AMERICAN JOURNAL OF PHYSIOLOGY
REGULATORY, INTEGRATIVE AND COMPARATIVE PHYSIOLOGY

Am J Physiol Regul Integr Comp Physiol 322: R389–R399, 2022.
First published February 24, 2022; doi:10.1152/ajpregu.00004.2022

RESEARCH ARTICLE

No evidence for pericardial restraint in the snapping turtle (*Chelydra serpentina*) following pharmacologically induced bradycardia at rest or during exercise

Brandt Smith,[1] Dane A. Crossley 2nd,[1] Tobias Wang,[2] and William Joyce[2]

[1]*Department of Biological Sciences, University of North Texas, Denton, Texas and* [2]*Department of Biology—Zoophysiology, Aarhus University, Aarhus C, Denmark*

Abstract

Most animals elevate cardiac output during exercise through a rise in heart rate (f_H), whereas stroke volume (V_S) remains relatively unchanged. Cardiac pacing reveals that elevating f_H alone does not alter cardiac output, which is instead largely regulated by the peripheral vasculature. In terms of myocardial oxygen demand, an increase in f_H is more costly than that which would incur if V_S instead were to increase. We hypothesized that f_H must increase because any substantial rise in V_S would be constrained by the pericardium. To investigate this hypothesis, we explored the effects of pharmacologically induced bradycardia, with ivabradine treatment, on V_S at rest and during exercise in the common snapping turtle (*Chelydra serpentina*) with intact or opened pericardium. We first showed that, in isolated myocardial preparations, ivabradine exerted a pronounced positive inotropic effect on atrial tissue but only minor effects on ventricle. Ivabradine reduced f_H in vivo, such that exercise tachycardia was attenuated. Pulmonary and systemic V_S rose in response to ivabradine. The rise in pulmonary V_S largely compensated for the bradycardia at rest, leaving total pulmonary flow unchanged by ivabradine, although ivabradine reduced pulmonary blood flow during swimming (exercise × ivabradine interaction, $P < 0.05$). Although systemic V_S increased, systemic blood flow was reduced by ivabradine both at rest and during exercise, despite ivabradine's potential to increase cardiac contractility. Opening the pericardium had no effect on f_H, V_S, or blood flows before or after ivabradine, indicating that the pericardium does not constrain VS in turtles, even during pharmacologically induced bradycardia.

activity; cardiovascular; ectotherm; reptile; Testudines

INTRODUCTION

Regulating cardiac output (i.e., systemic blood flow; $\dot{Q}_{sys}$), the product of heart rate (f_H) and stroke volume (V_S), is essential to sustain aerobic metabolism when oxygen consumption increases. During exercise, vertebrates typically oxygen demand, a rise in f_H is energetically less efficient than to increase V_S (17–21), as could be attained via the Frank-Starling mechanism. However, exercise tachycardia may be necessary to increase $\dot{Q}_{sys}$ if V_S were to reach its maximum under an unchanged f_H. In dogs with atrioventricular block, permitting the precise control of f_H