Last Word on Point:Counterpoint: Pulmonary edema does not occur in human athletes performing heavy sea-level exercise

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TO THE EDITOR: We appreciate the interest and input of the accompanying commentaries (see Ref. 7). However, we wish to restate that this Point:Counterpoint is related to human athletes performing sea-level exercise. Reports of edema with exercise at altitude or breath-hold diving, while interesting, represent an entirely different physiological scenario and are not central to the present debate. Furthermore, we are puzzled by the arguments used by some of our colleagues to support the case of exercise-induced pulmonary edema. What is the actual published evidence that sea-level exercise produces pulmonary edema? In our Counterpoint (3) we presented a summary of the many unsuccessful attempts to document pulmonary edema and we raised serious methodological concerns over the few studies that claim to have detected evidence of edema. In our opinion, interpreting these studies as “proof” of edema is based on an uncritical assessment of the collective data.

We agree with Dr. Hopkins’ (2) assertion that timing of any imaging method needs to be considered. It is suggested that those studies that have used post-exercise CT or MRI technologies occurred with such delay that any exercise-induced edema would have likely cleared (2). We concede that this is possible. On the other hand, it is argued that by using chest radiographs it is possible to detect edema 33 min postexercise (5). For edema to be visible to the viewing radiologist, the amount of extravascular fluid must be substantive. For example, in anesthetized canines, pulmonary vascular permeability was experimentally increased (4). Extravascular lung water needed to be increased by more than 37% from control and only then did chest radiographs begin to show signs of pulmonary edema. Moreover, this amount of fluid accumulation was accompanied by decreases and increases in the arterial partial pressures of O₂ and CO₂, respectively. This is not the case in humans following exercise and further argues against exercise-induced accumulation of lung water at least to the degree that could be viewed by radiography. We have difficulty with the argument that the development of exercise-induced edema would be of sufficient magnitude that it could be visible on a chest radiograph 33 min postexercise (5) and yet not at all detectable 48 min postexercise using much more sensitive and quantitative methods (1). Note that not one subject in this study demonstrated the slightest increase in lung density (see Ref. 1, Fig 1). We respectfully suggest radiographic reports of edema (5) likely reflect an unintentional bias during the subjective viewing of thoracic images rather than a true index of extravascular lung water. If transient interstitial edema does exist, the weak link is at the pulmonary microvascular level, as noted in the commentaries; however to date, technology is unable to quantify these changes in the human model.

Finally, we disagree that the findings from multiple studies using very different methods and experimental conditions (including hypoxia) can be compiled into a pseudo meta-analysis (6) and argued as evidence of sea-level edema. The calculations of “prevalence of edema” from such an approach is, in our view, not appropriate.

REFERENCES


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