Heart Rate and Hemolymph Pressure Responses to Hemolymph Volume Changes in the Land Crab *Cardisoma guanhumi:* Evidence for "Baroreflex" Regulation

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Accepted 9/16/88

Abstract

*Hemolymph volume in restrained land crabs (*Cardisoma guanhumi*) was altered in 5% increments by rapid infusion of filtered seawater into, or rapid withdrawal of hemolymph from, an infrabranchial sinus. Associated changes in intracardiac hemolymph pressure and in heart rate were measured. Elevation of hemolymph volume resulted in a brief (<20 s) rise in hemolymph pressure followed by a return toward control levels. Heart rate changed in a reciprocal fashion to hemolymph pressure, briefly decreasing during and immediately following infusion and then rising back toward control levels. The net effect after infusion or withdrawal was a readjustment in both hemolymph pressure and heart rate to stable new levels. A significant inverse correlation between heart rate and hemolymph pressure was evident, with heart rate decreasing approximately 2 beats · min^{-1} · $mmHg^{-1}$ rise in hemolymph pressure. Collectively, these data indicate that changes in hemolymph pressure result in reflex adjustments in heart rate in a manner analogous to baroreceptor reflexes in vertebrates.*

Introduction

The arthropod circulation historically has been regarded as a simple, low-pressure, "open" system, in which hemolymph slowly courses through a

Physiological Zoology 63(1):167–181. 1990. 0031-935X/90/6301-8844$02.00

system of vessels and sinuses. This view is now being challenged by repeated demonstration of high hemolymph pressures, rapid circulation times through "capillary" networks, and complex cardiorespiratory responses to hypoxia or exercise in a diverse array of arthropods (Blatchford 1971; Fyhn, Petersen, and Johansen 1973; Stewart and Martin 1974; Belman 1976; Burnett, deFur, and Jorgensen 1981; Wilkens 1981; McMahon and Wilkens 1983; Burggren et al. 1985; Burggren and McMahon 1988).

While the most basic of hemodynamic properties are becoming understood, particularly in the circulation of decapod crustaceans, very little is known of cardiovascular reflexes that might modulate changes in circulatory performance (McMahon and Wilkens 1983; Burggren and McMahon 1988). Yet, by even the most conservative of analogies with vertebrate cardiovascular systems (Bagshaw 1985), it is likely that mechanoreceptors responding to deformation or stretch in vessels are involved in reflex regulation of hemolymph pressure and flow in decapods.

The intent of the present study was to seek evidence for reflex regulation of hemolymph pressure in the open circulatory system of the land crab *Cardisoma guanhumi*. Blood pressure in any circulatory system, open or closed, will vary with cardiac output and peripheral resistance. We thus hypothesized that experimentally induced changes in hemolymph volume and the associated changes in hemolymph pressure might reflexly stimulate changes in heart rate appropriate for stabilizing cardiovascular dynamics.

Material and Methods

Experiments were performed on 13 *Cardisoma guanhumi* captured on the Atlantic coast of Panama near the Galetta Marine Laboratory and were transported to and maintained at the Naos Marine Laboratory, Smithsonian Tropical Research Institute, on the Pacific coast of Panama. All experiments were performed at 30° ± 2°C.

Recording Techniques

Immediately before experimentation, each crab was firmly restrained with elastic bands affixed to a large ceramic plate. The restrained crab was placed in a shallow aquarium containing sufficient aerated brackish water (50% SW) to cover just the limb bases. A shallow 1-mm-diam hole was drilled in the carapace over the heart. A 1-cm square of rubber dam was glued over the hole, providing a seal through which a polyethylene catheter could be introduced (McDonald, McMahon, and Wood 1977). A 40-cm-long PE 160 cathe-

ter fitted with a PE 60 tip was used for recording intracardiac pressure. The tip of this catheter was introduced approximately 3 mm through the carapace into the lumen of the heart. This catheter was filled with filtered seawater and attached to a Narco P1000B pressure transducer and a Narco Mk IV rectilinear recorder. The pressure signal from the transducer was also sent to a Narco Biotachometer, which computed and displayed instantaneous heart rate (f_H) on the recorder.

To provide a route for hemolymph volume adjustment, the tip of a 23-gauge stainless needle fitted into a PE 60 catheter was carefully inserted through a rubber membrane glued to an arthrodial membrane at the base of the second walking leg on the crab's right side and was guided through the leg sinus superiorly into the infrabranchial sinus. Positioning of the catheter, which was relatively straightforward given the size and shape of these crabs, was confirmed by postmortem dissection in preliminary experiments.

The rubber membranes at the sites of cardiac and infrabranchial catheter entry formed efficient leakproof seals. The point at which the catheter penetrated the rubber membrane was viewed frequently to ensure that no hemolymph leakage was occurring, and accidental hemolymph loss was negligible in most preparations. Visible hemolymph loss suddenly developed during the experiments in a few crabs, and subsequent data from these animals were discarded. Importantly, the frequency and length of pause of the scaphognathite motion as well as cardiac frequency in restrained crabs fitted with cardiovascular cannulae were not significantly different ($P > 0.1$, t-test for independent means) from those values recorded in restrained crabs without cannulation of the cardiovascular system, as detected by noninvasive impedance techniques. This suggests that invasive vascular cannulation was not disruptive to respiratory or cardiac function in restrained crabs. Clearly, however, the crabs were not in a "resting" condition owing to their restraint.

The catheter in the infrabranchial sinus was attached via a three-way stopcock to a 10-cm^3 glass syringe filled with filtered seawater, which allowed controlled withdrawal of hemolymph from, or infusion of seawater into, the infrabranchial sinus. Small differences in osmolality and ionic composition between filtered seawater and the hemolymph of each crab were not corrected for, especially since these parameters are normally quite variable in the short term both within and between individuals in *C. guanhumi* and other brachyuran land crabs (see Greenaway 1988). In any event, preliminary experiments involving removal of 5% and 10% hemolymph volume and immediate replacement with filtered seawater caused no apparent long-term changes in f_H or hemolymph pressure.

Transient adjustments in hemolymph pressure and f_H lasting a few sec-

onds were often associated with locomotor movements or with the brief periods of reversed scaphognathite beating, which are characteristic of *Cardisoma* (Burggren et al. 1985) and aquatic brachyurans generally (McMahon and Wilkens 1983). Consequently, infusions or withdrawals were initiated only on quiescent animals during continuous forward scaphognathite pumping. In order to avoid starting an experimental injection or withdrawal during reversed beating, scaphognathite beat was monitored. A pair of 40-gauge copper wires were inserted into one of the exhalant canals. These electrodes were connected to a Biocom Impedance convertor, whose meter display clearly revealed the status (forward or reverse) of scaphognathite beating.

Experimental Protocol

After control levels of hemolymph pressure and f_H were recorded for 2–4 h, experimental manipulation of hemolymph volume was begun. Initial hemolymph volume for each crab was calculated from total body weight and a total hemolymph volume assumed to be 30% of body weight. Subsequently gathered data for this genus by M. G. Wheatly (unpublished) indicate a mean hemolymph volume of 26% body weight. Hemolymph volume was increased a total of 10%, by two 5% infusion increments each applied steadily over a 10–15-s period and separated by an interval of approximately 1 min. A 1-min interval was more than adequate for a postinjection stabilization of hemolymph pressure and f_H. Hemolymph volume was then restored to control levels by two equal 5% withdrawals of hemolymph performed over the same time course as described for withdrawal. Using the same timing and sequencing, we then reduced hemolymph volume to 5% below control levels, followed after approximately 1 min by reinfusion of withdrawn hemolymph back up to control levels of hemolymph volume. Preliminary experiments indicated that repeated cycles of withdrawal of hemolymph and infusion with seawater eventually generated hemolymph clots in the infrabranchial catheter as well as produced hemodilution. Thus, only data for the first infusion series above the original hemolymph volume, followed by the first withdrawal series below original volume, were analyzed. Occasionally, spontaneous locomotor movements or changes in hemolymph pressure associated with reversed scaphognathite beating resulted in transient distortion or complete loss of the heart-rate trace emanating from the biotachometer or of the hemolymph pressure record measured by the pressure transducer. Thus, in some crabs heart-rate data for the complete protocol are not available. Finally, in four crabs hemolymph volume was reduced by 15% to examine the effects of severe hemolymph loss.

Data Analysis

Only stabilized, steady-state values recorded 30–60 s after experimental manipulation of hemolymph volume, rather than the transient values during or immediately following withdrawal or infusion, were included in the data analysis. Linear regression of hemolymph pressure on hemolymph volume and of f_H on hemolymph volume was performed by the method of least squares. To assess possible hysteresis effects associated with the experimental protocol, we used analysis of variance (ANOVA) to determine the effects of hemolymph volume adjustments on intracardiac systolic pressure and, separately, on f_H. Where significant treatment effects were indicated by ANOVA, *t*-tests for paired comparisons were used to assess the significance of any differences between treatment means.

Results

Control Values

Intracardiac systolic and diastolic pressures in restrained *Cardisoma guanhumi* in the 2–4-h period prior to any manipulation of hemolymph volume were 14.1 ± 3.2 mmHg and 5.5 ± 3.6 mmHg (±1 SD; n = 13 crabs), respectively. The f_H was 126 ± 54 beats · min^{-1}. These data agree well with previously reported values for this species under restraint at similar temperatures (Burggren et al. 1985).

Patterns of Hemolymph Pressure Response

Infusion of seawater or withdrawal of hemolymph had highly reproducible effects on intracardiac systolic, diastolic, and pulse pressures. The response of intracardiac pressures to hemolymph volume change usually had two phases. Infusion of a 5% hemolymph volume increment usually produced a large transient rise of 3–8 mmHg in intracardiac pressures coincident with injection (figs. 1, 2). Immediately after infusion was stopped intracardiac pressures began to decrease back toward preinfusion levels, typically stabilizing within 5–10 s at a level 1–5 mmHg higher than preinfusion levels (fig. 1). In a few infusion experiments, however, hemolymph pressure stabilized at preinfusion levels with no apparent effect on either systolic or diastolic pressure (fig. 2). Generally, systolic and diastolic pressure changed in parallel during infusions, with pulse pressure remaining unaltered at about 5–10 mmHg.

This general pattern and magnitude of change in intracardiac hemolymph

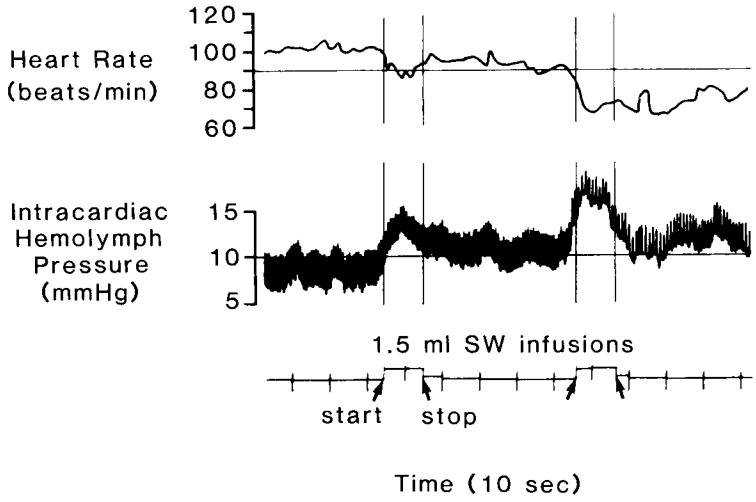

Fig. 1. Adjustment in intracardiac hemolymph pressure and heart rate in response to infusion of seawater into the circulation in a restrained, 90-g Cardisoma guanhumi. *Each infusion, which was delivered into the infrabranchial sinus during the period indicated by the displacement of the time marker, was 1.5 mL (5% of estimated hemolymph volume). The hatch marks on the time marker are in 10-s intervals in this figure (and all subsequent figures showing actual records).*

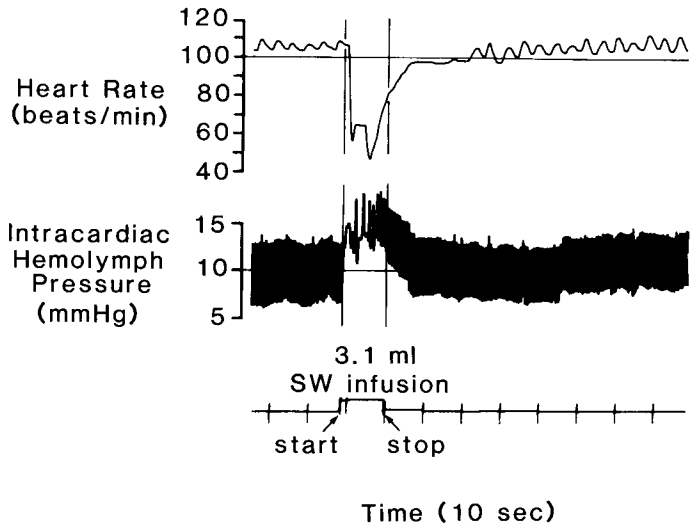

Fig. 2. Adjustment in intracardiac hemolymph pressure and heart rate in response to infusion of seawater (equivalent to 5% hemolymph volume) into the circulation in a restrained, 207-g Cardisoma guanhumi. *See legend to fig. 1 and text for further details.*

pressure also accompanied a second 5% hemolymph volume infusion, increasing hemolymph volume from 5% to 10% above control volume. In most crabs, this second cumulative injection caused a larger transient increase in hemolymph pressure during the actual infusion than resulted during the first infusion (fig. 1).

Withdrawal of hemolymph to a volume 5% below control levels produced transient decreases in hemolymph pressure, with intracardiac pressures usually stabilizing within 5–10 s at levels 2–5 mmHg below prewithdrawal levels. Thus, the patterns of intracardiac pressure adjustment during withdrawal and infusion were essentially mirror images of each other.

Patterns of Heart-Rate Response

Highly characteristic changes in f_H were associated with experimentally induced changes in intracardiac hemolymph pressure. In most instances, the rise in hemolymph pressure during actual seawater infusion was accompanied within just one or two heartbeats by a decrease in f_H of 5–20 beats · min^{-1} (fig. 1). In some crabs a much more severe bradycardia developed with the first transient rise in hemolymph pressure (fig. 2), occasionally stopping the heart for a few beats.

One of two cardiac responses resulted when seawater infusion was stopped. In 10 of 13 crabs f_H stabilized within a few seconds at a mean rate 4–6 beats · min^{-1} lower than preinfusion rates (fig. 1). In three crabs following seawater infusions, f_H quickly returned to and stabilized at or near preinfusion rates (fig. 2). In all three cases intracardiac hemolymph pressure returned to control levels within 5–10 s.

Hemolymph withdrawal, resulting in a large transient fall in hemolymph pressure, was accompanied in most crabs by a 5–20 beat · min^{-1} increase in f_H within 1–2 heartbeats. After withdrawal was stopped, f_H increased back toward prewithdrawal levels, stabilizing within 5–10 s at an f_H 10–12 beats · min^{-1} higher than before withdrawal.

Quantitative Analysis of Pressure and Rate Effects

The effects of experimentally induced changes in hemolymph volume (expressed as a percentage of the initial control value) upon intracardiac pressure and f_H for 13 *C. guanhumi* are quantified in figure 3. Pressure and rate data are expressed in terms of the absolute change (increase or decrease) from the control value before the start of hemolymph volume adjustment. The two upper panels in figure 3 show the responses of individual crabs to a hemolymph volume increase or decrease relative to control hemolymph

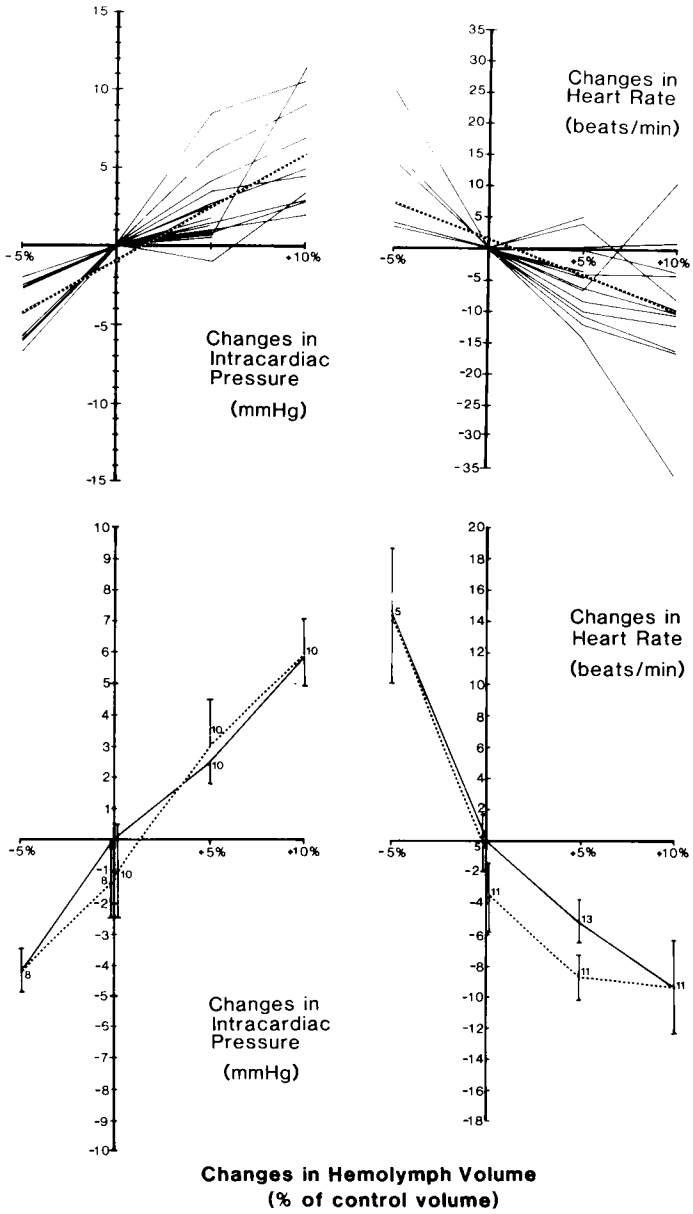

Fig. 3. Changes in intracardiac systolic pressure and heart rate induced by 5% incremental adjustments in hemolymph volume in the land crab Cardisoma guanhumi. *The upper left and upper right panels show raw data for intracardiac pressure and heart rate, respectively, for each crab during hemolymph removal and addition. The lower left and lower right panels show mean values ± 1 SE of hemolymph pressure and heart rate, respectively, at hemolymph volumes above and below control levels. The solid lines connect means recorded upon first increase or decrease in hemo-*

volume. Data for the return to control values are not shown for the sake of clarity. Also presented in the two upper panels are the lines resulting from the regression of the ordinate variable on hemolymph volume. The points at the graph's origin were excluded from the regression.

The regression of intracardiac hemolymph pressure on hemolymph volume is described by the equation $y = -0.86 + 0.70x$. The correlation coefficient is 0.75 ($n = 50$), with a P of <0.01. The confidence intervals of the slope are 0.59 and 0.82. Thus, intracardiac hemolymph pressure shows a significant positive correlation with hemolymph volume.

The regression of f_H on hemolymph volume is described by the equation $y = 1.46 + -1.13x$. The correlation coefficient is 0.40 ($n = 45$), with a P of <0.01. The confidence intervals of the slope are −1.56 and −1.13. Essentially, heart rate shows a significant negative correlation with hemolymph volume. As evident from examining the raw data in figure 3*b*, a few crabs showed highly aberrant response to hemolymph volume adjustment, contributing to the variance in the data.

Comparison of the slopes of the regressions of f_H and of hemolymph pressure on hemolymph volume (fig. 3) indicates that f_H decreased about 2 beats · min^{-1} for every mmHg increase in stabilized hemolymph pressure over the range of hemolymph volumes induced in these experiments.

To help assess any hysteresis in the pressure and rate responses, the two lower panels in figure 3 present mean values for intracardiac hemolymph pressure and f_H at the same hemolymph volumes shown in the top panels. In addition, these two panels show mean values for intracardiac hemolymph pressure and f_H during the return to control hemolymph volume following the initial increase or decrease in volume. Analysis of variance of all hemolymph pressure data (excluding values of 0 at the origin of the graph in common with all animals) indicated a highly significant effect ($P < 0.001$) of hemolymph volume on intracardiac hemolymph pressure. The mean values for the two sets of hemolymph pressures at a relative hemolymph volume of +5% were not significantly different (*t*-test for paired comparisons, $P > 0.1$). Similarly, there was no significant difference ($P > 0.1$) between mean values of hemolymph pressures at control hemolymph volume following either hemolymph withdrawal or sea-

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lymph volume. The dashed lines connect means recorded during subsequent return to control hemolymph volume. ANOVAs indicate treatment effects significant at the 0.001 level for data in both lower panels (see text for discussion of statistical analysis). Numbers beside each mean indicate number of crabs contributing to that point.

water injection. Thus, there was no hysteresis in the response of hemolymph pressure to change in hemolymph volume.

A similar analysis for changes in f_H following hemolymph volume adjustment showed a small hysteresis effect in the heart rate response to hemolymph adjustment. There was no significant difference ($P > 0.1$) between f_H at +5% relative volume before and after increase in volume to +10%. However, f_H did not return to control values within the 30–60-s measurement period following return to control hemolymph volume, but rather was significantly lower ($P < 0.25$) than the initial control values. The difference between means was less than 4 beats · min^{-1}, however, with a few aberrant crabs once again contributing to much of the difference. Withdrawal of hemolymph to −5% of control volume and subsequent restoration of hemolymph volume back to control values showed no hysteresis effects, with hemolymph pressures before and after hemolymph withdrawal showing no significant difference ($P > 0.1$).

The disruptive effect of excessive hemolymph removal on cardiovascular homeostasis was assessed in four crabs and is clearly shown in figure 4. In the illustrated experiment, the equivalent of 15% hemolymph volume was removed over a 40-s period. During the course of withdrawal, intracardiac hemolymph diastolic pressure fell from about 10 mmHg to 0 mmHg. The f_H initially remained stable in this crab (not initially increasing as in most crabs with only 5% hemolymph removal), but after about 15–20 s it fell to under 20 beats · min^{-1} and stopped momentarily toward the end of the period of hemolymph withdrawal. After withdrawal of the equivalent of 15% hemolymph volume, mean hemolymph pressure showed little recovery back to control levels, and a pronounced arrhythmia developed. Reinfusion of the equivalent of 5% hemolymph volume immediately restored f_H to control values and caused an increase in mean hemolymph pressure, but not to control values. Only after infusion of the remaining 10% of hemolymph volumes was intracardiac hemolymph pressure restored.

Although the magnitude of change of hemolymph pressure and f_H in response to hemolymph removal beyond 10% varied from crab to crab, the general pattern of arrhythmia development in response to hypovolemia was observed consistently. This suggests that (1) a hemolymph volume of between 85% and 90% of normal, hydrated volume is required to maintain f_H as well as hemolymph pressure, and (2) excessive decreases in hemolymph volume apparently can disrupt the normal relationship between hemolymph volume and f_H.

Discussion

Several ecologically and physiologically relevant stresses may disrupt hemolymph pressures and volume in *Cardisoma guanhumi*. Although changes

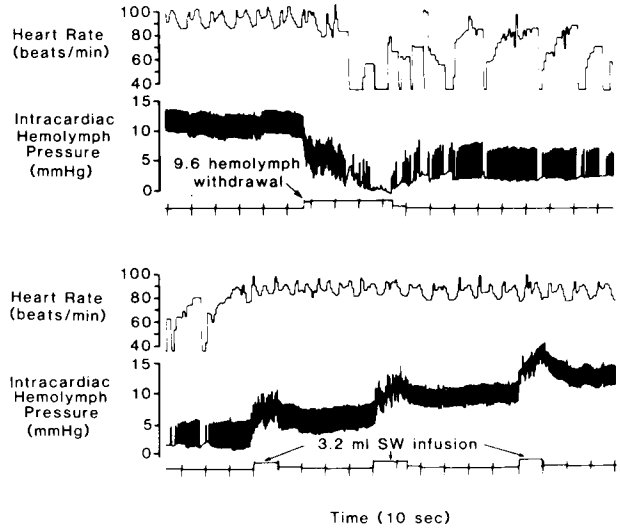

Fig. 4. Adjustment in intracardiac hemolymph pressure and heart rate in response to an unusually large withdrawal of hemolymph (equivalent to 15% total hemolymph volume) from the circulation of a restrained, 207-g Cardisoma guanhumi *(top panel). At several points the tracing of heart rate "bottoms" out, indicating a heart rate below 40 beats · min^{-1}. Bottom panel shows the effects in the same crab of reinfusing hemolymph in three increments each equivalent to 5% total hemolymph volume, bringing total hemolymph volume back to prewithdrawal levels. In this experiment, severe bleeding and subsequent restoration of hemolymph did not cause the changes in heart rate typical of more moderate withdrawals and infusion. See text for further details.*

in cardiac output during activity have not been measured in *C. guanhumi,* it is likely that changes in hemolymph pressure would result (see Burggren and McMahon 1988 for a detailed description of hemodynamics in land crabs). Hemolymph pressures in *C. guanhumi* also are profoundly affected by periods of reversed scaphognathite beating (Burggren et al. 1985). In the longer term, sizable variations in hemolymph volume can occur during the seasonal hydration and dehydration typically experienced by brachyuran crabs (Greenaway 1988). Thus, a variety of physiological and environmental stresses may act to disrupt hemolymph volume and pressure in *Cardisoma.*

Although the importance of regulation of blood pressure within a relatively narrow range is considered critical in high-pressure vertebrate circulations, the ability to detect and then offset fluctuations in hemolymph pressure may also be of great importance in the open, relatively low-pressure

circulation of many invertebrates such as *C. guanhumi.* Certainly in *Cardisoma,* which has an intracardiac systolic pressure of only about 13–15 mmHg, small fluctuations in arterial pressure may have several important physiological consequences. For example, ultrafiltration of urine in the antennal gland (Greenaway 1988) and mechanical internal support for individual gill filaments in air (Burggren and McMahon 1988) both depend on arterial hemolymph pressures. From a general cardiovascular perspective, a fall in intracardiac systolic pressure by the seemingly small amount of 4–5 mmHg will, with an unchanged venous pressure, reduce by 50% the pressure gradient at systole propelling hemolymph through the circulation. This occurs because the total pressure gradient at systole between the heart and the venous side of the circulation is only about 8–10 mmHg (Burggren et al. 1985). These examples collectively illustrate the importance in *C. guanhumi* of a mechanism for regulating arterial hemolymph pressure within narrowly defined limits. How is such regulation achieved?

The present study on *Cardisoma* has revealed that experimentally increasing or decreasing hemolymph volume causes large transient changes in hemolymph pressure, and that in most crabs these changes are partly compensated for within several heartbeats. Certainly, some of this compensation will be achieved by "passive" distribution of hemolymph within the compliant vasculature. Such compensation can be completely independent of changes in cardiac output. Indeed, a return of hemolymph pressure to preinfusion or prewithdrawal values is not absolutely contingent on a change in f_H (figs. 2, 4). However, in the face of an initially unchanged peripheral resistance, the reciprocal change in f_H (and presumably cardiac output) can also be expected to have a compensatory, stabilizing effect on hemolymph pressure. Adjustment in f_H is a major mechanism by which blood pressure is adjusted in "closed" circulations (Jones and Milsom 1982; Bagshaw 1985), and such a mechanism should, in theory, also be effective in regulating hemolymph pressure in *Cardisoma.*

That perturbations in hemolymph pressure produced by hemolymph volume changes can be partly compensated for in *C. guanhumi* is clear, as is the negative correlation between f_H and hemolymph volume. A crucial point, however, is whether the change in f_H associated with hemolymph pressure adjustment actually represents the effector limb of a neural reflex regulating hemolymph pressure. That is, do changes in f_H modify hemolymph pressure via a baroreceptor reflex? The data we present on the interaction of f_H and hemolymph pressure do not in themselves constitute unequivocal proof of a baroreceptor reflex in *C. guanhumi,* because we have not yet been able to compare the effects on hemolymph pressure produced by hemolymph volume change in crabs in which the f_H response is experimentally blocked.

If a baroreceptor reflex exists, then a change in hemolymph pressure induced by a change in hemolymph volume will be larger when compensatory changes in f_H are prevented. Nonetheless, three distinct lines of evidence from this study lead us to hypothesize that changes in hemolymph pressure in *Cardisoma* are indeed being sensed by vascular mechanoreceptors, whose afferent activity mediates neurogenic reflex adjustments in f_H acting to minimize changes in hemolymph pressure.

First, we discount the role of direct mechanical effects (nonreflexogenic effects) on the heart acting to alter f_H during hemolymph volume adjustment. In theory, it is possible that changes in f_H were the result of direct effects of changes in heart volume and thus in mechanical deformation of the heart. Both in vitro and in situ experiments on the hearts of decapod crustaceans (Maynard 1960; Kuramoto and Ebara 1984) and a variety of molluscs (Smith 1985; Wells and Smith 1987) invariably indicate that invertebrate hearts respond directly to increased preload by *increasing* heart rate. This Frank-Starling relationship, which is typical of vertebrate hearts, no doubt will be useful in preventing venous and pericardial pooling but clearly cannot account for the *decrease* in f_H accompanying increased intracardiac pressure in our in vivo experiments.

Second, the extreme rapidity (a few heartbeats) of the onset of the chronotropic compensation to transient changes in hemolymph pressure would indicate a neurally mediated response. The time course of the response certainly seems inconsistent with the involvement of hemolymph-borne cardiac modulators released in direct response to general changes in tissue perfusion in some way caused by changing hemolymph volume.

Finally, the consistent inverse correlation between stabilized f_H and stabilized hemolymph pressures, both above and below control hemolymph levels, is highly suggestive of the low "steady-state error" (i.e., small difference between control pressure and "stabilized" pressure) inherent in physiological baroreflexes (see Cecchini, Melbin, and Noordergraaf 1981; West and Van Vliet 1983). Moreover, the fact that the much larger disturbance to hemolymph pressure produced by a withdrawal compared to an equivalent infusion volume nonetheless is precisely matched by a correspondingly larger adjustment in heart rate (fig. 3) suggests that heart rate is responding precisely to changes in hemolymph volume, rather than to general systemic disturbances associated with the mechanical act of withdrawal.

To our knowledge, this is the first study of a decapod circulation (in fact, of any invertebrate circulatory system) that indicates that hemolymph pressure may be regulated by a reflex change in cardiac performance. As such, this study helps to dispel the traditional, outdated notion that invertebrate circulations are inherently less efficient or sophisticated than the closed circula-

tion of most vertebrates. The gross location, structure, and function of the putative sensory cells monitoring hemolymph pressure in *Cardisoma,* as well as the efferent neural pathways to the heart and the extent of involvement of neurosecretory substances, are as yet unknown. Our present experimental procedure of adjusting hemolymph volume no doubt changed hemolymph pressure throughout large regions of the vascular system. Experiments involving isolation and pressurization of various regions within the circulation will be required in future studies to characterize further baroreceptor-like responses in decapod Crustacea.

Acknowledgments

This study was carried out at the Smithsonian Tropical Research Institute, Panama. We are grateful for financial support by the University of Massachusetts and U.S. National Science Foundation (grant DCB-8608658 to W.W.B.) and the Travel (T 1825), International Collaborative (IC0072), and Operating Grant (A5276) Programs of the Canadian National Science and Engineering Research Council (awarded to B.R.M.). We also thank Dr. J. Cubit of the Galetta Marine Laboratory for his help in locating animals.

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