**Online Supplement**

*What is the contribution of respiratory muscle O2 uptake () to the amplitude of the slow component of O2 () kinetics?*

It is exceedingly difficult to quantify the precise fraction of  contributed by  during strenuous cycling. The primary reason for this difficulty is that there is no “gold-standard” method for directly obtaining  during exercise. Perhaps the closest method available is the voluntary ‘mimicking’ technique described by [Aaron *et al.* (1](#_ENREF_1)), whereby participants voluntarily ‘mimic’ oesophageal pressure-volume (PV) loops obtained during exercise, while seated at rest. The corresponding change in pulmonary O2 uptake measured during these mimicking trails is thought to reflect . Importantly, however, the validity of  obtained with this method depends heavily on the ability to control for end-expiratory lung volume (EELV) during mimicking trials.

The consequences of mismatching EELV during mimicking trials are that: (i) initial fiber-length of the inspiratory muscles are altered such that gross respiratory muscle efficiency is affected; and (ii) the resistive and elastic work expended per breath is different between mimicking and exercise trials. Indeed, the original study by [Aaron *et al.* (1](#_ENREF_1)) shows that despite efforts to control for lung volume (via visual inspection of end-expiratory oesophageal pressures), participants significantly underestimated their exercise EELV during mimicking trials ([See Table 2 in ref 1](#_ENREF_1)). Further to the above, the methods outlined by [Aaron *et al.* (1](#_ENREF_1)) describe that  is calculated from expired gases obtained after ~4–5 min of ventilatory mimicking. The steady-state in  observed at the end of such mimicking trials is certainly different from that incurred during the non-steady state conditions of strenuous exercise, for any given level of Pb. Lastly, it is unlikely that all respiratory muscle work expended during exercise is faithfully “mimicked” while at rest, even if the PV loops appear to overlap (c.f., chest wall distortion).

Without a confident estimate of respiratory muscle energetic expenditure, it is impossible to assign an absolute value to the contribution of  to the . In spite of this limitation, it is possible to estimate the relative difference in this “respiratory” contribution between two strenuous-intensity work rates, using only the information obtained from respiratory muscle power (Pb). Firstly, let us assume that the quantitative relations between , Pb and respiratory muscle efficiency (*e*resp) are approximated by:

  **Eqn. 1**

where *α* is the “heat equivalent” of O2 in kJ ([3](#_ENREF_3), [4](#_ENREF_4)) and ∆ signifies the change in values over the “slow component” phase. Accepting that *α* does not grossly affect the determined quantity, we then conclude that  is inversely related to Pb by the proportionality constant *e*resp. Following on from this, we compute the energetic contribution from the respiratory muscles to the  amplitude as:

  **Eqn. 2**

Substituting **Eqn. 1** into **2** after neglecting *α*, we find that:

  **Eqn. 3**

It is apparent from **Eqn. 3** that the energetic contribution from the respiratory muscles to the  is primarily determined by the ratio of ∆Pb/, scaled in magnitude by the inverse of *e*resp. According to the rationale outlined above, we may infer differences in the “respiratory” contribution to  amplitude between work rates by interpreting the ratio ∆Pb/ alone, provided that *e*resp remains constant (a conservative assumption). For this reason, we may evaluate the *difference* in the contribution of  to the  between heavy- and severe-intensity work rates without knowing *e*resp. We have attempted to illustrate this line of reasoning in **Figure A** below.

**Panel A** in **Figure A** presents the absolute contributions of  to the  for heavy and severe work rate transitions. These magnitudes were calculated using our values for ∆Pb and by assuming a range of hypothetical *e*resp. It can be seen from this panel that the absolute fraction of the  contributed by  is heavily influenced by *e*resp. **Panel B** illustrates that the absolute differences in the / ratio between heavy and severe cycling are likewise dependent on *e*resp. However, when this difference is expressed relative to the heavy-intensity bout of cycling, we see that the contribution of  to the  is consistently 8.7 ± 2.6 fold higher during severe cycling transitions, irrespective of the assumed value for *e*resp. Most importantly, we immediately arrive at this result by calculating the relative difference in the ∆Pb/ ratio between heavy and severe cycling bouts (i.e., the ratio is 8.7 ± 2.6 fold higher for severe work rate cycling). Thus, although we may not know the *absolute* magnitude of /, it may be stated that the fraction of the  contributed by  is necessarily higher for severe compared with heavy work rate transitions.

The only condition under which the above rationale does not hold true would be if *e*resp is in fact *higher* during severe compared with heavy cycling transitions. This is very unlikely for two reasons: 1) *e*respreportedly declines at higher (not lower) minute ventilations ([2](#_ENREF_2), [5](#_ENREF_5)); and 2) the requisite change in *e*respbetween work rates needed to falsify our conclusion is unreasonable. For example, if we take the very conservative estimate of *e*resp as 0.25 during severe-intensity exercise, the corresponding value for / would be 5.3 ± 0.9%. For this contribution to be significantly (*P* < 0.05) lower than that observed for heavy work rates, the value for *e*resp during heavy-intensity exercise must fall to less than 0.02! With the above in mind, we may therefore conclude that the energetic contribution from the respiratory muscles to the  amplitude is *at least* 8.7-fold higher for severe compared with heavy work rate cycling.

**Figure A. The contribution of respiratory muscle O2 uptake (****) to the amplitude of the slow component of O2 (****) during heavy and severe work rate cycling.** Values represent means ± *S.E.M.*\*Significantly different from heavy-intensity cycling trial, *P* < 0.001. See text for detailed explanation.

**References**

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