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LETTER TO THE EDITOR

Central command activation and the dive response in marine mammals

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TO THE EDITOR: The recent study by Araújo et al. (1) reports that the activation of central command during voluntary exercise does not inhibit the bradycardia induced by trigeminal stimulation, but rather helps to maintain, and in some cases enhance, bradycardia relative to rest. In contrast, when the volitional component of exercise is removed (via passive exercise or the cold pressor test), the bradycardic response is attenuated. These findings indicate that exercise per se does not override the diving response and that central command plays a role in facilitating or sustaining vagal control of heart rate. This observation has direct relevance for interpreting cardiovascular control in marine mammals and, in this letter to the editor, we aim to highlight its implications for the field of marine mammal diving physiology.

In freely diving pinnipeds and cetaceans, heart rate is typically reduced below resting levels during dives and decreases further during longer dives despite active swimming (2). This apparent contradiction between diving-induced parasympathetic control and exercise-associated sympathetic activation has been framed as an "autonomic conflict," which may promote cardiac instability and increase the occurrence of arrhythmias during dives (3, 4). ECG data from Weddell seals and dolphins have been interpreted within this framework, where ectopic beats during diving are attributed to a hypothetical sympathetically driven exercise response superimposed on a strong diving bradycardia (4). A proposed alternative mechanism is that of "accentuated antagonism," whereby cardiac control is dominated by parasympathetic input while sympathetic tone is maintained, and may even increase, but its chronotropic effects are functionally suppressed by concurrent vagal inhibition (5). Consequently, adjustments in heart rate during exercise are thought to be achieved through vagal modulation rather than changes in sympathetic tone. Under this model, the arrhythmias commonly observed in diving marine mammals reflect dynamic autonomic regulation rather than physiological conflict or pathology (6).

The findings of Araújo et al. (1) provide an intriguing extension of this idea. If central command can enhance or sustain vagal activity during exercise, then locomotor effort during a dive would not necessarily oppose the mechanisms underlying diving bradycardia. Instead, exercise and the diving response may act in a coordinated manner, allowing simultaneous regulation of cardiac output, tissue perfusion, and oxygen conservation. Under this proposed framework, increased sympathetic activity during exercise could help support vascular control, whereas central command-mediated input helps maintain vagal activity to sustain the bradycardia. Given dolphins' and seals' ability to modulate their heart rate in anticipation of dives of varying durations (7), central command activation could, in theory, be tied to conditioned control of heart rate. Rather than representing a physiological conflict, the low heart rates observed during marine mammals' longest dives, despite exercise, may be modulated in this manner to promote reliance on muscle-bound oxygen (e.g., myoglobin) and optimize whole body oxygen use (8).

Despite the mechanistic insight provided by Araújo et al. (1) that central command activation reinforces bradycardia, it is notable that the dominant heart rate response to exercise on shorter dives is a transient increase in heart rate. Though heart rate may increase temporarily while animals exercise during diving, it still remains bradycardic, or below resting values, during the entire submersion (4, 9). Evidence from both experimental and free-ranging studies suggests that these transient elevated heart rates are caused by reductions in vagal tone rather than by a dominant sympathetic chronotropic response. During particularly shallow diving when vagal tone is likely to be more moderate, some marine mammals exhibit a moderate dive response with dive heart rates that resemble that of maintained, rather than attenuated or intensified, bradycardia during exercise (10), as seen under trigeminal nerve stimulation during moderate exercise in the study by Araújo et al. (1). In this case, sympathetic afferent stimulation may be masked either by overriding parasympathetic activity or, as highlighted in this study, by the effect of central command activation. During the longest dives of marine mammals, when vagal tone is expected to be the highest, the dive response is similarly minimally affected by exercise. Taken together, it appears that when vagal tone is both fairly low and at its highest, exercise does not modulate bradycardia, likely either as a product of central command activation or complete parasympathetic override. At moderate parasympathetic activation, exercise results in exercise-modulated, vagally driven increases in heart rates. Thus, the cardiovascular adjustments associated with exercise during diving of marine mammals may largely reflect graded vagal modulation superimposed upon the counter regulation of central command activation and sympathetic afferents.

Marine mammals' physiological studies often lack the measurements needed to fully assess the mechanisms regulating cardiovascular function during diving. As a result,

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heart rate has been one of the few variables collected consistently across species and has frequently been used to infer changes in autonomic control and perfusion. Araújo et al. (1) demonstrate an experimental design whereby heart rate patterns alone provide experimental evidence that voluntary exercise may enhance, rather than attenuate, vagally induced bradycardia. Though the study lacks breath-holding as part of the experimental design, we suspect that a detectable effect of central command activation may remain under the conditions of a breath-hold, given the dominant effect of trigeminal stimulation on the bradycardic response. When interpreted in a broader physiological context, these findings offer a potential mechanism for understanding the low heart rates associated with active swimming in marine mammals. We propose that this study has implications that extend beyond human physiology and may improve and inspire future studies of cardiovascular control in diving mammals.

DISCLAIMERS

The content and any opinions expressed in this article are those of the authors and do not necessarily represent the views of the American Physiological Society.

DISCLOSURES

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AUTHOR CONTRIBUTIONS

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REFERENCES

1. **Araújo MS**, **Lima AS**, **Guerrero RVD**, **Vianna LC.** Central command activation during exercise is an essential mechanism to sustain diving bradycardia. *J Appl Physiol 985)* 139: 355–364, 2025. doi:10.1152/japplphysiol.00274.2025.
2. **Kooyman GL**, **Campbell WB.** Heart rates in freely diving Weddell seals, Leptonychotes weddelli. *Comp Biochem Physiol A Comp Physiol* 43: 31–36, 1972. doi:10.1016/0300-9629(72)90465-3.
3. **Shattock MJ**, **Tipton MJ.** 'Autonomic conflict': a different way to die during cold water immersion? *J Physiol* 590: 3219–3230, 2012. doi:10.1113/jphysiol.2012.229864.
4. **Williams TM**, **Fuiman LA**, **Kendall T**, **Berry P**, **Richter B**, **Noren SR**, **Thometz N**, **Shattock MJ**, **Farrell E**, **Stamper AM**, **Davis RW.** Exercise at depth alters bradycardia and incidence of cardiac anomalies in deep-diving marine mammals. *Nat Commun* 6: 6055, 2015. doi:10.1038/ncomms7055.
5. **Signore PE**, **Jones DR.** Autonomic nervous control of heart rate in muskrats during exercise in air and under water. *J Exp Biol* 199: 1563–1568, 1996. doi:10.1242/jeb.199.7.1563.
6. **Ponganis PJ**, **McDonald BI**, **Tift MS**, **Williams CL.** Heart rate regulation in diving sea lions: the vagus nerve rules. *J Exp Biol* 220: 1372–1381, 2017. doi:10.1242/jeb.146779.
7. **Fahlman A**, **Burggren W**, **Milsom WK.** The role of cognition as a factor regulating the diving responses of animals, including humans. *J Exp Biol* 227: jeb246472, 2024. doi:10.1242/jeb.246472.
8. **Davis RW**, **Kanatous SB.** Convective oxygen transport and tissue oxygen consumption in Weddell seals during aerobic dives. *J Exp Biol* 202: 1091–1113, 1999. doi:10.1242/jeb.202.9.1091.
9. **McDonald BI**, **Tift MS**, **Hückstädt LA**, **Jeffko M**, **Ponganis PJ.** Stroke effort and relative lung volume influence heart rate in diving sea lions. *J Exp Biol* 223: jeb214163, 2020. doi:10.1242/jeb.214163.
10. **McDonald BI**, **Elmegaard SL**, **Johnson M**, **Wisniewska DM**, **Rojano-Doñate L**, **Galatius A**, **Siebert U**, **Teilmann J**, **Madsen PT.** High heart rates in hunting harbour porpoises. *Proc Biol Sci* 288: 20211596, 2021. doi:10.1098/rspb.2021.1596.