On using the pathophysiology of obstructive sleep apnea as a teaching tool

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I read with interest the recent article by Dr. Michael Levitzky on using the pathophysiology of obstructive sleep apnea (OSA) to teach cardiopulmonary integration (2). With 10 years of experience of teaching courses in both respiratory physiology and sleep at the University of Toronto, I also find that sleep-related breathing disorders, including OSA, provide a rich resource of material to integrate fundamental physiological principles. Importantly, these disorders also provide a focus for interesting case presentations to illustrate these integrative concepts. Indeed, in a previous issue of Advances in Physiology Education, we first introduced the use of sleep as a teaching tool for integrative respiratory physiology, and a major focus of that article was OSA (5).

I would like to make some comments on the useful article by Dr. Levitzky, and these are made in the spirit of advancing the field and promoting the effective and successful use of such examples of common sleep-related breathing problems in the teaching of integrative physiology. The lack of citation of our previous original and related contribution notwithstanding (5), it is important to stress that one of the main figures presented in the article by Dr. Levitzky (Fig. 4) contains a significant error and also a major source of ambiguity. Given that this figure will likely be used in any teaching activity related to that article, this error and ambiguity needs to be brought to the attention of any potential users of the material.

The figure in question (Fig. 4 in Ref. 2) contains a representative tracing of an OSA event. First, with respect to the ambiguity, this arises because the recordings of the chest and abdominal motions do not denote which is expanding with each inspiration and which is moving in the opposite direction (i.e., out of phase). This is an important point, not in the least because it is a critical step in the actual diagnosis of OSA. During an airway obstruction, the inward motion of the chest wall occurs in the face of outward motion of the abdomen. This scenario can be readily appreciated, for example, by an attempted inspiration against a simulated upper airway obstruction, such as with the nose and mouth closed. During each attempted inspiration, the abdomen increases in cross-sectional area, reflecting the normal displacement of the abdominal contents by contraction and descent of the diaphragm, but the rib cage gets sucked into the chest during breathing because the rib cage muscles are primarily responsible for producing effective ventilation, but in these patients the flaccid diaphragm is sucked into the chest during breathing and the abdomen signal decreases, i.e., opposite to the motions in OSA (5).

This discussion of out of phase chest and abdomen movements during the OSA event was introduced first as it also relates to a significant error that is apparent in a later segment of Fig. 4 (2). This figure shows that as the obstruction is released and effective breathing resumes, as indicated by the airflow signal, the out of phase movement of the chest and abdomen continues. This is incorrect because now both the abdomen and chest wall can expand with each inspiration, because the obstruction is released. This error in Fig. 4 (2) will be a significant source of confusion if this trace is used for teaching the pathophysiology of OSA. I do not want to overly promote our previous report as there is a voluminous literature on the mechanisms underlying the pathophysiology of OSA, but a description of the typical changes in physiological signals occurring during an obstructive apnea is shown in Fig. 8 of that report (5) and as well as in others (e.g., Refs. 1 and 6).

My final comment on the pathophysiology of OSA as discussed in the article by Dr. Levitzky (2) relates to the mechanisms underlying the collapse of the upper airway per se in response to subatmospheric inspiratory pressure. I understand the need for brevity and attention to all facets of the pathophysiology of OSA in that article (2), but the discussion of the mechanical causes of the actual upper airway obstruction that produces OSA was incomplete and only focused on inspiratory effort dependence, a focus that does not fully represent current concepts. Indeed, although the subatmospheric pressure generated by contraction of the respiratory pump muscles can reduce upper airway size, these pressures will generally not collapse the upper airway (6). In this respect, the upper airway is best modeled as a collapsible tube, with maximum flow (\(V_{\text{max}}\)) determined by upstream nasal pressure (\(P_N\) and resistance (\(R_N\)) (3, 4). Airflow ceases in the collapsible segment of the upper airway at a value of critical pressure (\(P_{\text{cri}}\)) (3, 4). \(V_{\text{max}}\) in the upper airway is determined by the following equation:

\[
V_{\text{max}} = \frac{(P_N - P_{\text{cri}})}{R_N}
\]

Based on this relationship, increases in \(P_N\) lead to increases in \(V_{\text{max}}\) in the upper airway, with this effect being the essential basis for nasal continuous positive airway pressure therapy in

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OSA. In contrast, decreases in $P_N$ decrease $V_{\text{max}}$, with this relationship being linear for the upper airway. Importantly, therefore, in terms of collapsing pressures in the upper airway, $P_{\text{crit}}$ is not a direct product of downstream (hypopharyngeal) pressure but rather the pressure upstream to the collapsing segment (1, 3, 6). Interestingly, these mechanical determinants of upper airway collapsibility have links to the collapsibility of veins and therefore offer another source of integrating OSA to cardiopulmonary integration. Of relevance physiologically, the $P_{\text{crit}}$ for upper airway closure is progressively more positive, i.e., indicating a more collapsible upper airway, from groups of normal sleeping subjects, to snorers, and to patients with OSA. Importantly, subjects in whom the upper airway is closed, or nearly closed, at pressures near (or above) atmospheric require upper airway muscle activation to permit adequate airflow and are therefore highly susceptible to OSA when muscle tone is decreased by sleep or certain drugs (e.g., anesthesia).

Overall, the article by Dr. Levitsky (2) offers many excellent examples of using the pathophysiology of OSA to teach cardiopulmonary integration. My comments are given to add to this contribution and promote a more complete use of such material.

REFERENCES