TO THE EDITOR: Amann et al. (1) tested a fine and worthwhile hypothesis, but their results may raise more questions than are answered.

They show that intrathecal fentanyl produced a small reduction in minute ventilation and significantly raised PETCO₂ during 100–325 W leg exercise. They propose that since PETCO₂ rose, this masked a further fentanyl-induced fall in minute ventilation. They quantify this further fall by applying a novel calculation on minute ventilation using the measured CO₂ sensitivity value from the same subjects at rest. Three points could be considered further.

1) Publications show CO₂ sensitivity during exercise either increasing, not changing, or decreasing vs. that measured at rest. The METHODS section does not mention any CO₂ sensitivity measurements made during exercise. Do they have any data measuring CO₂ sensitivity during exercise in these same subjects? If not, what is the justification in their novel calculation of choosing to apply the resting CO₂ sensitivity value to breathing during exercise?

2) Might the significant PETCO₂ rise during both leg (Fig. 2E) and arm exercise (Table 3) after intrathecal fentanyl indicate that intrathecal fentanyl does produce a decrease in CO₂ sensitivity that is detectable during exercise but undetectable at rest (except in the 2 additional subjects that were then excluded)?

3) If this novel calculation is valid, surely it is equally valid to apply it on minute ventilation during the significant PETCO₂ rise found during 50 and 75 W arm exercise? The numerical results of deliberately applying it during arm exercise could then be given greater consideration in their debate on whether fentanyl has any apparently central effects.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author.

REFERENCE