discussion, though not all. First, it is true that our conclusion about the usefulness of P0.1 as a reliable variable for bedside setting of pressure support ventilation (PSV) is in keeping with their recent results [2], though it is clear that the purpose as well as the approach of our study was different from theirs. It seem fair to state that these two studies support, from different perspectives, the use of P0.1 to tune PSV. Second, Iotti and colleagues assert accurately that the group by group comparison loses meaning when ANOVA is not significant, as was the case for VT and VE in our study (Table 2). Nevertheless we used the paired test to underscore the message that the patients' breathing pattern is influenced by the PSV level, as Iotti and colleagues themselves emphasized in their conclusions. We also agree that, at the two lowest levels of PSV, changes in P0.1, though present, were not significant. However it is noteworthy that, whereas P0.1 progressive-

ly increased from 100% to 85, 70, and 50% of the initial pressure support, the frequency do not change at all after PS85. The comparison between Figs. 3 and 4 clearly shows that the average changes in P0.1 nicely paralleled changes in WOB, whereas this was not the case for any variable of breathing pattern, as clearly illustrated by Figs. 1 and 2. Indeed, the regression analysis through the individual data points (Figs. 5–6) provided additional strength to the message. The different statistical results obtained by Iotti and colleagues on our data may be explained by two facts: i) the approximation of data collection from the scattergrams, as they also stated; ii) the Bonferroni's correction implemented in our analysis upon the reviewers' request. However, the major point of the debate is the interpretation of changes in the breathing pattern more or less associated with levels of PSV. The amount of PSV can be set primarily either to sustain a pre-selected value of VE (for example to improve the arterial blood gases) or to reduce the ventilatory load upon the patient's respiratory muscles. We agree with Brochard and colleagues [3] that the latter was the main goal for which PSV was introduced. Hence we assumed that the patient's WOB and not VE was the leading physiologic variable to tune PSV. Then, we found that P0.1 appeared to be a simple, non-invasive and reliable variable to set PSV at a level where both insufficient and excessive support could be avoided [4]. Neither changes in VT nor in breathing frequency could provide the same piece of information. And not only for statistical reasons. Indeed, variations of VT were limited by the fact that the increase in patient's WOB with decreasing PSV prevented any proportional reduction in VT and VE [5]. Moreover the respiratory frequency, although better related than VT to WOB variations at decreasing levels of PSV, does not reflect the overall timing of ventilation. Ti/Ttot is the other important variable. In fact, if the patient's inspiratory muscles relax just after having triggered the pressure boost, the passive lung inflation is included in the inspiratory time (Ti) from the ventilatory point of view, but in the expiratory time (Te) for the relaxed inspiratory muscles and more importantly for the neural respiratory centers that "ceased firing". If the time required to deflate the lungs is considered, the expiratory time for the centers becomes even longer. Under those circumstances, a clear discrepancy between the "central" and the "ventilatory" Ti/Ttot occurs, which might be incompatible with the frequency set by the "central controller" [6]. If that discrepancy becomes excessive, the respiratory centers try to gather the control on the timing of ventilation by inducing either expiratory efforts during the lung inflation or inspiratory efforts during the lung deflation [7]. Therefore, the patient's breathing pattern is influenced not only by the level of support, but also by the activity of the central controllers adjusting the central drive to the variations of the respiratory load. P0.1 provides, with well-known limitations, a measurement of neuromuscular drive independent of timing. In other words, the modifications of the breathing pattern during PSV represent the final result of the patient-ventilator interaction, whereas changes in P0.1 may more closely reflect the respiratory muscle activation, which is what one wants to know, according to the initial purpose.

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Transient post-renal obstruction and renal protection against nephrotoxic damage

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Sir: We read with great interest the report of Navis et al. [1] describing ipsilateral protection of an obstructed kidney against nephrotoxic damage. Our experience with a similar observation we published previously [2] raises some additional comments.

We agree with the authors that unilateral renal obstruction may protect the ipsilateral kidney against nephrotoxic factors. Although relatively scarce, experimental and clinical observations suggest that protection may be directed against various subtypes of acute renal failure including cortical necrosis [3], reversible acute tubular necrosis [2] and glomerulonephritis [4]. We also agree with them that disseminated intravascular coagulation could have been a major factor of non-reversible unilateral acute renal failure. Although renal biopsy was not performed, the absence of recovery of the left kidney is consistent with cortical necrosis. More than 25 years ago, a very close animal model of unilateral cortical necrosis was reported by Watchi et al. [5] using the Shwartzman-Sanarelli reaction.

However, Navis et al. did not consider the potential aggravating factor of post-renal obstruction on the contralateral kidney. Indeed, experimental models have shown that unilateral ureteral obstruction can induce a contralateral renal arterial vasoconstriction [6] that could precipitate severe necrosis. In their observation, the unusual severity of the non-obstructed kidney injury might support this hypothesis.

Finally, this privileged observation could be of value in view of further experimental studies, since it emphasizes the theoretical interest of unilateral ureteral obstruction models to investigate the involvement of renal mediators in acute renal failure.

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