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Airway Occlusion Pressure Revisited

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The use of airway occlusion pressure (P0.1) as a measure of respiratory drive was introduced by Whitelaw and colleagues 45 years ago based on two basic assumptions (1). First, in the absence of flow or volume change during the occlusion, pressure generated by the inspiratory muscles is transmitted directly (1:1 ratio) to the external airway. Second, if the occlusion is brief (i.e., 0.1 s), there is no time for behavioral responses to influence the pressure output of the i a constant brief time reflects t spontaneous inspiration, v piratory muscle activity, at l

A bsequently identified that can a electrical diaphragm muscle output in the absence of volume changes or behavioral responses. As noted by Whitelaw and

Derenne in 1993, these factors include the presence of dynamic hyperinflation, expiratory muscle activity, chest wall distortion, respiratory muscle weakness, neuromuscular junction blockade, and the shape of the inspiratory pressure waveform (2). All of these modifiers apply to critically ill patients. In addition, the method used to measure P0.1 in such patients is of critical importance depending on whether a true occlusion is implemented, whether measurements are made close to the patient or remotely in the ventilator, what type of triggering is used, and other technical factors. Almost certainly, because of the numerous variables that modify the relation between P0.1 and inspiratory muscle pressure output or drive, the results of P0.1 in weaning assessments, even when measured properly with specialized equipment, have been variable and generally not impressive. There is, however, evidence that an excessively high or excessively low respiratory drive in patients is an important risk factor for continued ventilator dependence (3, 4). Identifying such patients would be of clinical value because this might spare them from being subjected to unsuccessful weaning trials and point to the abnormality that needs to be addressed.

The use of specialized equipment to measure P0.1 in the ICU is a major deterrent to such studies because the setup, proper application of occlusion, and assessment of the quality of the results require considerable expertise. Several commercially available ventilators measure P0.1 and display the results on the ventilator screen. The methods used by these ventilators vary but do not include the desirable application of occlusion near the patient's airway, and some ventilators do not even apply a true occlusion. An important practical question, therefore, is whether the ventilator-generated P0.1 is an adequate surrogate for the more complex and demanding use of specialized equipment.

In a study presented in this issue of the *Journal*, Telias and colleagues (pp. 1086–1098) compared P0.1 estimated by different commercial ventilators (P0.1*vent*) with values obtained in the proper way (P0.1*ref*) in critically ill patients and in a bench test using a simulator (5). In addition, they determined the relation between P0.1 and the pressure–time product of the inspiratory muscles. Not surprisingly, there were good correlations between P0.1 and pressure output in individual patients, consistent with the fact that airway pressure is directly related to respiratory muscle output during occluded breaths. Also, as expected from the various known modifiers of the relationship between P0.1 and inspiratory muscle pressure, there was much scatter in this relationship among patients. There are several important novel findings from this study. First, P0.1 measured by ventilators that apply a true end-expiratory occlusion accurately reflects P0.1*ref* in bench testing, whereas ventilators that do not apply occlusion are inaccurate. Second, on average, P0.1 measured by the more accurate ventilators in patients has little systematic error (minimal bias), and therefore these average values can be used to evaluate the i lefined a high respiratory c sured by P0.1*ref*, and r *ef* in individual patients, F with reasonable accuracy. A li en validated, those of F

These results are encouraging in that they suggest that P0.1 displayed in select ventilators can be used to identify patients with abnormally high and low values. Some caveats remain, however. First, as the authors acknowledge, the thresholds used to set limits on high risk are derived from retrospective analyses of patients with weaning failure. It is not clear whether respiratory muscle output in these patients was the only or main reason for weaning failure. Second, the thresholds selected here apply to only a small fraction of the patients studied; in most patients, P0.1 was between the high and low thresholds, and in such patients the P0.1 results would be equivocal.

Knowledge in physiology in the interpretation of P0.1 is indispensable. In the extremes, as mentioned above, variables that modify P0.1 measurements may create conflicting results regarding the relationship between P0.1 (as an estimate of respiratory drive) and inspiratory muscle pressure output. In the presence of muscle weakness, high chest wall elastance, dynamic hyperinflation, or chest wall–abdominal paradox, a high respiratory drive associated with respiratory distress may yield low inspiratory muscle effort and P0.1 (6). Conversely, a low respiratory drive is not inevitably associated with low inspiratory muscle effort and low P0.1. For example, expiratory muscle recruitment in response to external positive end-expiratory pressure may be associated with high P0.1 despite a weak inspiratory effort (2). Important questions remain: *1*) what is the threshold of inspiratory muscle effort–induced injury, and *2*) does monitoring of P0.1 and inspiratory muscle efforts in critically ill patients receiving mechanical ventilation alter clinical outcomes? Future prospective studies will need to address these questions.

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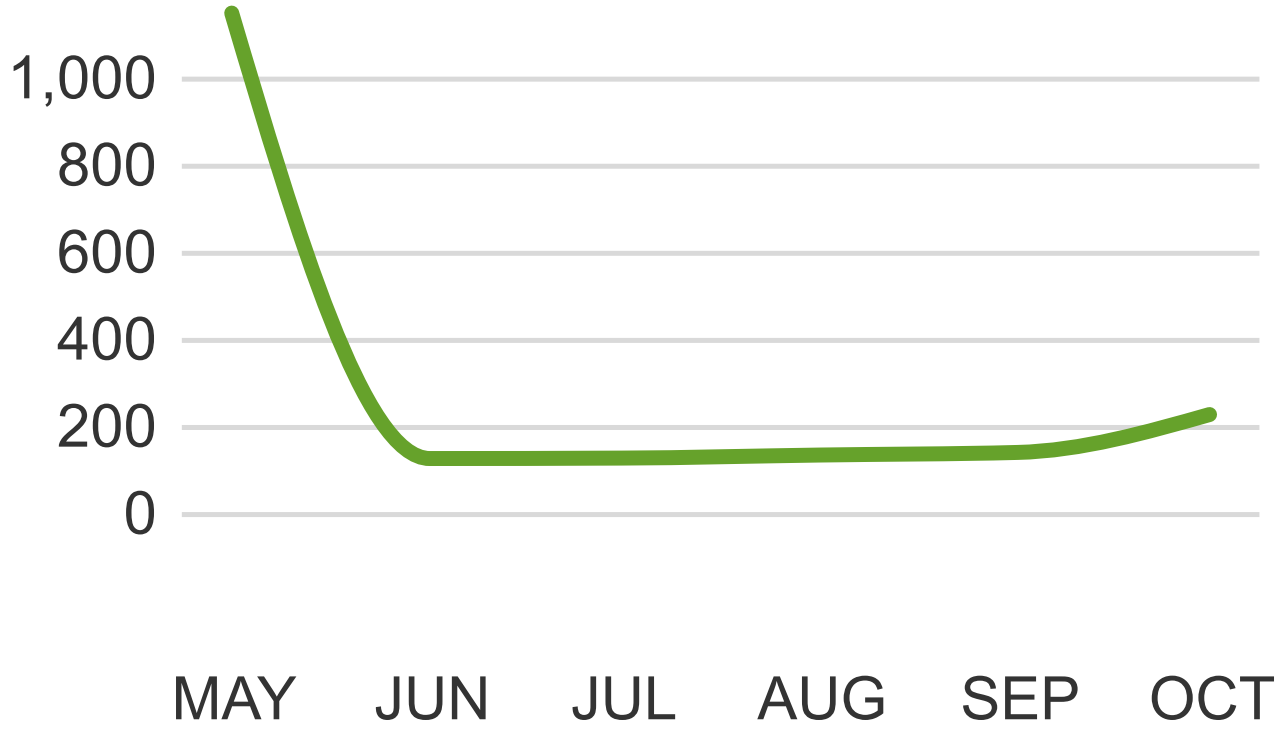

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