The Crustacean Open Circulatory System: A Reexamination

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Abstract

Open circulatory systems have been portrayed as poorly designed systems with poor performance characteristics, lacking in adequate tissue perfusion or fine control mechanisms. Recent studies cast doubt on these assumptions. Open circulatory systems of at least the higher Malacostraca have elaborate capillary beds in many tissues. Cardiac outputs are generally higher than those of the closed systems of poikilothermic vertebrates of equivalent size and activity potential. Although arterial pressures and flows are often lower than those characteristic of poikilothermic vertebrates, the crustacean arterial system is adapted to deliver equivalent flows under these conditions. The control systems of the crustacean neurogenic heart appear capable of fine graded control over cardiac output. In addition, although crustaceans lack the arteriolar smooth muscle upon which much of the peripheral circulatory control of vertebrate closed systems depends, cardioarterial valvular mechanisms under neural and neurohormonal control appear to be capable of selective distribution of this output between the several separate arterial systems. Thus, although open circulatory systems differ greatly in anatomy from closed blood systems, they are, nonetheless, functionally equivalent.

Introduction

This topic was last treated comprehensively in the perceptive review of Maynard (1960). Since then, although there have been frequent reviews of the neuromuscular physiology of the heart, no review has tackled the broader perspective—that is, the physiology of the open circulatory system itself. With this review we seek to complement our expanded understanding of cardiac function with new information on the functioning of the entire circulatory system, and to provide a revised description of the capabilities of the open

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circulatory system in decapod crustaceans. Finally, the capability of the open circulatory system (assessed as the ability to deliver oxygen) is reexamined at rest, in activity, and when stressed (i.e., as by hypoxic exposure).

In a group with morphology as diverse as the Crustacea it is very difficult to present a description of the circulatory system that is both general and inclusive (Maynard 1960). Open circulatory systems of crustaceans range from extremely simple, as in some Copepoda in which only a single tubular contractile vessel has been described, to the very complex system of the higher decapods. The decapods possess a condensed muscular heart, which pumps hemolymph through complex arterial systems that perfuse the tissues via a network of very fine vessels that are morphologically and functionally equivalent to the capillaries of closed circulatory systems. (Circulating body fluids within the crustacean open circulatory system [hemocoel] are designated hemolymph here to separate them from blood circulating in closed systems of vertebrates. Hemolymph serves the functions of both the blood and lymph fluids of closed systems.) Venous return occurs via distinct channels to the gills or branchiostegal membrane circulations, then to the pericardial cavity, and finally to the heart. Maynard's (1960) review includes a comprehensive account of the range of complexity found in open circulatory systems. Since recent physiological observations have, however, been largely restricted to the circulatory systems of isopod and particularly decapod Crustacea, this account is similarly restricted to the capabilities of these "top of the line" crustacean open circulatory systems.

The crustacean open circulatory system is generally characterized as a sluggish system with a single-chambered heart, capable of developing only low pressures and flows. The tissues are thought to be bathed in hemolymph circulating loosely through the hemocoel. Little capability to control either hemolymph pressure or flow, either within or between various organs or between circulatory beds, has been recognized. In the course of this review we will reexamine these tenets and undertake a reinterpretation of the functioning of these open circulatory systems.

Before going on to review the physiology of the circulatory system, we will briefly describe the anatomy of the system as it occurs in macruran and brachyuran crustacean body types (fig. 1*A*, 1*B*, 1*C*; from McLaughlin [1983]). Diagrams of other types of circulatory systems can be found in McLaughlin (1983).

The Heart and the Pericardial Cavity

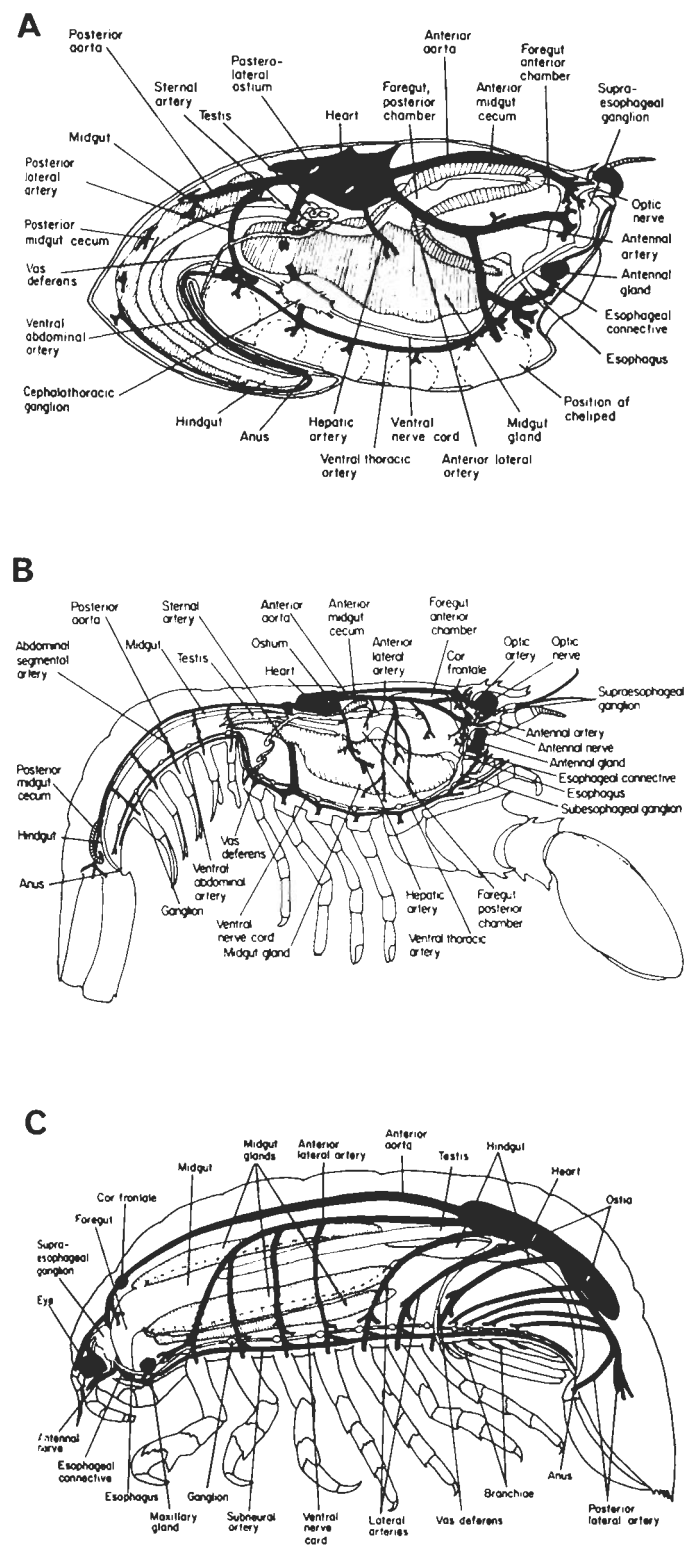
The architecture of the decapod crustacean heart is generally described as consisting of a single muscular chamber (ventricle). The ventricle is, how-

ever, suspended within a second chamber, the pericardial cavity, by a series of alary ligaments (fig. 2). At least in the decapod crustaceans, the walls of the pericardial cavity (pericardium) totally surround the heart, and in many ways this cavity functions as a second (primer) heart chamber, as suggested originally by Miller (1895). Unlike the case in most other cardiac systems, there is no direct tubular return, and hemolymph returning from the tissues collects in the pericardial cavity. Filling of the heart occurs through a series (normally three) of paired ostial valves in the ventricular walls (fig. 2) that communicate between the pericardial and ventricular cavities.

Within the ventricle, muscle is arranged in a complex series of trabeculae (Maynard 1960), presumably organized to ensure efficient cardioejection into the complex arterial system. This comprises seven separate outflow systems: a single anterior aorta (ophthalmic artery) at the anterior end, and a single posterior aorta (superior abdominal artery) and sternal artery posteriorly, in addition to paired antennary and hepatic arteries leaving anterolaterally (fig. 1). Each artery is isolated from the heart by a pair of semilunar valves at the origin. Like the aortic valves of vertebrate closed systems, these act to prevent reflux of hemolymph into the heart during diastole, but they differ in that they are also muscular and can contract rhythmically, perhaps allowing control over distribution of cardiac output (discussed below).

In the higher decapod crustaceans, hemolymph is distributed from the finer branches of these arterial systems into capillaries and/or lacunae in the tissues before passing into the hemocoel. Capillaries are defined by Maynard (1960) as small-caliber vessels (from 7 to 50 μm in diameter) lined with a single layer of endothelium but without intima. Lacunae are defined as irregular hemolymph spaces where a limiting membrane is apparently absent. It is at this level that hemolymph may bathe the tissues directly, giving rise to the term open circulatory system. Tissue perfusion depends considerably on its location within the animal, with nervous tissue (fig. 3), green gland, and stomach having the greatest density of capillaries.

En route back to the heart, hemolymph is channeled through a system of sinuses, often bounded by endothelium and intima, into the collecting sinuses that supply the gills and the branchiostegal circulations. Note that the gills do not constitute the only path of "venous" return to the heart. The alternative branchiostegal circulation is present in aquatic forms—for example, in *Cancer magister* (fig. 4)—but is particularly well developed in those air-breathing and amphibious crustaceans that utilize the branchiostegal membranes for gas exchange (Greenaway and Farrelly 1990).


A
Posterior aorta
Sternal artery
Postero-lateral ostium
Testis
Heart
Anterior aorta
Foregut, posterior chamber
Foregut anterior chamber
Anterior midgut cecum
Supra-esophageal ganglion
Midgut
Posterior lateral artery
Posterior midgut cecum
Vas deferens
Ventral abdominal artery
Cephalothoracic ganglion
Optic nerve
Antennal artery
Antennal gland
Esophageal connective
Esophagus
Position of cheliped
Hindgut
Anus
Hepatic artery
Ventral nerve cord
Midgut gland
Ventral thoracic artery
Anterior lateral artery
B
Posterior aorta
Sternal artery
Anterior aorta
Anterior midgut cecum
Foregut anterior chamber
Abdominal segmental artery
Midgut
Testis
Ostium
Heart
Anterior lateral artery
Cor frontale
Optic artery
Optic nerve
Supraesophageal ganglion
Antennal artery
Antennal nerve
Antennal gland
Esophageal connective
Esophagus
Subesophageal ganglion
Posterior midgut cecum
Hindgut
Anus
Vas deferens
Ventral abdominal artery
Ganglion
Ventral nerve cord
Midgut gland
Hepatic artery
Foregut posterior chamber
Ventral thoracic artery
C
Midgut glands
Anterior lateral artery
Anterior aorta
Testis
Hindgut
Midgut
Heart
Cor frontale
Ostia
Foregut
Supra-esophageal ganglion
Eye
Antennal nerve
Esophageal connective
Esophagus
Ganglion
Maxillary gland
Subneural artery
Ventral nerve cord
Lateral arteries
Vas deferens
Branchiae
Anus
Posterior lateral artery

Physiology of the Circulatory System

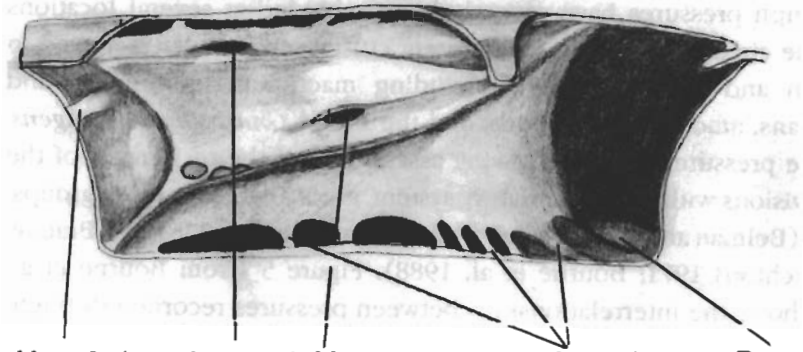
Hemolymph Pressure and Flow Relationships

Hemolymph pressures have recently been recorded at several locations within the circulatory system for different crustacean groups (reviewed in McMahon and Wilkens [1983]) including macrurans, anomurans, and brachyurans, among the decapods, and the mysid *Gnathophausia ingens.* Complete pressure profiles, allowing assessment of the resistances of the major divisions within the circulatory system, are available for three groups: Mysidae (Belman and Childress 1976), Macrura (Belman 1975), and Brachyura (Blatchford 1971; Bourne et al. 1988). Figure 5 (from Bourne et al. [1988]) shows the interrelationships between pressures recorded throughout the circulatory system of *Cancer magister.* Pressures in the pericardial cavity exceeded pressures in the ventricle (mean pressure difference = 0.176 kPa = 1.3 mmHg; fig. 5, $P - V$) during a large part of diastole and are clearly associated with diastolic filling through the open ostial valves. In these animals at resting heart rates of 70 beats/min, diastolic filling occupied 40% of the cardiac cycle (approximately 300 ms). Similar data were reported for the land crab *Cardisoma guanhumi* (Burggren et al. 1985). In either case pericardial pressures are normally maintained above ambient pressure throughout the cardiac cycle, even at resting heart rates, suggesting that the pumping action of the heart alone is sufficient to account fully for circulation through the entire system. Chronic recordings of pericardial pressure in animals exhibiting intermittent branchial ventilation (see fig. 16 below) indicate that these pressures are highest during the phase of hyperventilation seen immediately after a ventilatory pause and decrease slowly with the subsequent decline in ventilation. Toward the end of a ventilatory period, pressures in the pericardial cavity may approach zero, or even fall below ambient pressure briefly within each cardiac cycle (fig. 16).

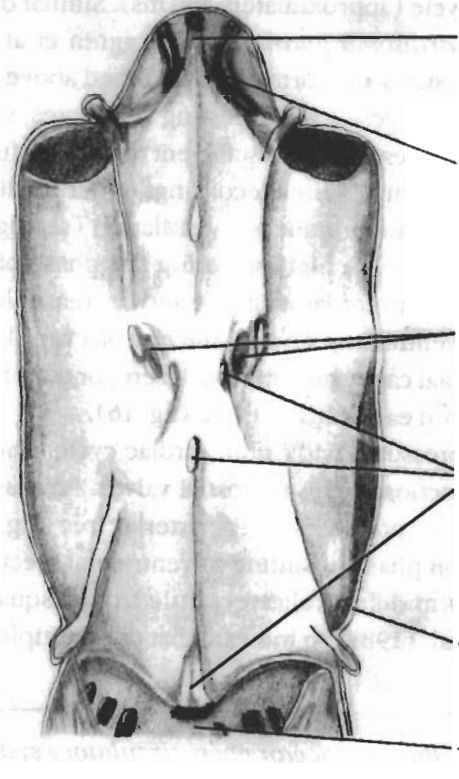
Let us return to pressures within the cardiac cycle. The commencement of ventricular contraction closes the ostial valves. Pressure in the ventricle (fig. 5, *V*) rises above pressures in the arterial tree (fig. 5, *A*) during the isometric contraction phase, resulting in ventricular ejection. The ventricular pressure waveform differs relatively little from a square wave, which is taken by Bourne et al. (1988) to indicate that the multiple arterial system of

Fig. 1. Diagrams of the crustacean open circulatory system of (A) *brachyuran (Portunid crab)* (B) *macruran (Homarid lobster) and* (C) *isopod* (flabelliferan) *crustaceans to show heart and major arterial distribution routes. (Reproduced, with permission, from McLaughlin* [*1983*].)

LATERAL



DORSAL



the decapods offers a relatively high capacitance, and thus more rapid runoff of hemolymph to the periphery, when compared with other circulatory systems in the literature. The mean pressure recorded during ventricular ejection in figure 5 is 1.9 kPa (14.3 mmHg). Following ventricular contraction, once the ventricular pressure falls below that in the arterial system (fig. 5, *A*) the arterial valves close and pressure falls very sharply. Hemolymph pressure in the arterial tree rises rapidly to levels similar to those recorded in the heart, confirming that there is little resistance to ejection of hemolymph. The low pulse pressures recorded in the arterial system support the suggestion of a high-capacitance system outlined above.

Pressures recorded from a leg artery greater than 5 cm from the heart in the crab *C. magister* are not highly pulsatile (fig. 5, *A*), suggesting that a substantial fraction of the cardiac pulse is absorbed by deformation of the elastic elements of the arterial wall during systole. The elasticity serves to maintain arterial pressure during diastole. Shadwick, Pollack, and Stricker's (1990) recent measurements show crustacean arterial walls to be remarkably thin structures containing no muscle tissue but consisting of an adventitial layer of collagen fibers surrounding a concentric network of organized elastic fibers. These authors report that, despite their thin walls, crustacean arteries are mechanically analogous to the aortas of cephalopod molluscs and lower vertebrates (fig. 6), are well designed for the elastic energy-storage role suggested above, but are designed to function at pressures lower than those found in vertebrates. Higher pressures can be sustained; indeed, peak pulse pressures of up to 6 kPa (45 mmHg) have been reported during activity in the dorsal abdominal artery of the lobsters *Panulirus* (Belman 1975) and *Homarus* (Jorgensen et al. 1989).

Pressure recordings from the infrabranchial cavity (a collecting sinus for hemolymph returning to the gills) for resting *Cancer* were occasionally pulsatile (fig. 5, *I*), but, unlike the situation reported by Belman (1975) for lobster, these pulsations were more usually associated with the scaphognathite-induced pressure fluctuations in the branchial chamber (not shown in fig. 5) rather than cardiac events. A similar situation has been reported for

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*Fig. 2. Drawing of the heart of a decapod crustacean (*Astacus fluviatilis*) showing alary ligaments, arterial outflows and ostial valves. Top, lateral view showing ostia and attachments to the ventral pericardial septum; bottom, dorsal view;* Opth. Art., *ophthalmic artery;* Ant. Art., *right antennary artery;* Post. Ab., *posterior abdominal artery complex;* Hep. Art., *left hepatic artery. (Redrawn from Baumann [1921].)*

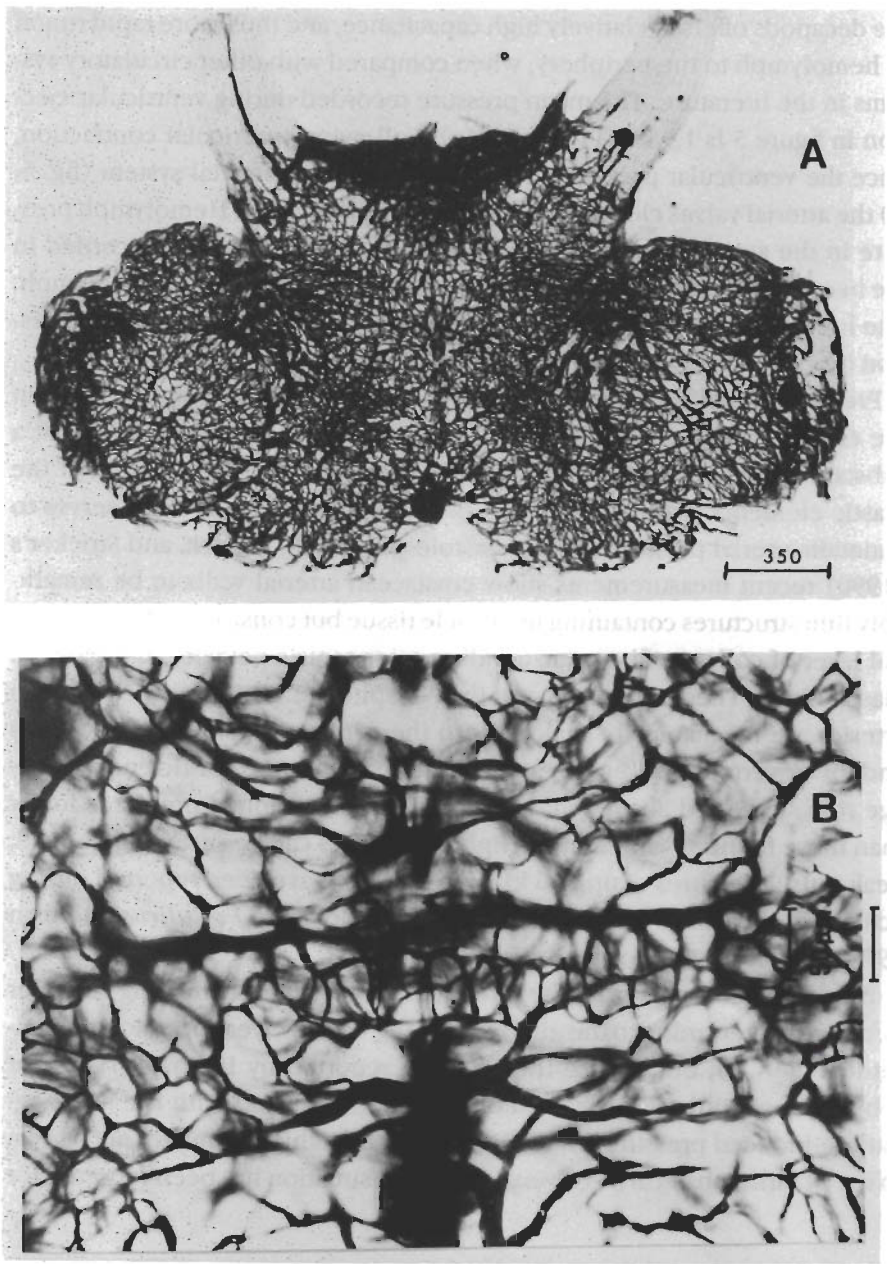

Fig. 3. Micrographs of the hemolymph vessel supply to the brain and to individual neuropil areas of the crayfish Cherax destructor *(reproduced with permission from R. and D. Sandeman, unpublished data).* A, *Horizontal 100-*μ*m Vibratome section through the brain;* B, *circulatory supply to a small area of the central body. Scale bar in* B = 50 μ*m.*

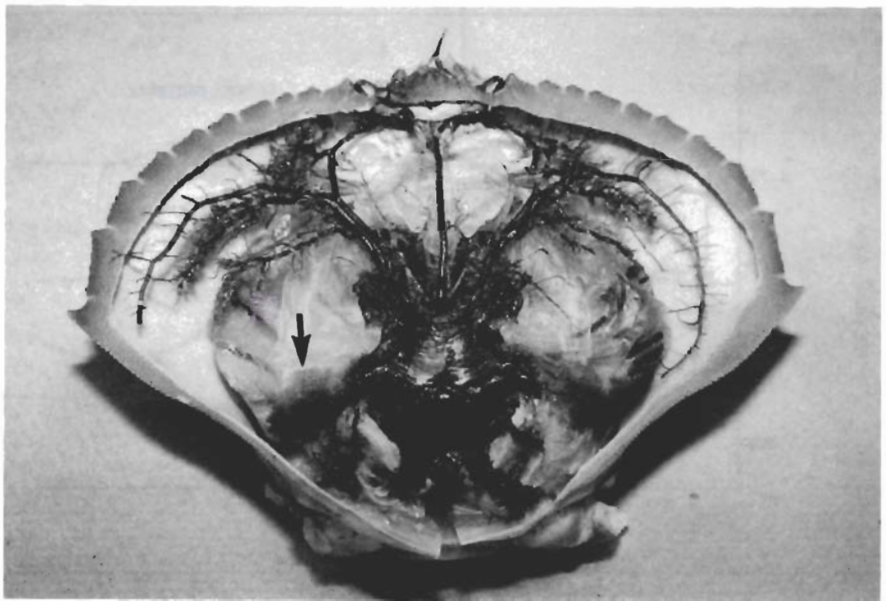

Fig. 4. Photograph of a cast (methacrylate: Batsons no. 17) of part of the circulation of the crab Cancer productus *to show the branchiostegal circulation (arrow). (P. Greenaway, S. Morris, and B. R. McMahon, unpublished data, reproduced with permission.)*

another crab, *Carcinus maenas* (Blatchford 1971). Pressure in the infrabranchial cavity, nonetheless, always exceeds that in the pericardium (mean pressure differential in *C. magister* $I - P = 0.30 \pm 0.125$ kPa [2.28 ± 0.93 mmHg]; fig. 5), this differential representing the pressure gradient needed to perfuse the branchial return pathways. By contrast, the average pressure differential between the infrabranchial and the arterial pressures (fig. 5, $A - I$) was 0.62 ± 0.054 kPa (4.63 ± 0.4 mmHg). The resistance of the systemic arterial network thus accounts for 67% of the total pressure drop in *C. magister,* and 75% in the lobster *Homarus americanus* (Maynard 1960).

Control of Hemolymph Pressure

Although there is little published evidence for reflex control of any aspect of circulatory performance in crustaceans, recent evidence (Burggren et al. 1990) suggests that receptors monitoring hemolymph pressure mediate a baroreceptor or baroreceptor-like reflex in the land crab *C. guanhumi.* Removal of hemolymph, or infusion of saline, into the infrabranchial sinus causes predictable, corrective changes in both heart rate and hemolymph

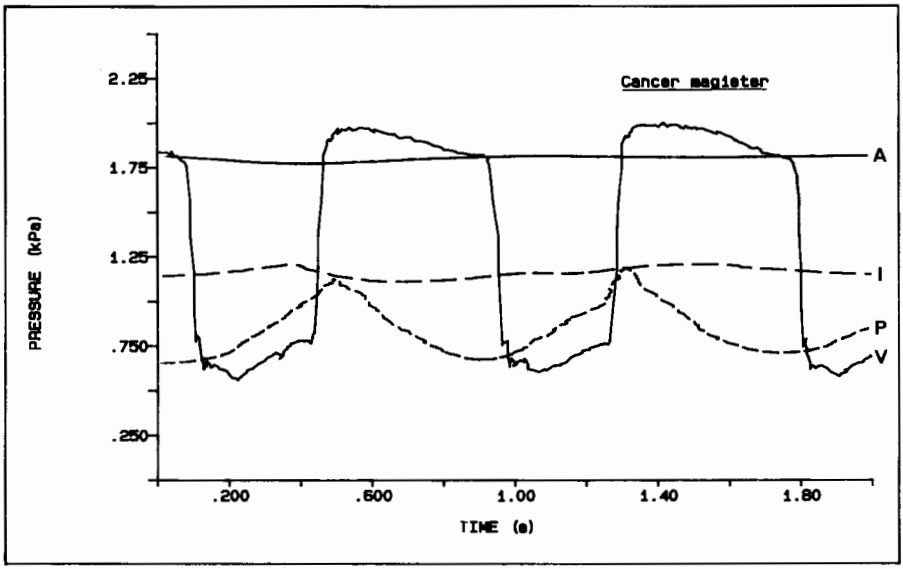

Fig. 5. Pressures recorded from the circulatory system of the crab Cancer magister. *Mean pressures recorded at several locations throughout the system are superimposed to show interrelationships.* A, *Leg artery;* P, *pericardial cavity pressure;* V, *intraventricular pressure;* I, *infrabranchial sinus pressure. Chart recordings of pressure waveforms were digitized with Sigma-scan software and further processed by Asystant+. The mean pressure waveforms were produced by smoothing through a Blackman low-pass filter. (Reproduced, with permission, from Bourne et al. [1988].)*

pressure, apparently to return hemolymph pressure to normal; that is, injection of saline (increase in hemolymph volume) causes an initial increase in pressure, but a rapid decrease in heart rate acts to return hemolymph pressure towards (but rarely to) prestress levels (fig. 7). The changes are rapid and not apparently mediated by change in the input pressure to the heart as discussed below, since this response is the opposite of the effect of stretch on the myocardium. The rapidity of the response (within 1 or 2 beats of the heart) suggests a neurally mediated or conceivably neurohormonal mechanism. Since there is no vascular smooth muscle, this effect may be mediated at the level of the heart or its valves (see below) or possibly at the level of the epimeral wall (Taylor 1990); the pathways remain obscure.

Assessment of Heart Performance

As stated above, the functioning of the cardiac ganglion and neuromuscular control systems of crustacean hearts have been reviewed relatively often

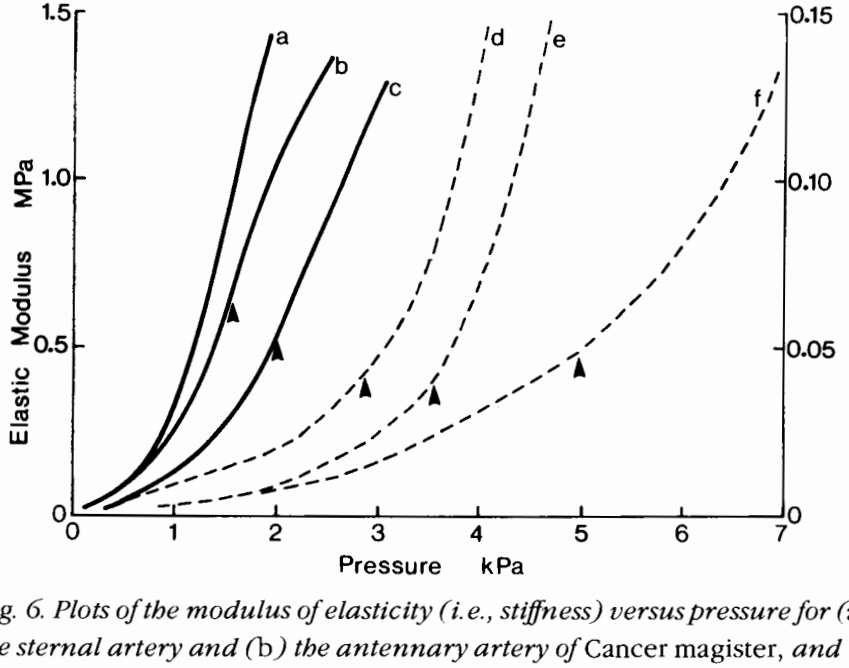

Fig. 6. Plots of the modulus of elasticity (i.e., stiffness) versus pressure for (a) *the sternal artery and* (b) *the antennary artery of* Cancer magister, *and* (c) *the posterior aorta of* Homarus americanus, *compared with the dorsal aortas of* (d) *the toad* Bufo marinus, (e) *the cephalopod* Octopus dofleini *(right ordinate), and* (f) *the snake* Thamnophis radix. *Arrows indicate approximate mean resting blood pressure for each artery. (Curves* a, b, *and* c *are from Shadwick et al.* [*1990*]; d *and* f *are from Gibbons and Shadwick* [*1988*]; e *is from Shadwick and Gosline* [*1985*]; *all reproduced with permission.)*

(see references in Wilkens [1987]), and most recently by Kuramoto (1990). Therefore, they will not be a focus here. Heart performance for the current purpose is best assessed as the cardiac output—that is, the volume of hemolymph pumped per minute. This has been difficult to quantify in crustaceans because of the large number of arterial outputs and the efficient, but uncontrollable, hemolymph-clotting mechanisms that usually confound all attempts to chronically implant flow probes. Most of the values for cardiac output reviewed by McMahon and Wilkens (1983) have been obtained indirectly, via measurements of oxygen uptake and oxygen concentrations in the hemolymph before and after passage through the heart/gill system, by means of the Fick equation. Recent direct measurement of cardiac output by a thermal dilution method (Burnett, DeFur, and Jorgensen 1981; Bourne et al. 1988) have corroborated simultaneous Fick estimates in situ (table 1), confirming a high resting cardiac output. These high cardiac outputs have

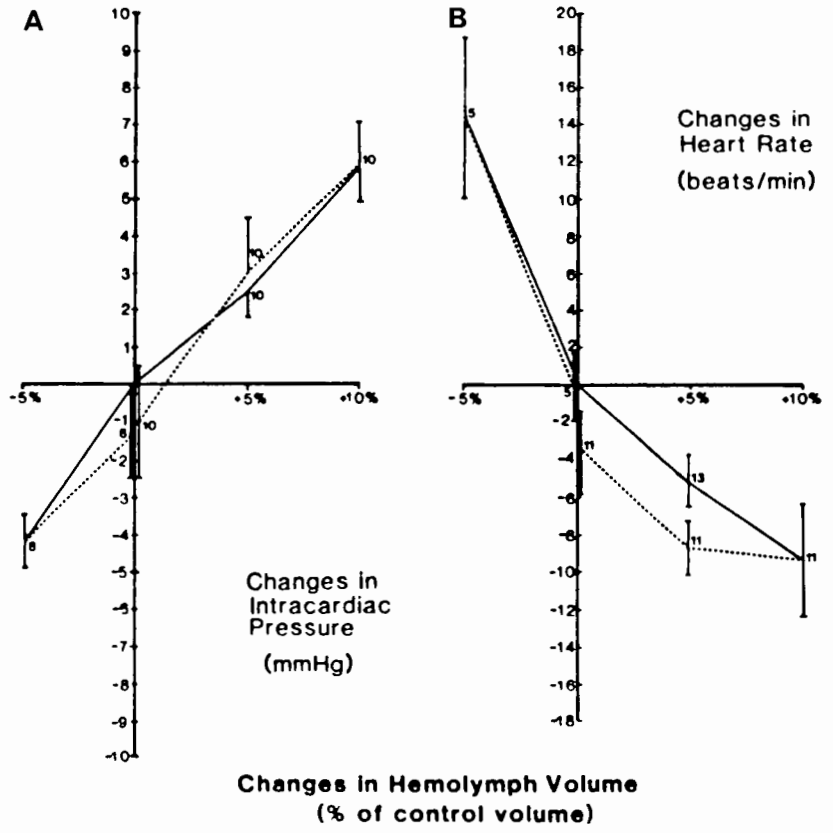

Fig. 7. Variation in (A) *intracardiac pressure and* (B) *heart rate during experimental manipulation of circulating hemolymph volume in the land crab* Cardisoma guanhumi. *Numbers at each point indicate the number of observations. Histograms' bars are ± 1 SEM. Solid lines display immediate response to volume change. Dotted lines display means following subsequent return towards control values. (Reproduced, with permission, from data of Burggren et al. [1990].)*

been related to lower oxygen-carrying capacities of crustacean hemolymph (Burnett 1979; McMahon 1985).

Regulation of Heart Performance

Adjustment of cardiac output can occur by variation of heart rate and/or stroke volume. In the neurogenic heart of crustaceans, heart rate is set by the output of a small number of neurons in the cardiac ganglion (Kuramoto 1990). Although the cardiac ganglion can initiate and maintain regular beat-

ing of the isolated heart, the rate in situ can be regulated externally. Several factors could influence the excitatory state of the pacemaker system, its burst rate, and hence the heart rate (see review by Wilkens [1987]). These include central regulation—that is, by changes in (1) central excitatory and inhibitory discharge to the pacemaker system or (2) levels of circulating neurohormonal agents (Cooke and Sullivan 1982)—and autoregulation mediated by stretch applied to the heart wall possibly acting on the dendritic trees of one or more types of pacemaker cell (Kuramoto 1990).

Because of the lack of direct tubular return to the crustacean heart the relationship between heart rate and cardiac output is unclear. Early reports (see Maynard [1960] for review) suggest that the end diastolic volume is a function of the time available for filling. If this is correct, increase of heart rate could have complex effects on cardiac output (fig. 8). For instance, if we start at very low rates, cardiac output may initially rise considerably with frequency, since the heart has time to fill maximally between beats. Above a critical frequency, however, complete filling will no longer be possible, and the increase in cardiac output with increase in beat frequency will tend to level off (fig. 8). At very high frequencies other aspects of cardiac performance may be compromised, causing cardiac output to decrease despite an increase in heart rate. Similar reasoning was expressed by Maynard (1960).

The reasoning above, however, is simplified by the assumption that active mechanisms to regulate stroke volume are absent. This assumption is certainly not true for the crustacean open system, where variation in stroke volume independent of heart rate has been measured (Jorgensen et al. 1982; Bourne et al. 1988). The mechanisms involved, however, have been little studied, and our lack of information restricts the present discussion largely to potential mechanisms. For ease of discussion these will be divided into those that increase diastolic filling and those that increase the percentage of ejection, with the realization that these are not fully independent.

Diastolic filling (input loading, preloading) will vary with the pressure differential across the ostial valves. This, in turn, could vary with either the diameter of the ostial aperture or the degree of expansion of the ventricle. The ostial valves consist of innervated muscular tissue, but there are no published data suggesting variable distension. The effect of variation in the degree of ventricular expansion is complicated, again because of the lack of a direct venous return. Several factors could be involved. The heart is suspended in a largely rigid box, the pericardial cavity, by the alary ligaments (fig. 2). These act as elastic tensors that become extended in systole and thus store some of the energy of contraction, which is available in diastole to return the heart to its resting position. Since valves at the entrance of each arterial tree are thought to prevent reflux of hemolymph from the aortas,

TABLE 1

Mean values (or ranges) for cardiovascular variables for representative aquatic and terrestrial decapod crustaceans under resting,[a] normoxic conditions

Species	Temperature (°C)	Body Mass (g)	Resting Heart Rate (beats · min^{-1})	Stroke Volume (mL · kg^{-1})	Cardiac Output (mL · kg^{-1} · min^{-1})	Intracardiac Pressure: Systolic/Diastolic (mmHg)	Pericardial Sinus Pressure (mmHg)	Reference
Aquatic species:								
Cancer magister	12	1,282	76 ± 8	1.63 ± .58	159 ± 56[b]	15/4.5	8 (systole) 5 (diastole)	Bourne et al. 1988
C. magister								
Cancer productus	9	850	66	1.5–2.6	92–170[c]		6.3	McMahon et al. 1979
C. productus	16	379	150	. . .	. . .	10.4/6.0	. . .	Belman 1976
Carcinus maenas	12	346	94–100	1.9–2.8	103–275	. . .	15–3.0	McMahon and Wilkens 1977
C. maenas	15	50	92	1.3	118	. . .	. . .	Taylor and Butler 1978
Panulirus interruptus ...	20	Not stated	108	. . .	. . .	10.3/0	0	Blatchford 1971
	15	501	64	1.4	128	36/16	17.9	Belman 1976

Terrestrial species:								
Cardisoma guanhumi	30	127	134	. . .	. . .	14.1/5.5	8.4 (systole) 6.1 (diastole)	Burggren et al. 1985
Cardisoma carnifex	25	250–500	85	1.44	122	. . .	. . .	Wood and Randall 1981
Gecarcinus lateralis	25	30	97	1.41	137	. . .	. . .	Taylor and Davis 1981
G. lateralis	Not stated	28–64	160	. . .	. . .	. . .	. . .	O'Mahoney-Damon 1984
Ocypode quadrata	25	10–87	Not stated	. . .	70	. . .	. . .	Burnett 1979
Coenobita clypeatus	23	20–40	120	.32	38	. . .	. . .	McMahon and Burggren 1979
Birgus latro	27–30	623–1,130	300	. . .	. . .	49/3	0	Cameron and Mecklenberg 1973

[a] Conditions defined as "resting" vary considerably between studies, which no doubt contributes to some of the variations in reported values.
[b] Direct measurement by thermal dilution.
[c] Indirect measurement by Fick calculation.

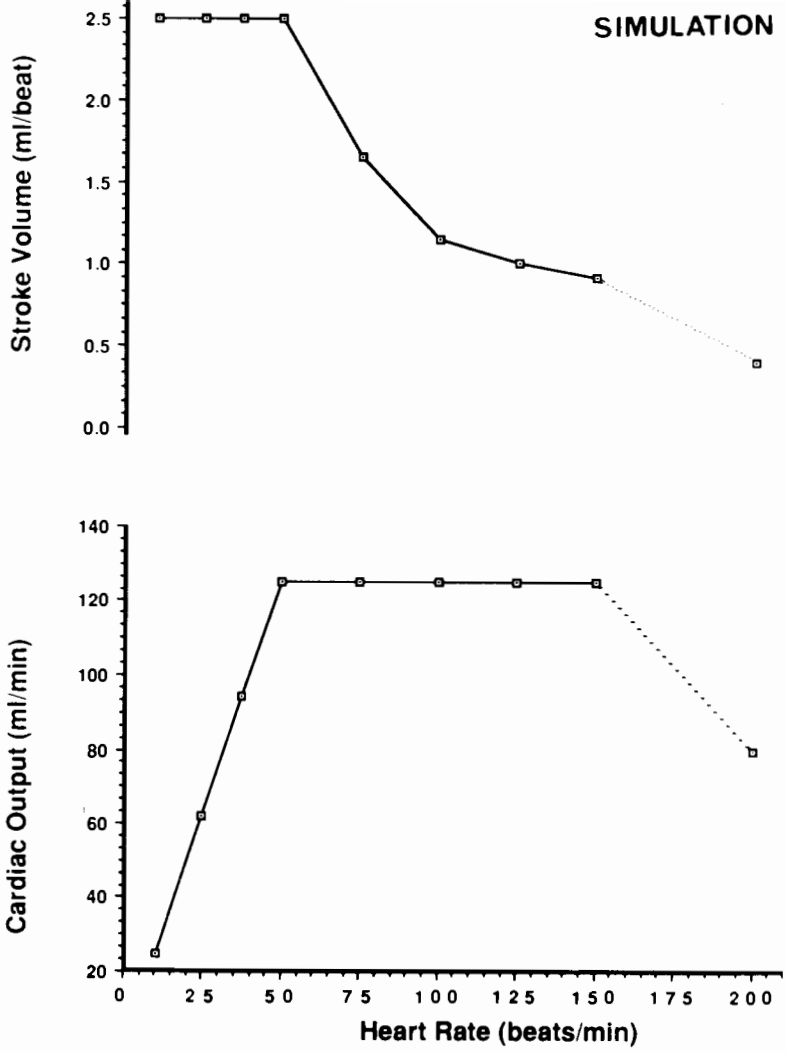

Fig. 8. Theoretical plot of potential passive variation in stroke volume and cardiac output with change in heart rate in crustacean heart. This graph is based on the assumption that no active regulation of stroke volume occurs (see text). The simulation is based upon the relationship cardiac output = frequency × stroke volume, expressed over an estimated maximal range of beat frequencies (for a midsize decapod crustacean). Below 50 beats/min an estimated maximum stroke volume (midsize decapod) was used. Above this range, stroke volume was decreased incrementally to simulate decreased filling time. Above 150 beats/min, decrease in stroke volume was increased to simulate functional breakdown.

volume expansion of the heart can result only from influx of hemolymph through the ostia.

Maynard's (1960) contention that alary ligaments contain little or no muscle and are attached to rigid skeletal elements has led to a general view that they make only this passive contribution to cardiac filling. Two factors, however, cause us to modify this view. First, many of the dorsal and lateral ligaments connect to the body wall epimera via muscular attachments (Pearson 1908). These muscular attachments have recently been shown to be both contractile and innervated (Volk 1988), and thus changes in their contractile state could affect heart tension and, possibly, diastolic filling. The second factor involves the ligaments that attach to the ventral surface of the heart (fig. 2). These are connected to the pericardial septum, which is flexible, muscular, and innervated (Maynard 1960). In *Cancer pagurus* (Mangold [1925], as cited in Maynard [1960]) stimulation of the septal (alary) muscles and in *Panulirus polyphagus* (George, Nair, and Muthe 1955) stimulation of the specific nerve supply both caused a depression of the ventral septum. George et al. (1955) also made the important observation that rhythmic depression of the pericardial septum occurred in phase with diastole. At this time—that is, when the ostial valves are open—a downward pull on the ligaments connected to the ventral heart wall would occur, thus expanding heart volume and hence end diastolic volume. Maintained ventral displacement of the pericardial septum would, in addition, cause an expansion of pericardial cavity volume, which would act to aspirate additional hemolymph from the branchial and/or branchiostegal circulations, providing an additional reserve of hemolymph for increased cardiac filling. Although the potential importance of this mechanism in varying cardiac output has been suggested (Maynard 1960; Wilkens 1987) it has not been tested. Additional work is required to confirm active regulation of pericardial volume and thus the hypothesis, presented above, that the pericardial cavity acts as a working (primer) chamber of the heart.

Systolic ejection varies with the force of myocardial contraction and with the output loading or resistance of the arterial system. In the crustacean heart, control of the former is extremely complex and suggests several potential routes for modification of cardiac output, including pacemaker regulation, autoregulation, and the influence of the central nervous system (CNS).

The neurogenic pacemaker system of the crustacean heart resides in the cardiac ganglion located on the inner dorsal wall of the heart (Alexandrowicz 1932). The ganglion is not only responsible for initiation and control of heart rate (see above) but can also influence the contractility of heart muscle (Maynard 1960; Hartline 1979) and hence the force of heart contraction and ventricular ejection. Pacemaker function is generally assumed to

reside in the small cells of the ganglion, whereas innervation of the myocardium occurs via the larger motor (follower) cells. Innervation is polyneuronal and multiterminal, and the fields of innervation of each motor cell overlap (Kuramoto and Kuwasawa 1980; Kuramoto 1990) allowing the possibility that each motor neuron may innervate large areas of the myocardium. Excitation of the myocardium is, therefore, not all-or-none, as in the vertebrate heart. Rather, stimulation of the muscle is via a burst of spikes; thus, gradations of force may be achieved by alteration of burst intensity, or burst length, or by an increase in the number of neurons or muscle units excited.

Autoregulation of cardiac output could occur at the level of the heart, via effects similar to those proposed for vertebrate heart by Frank (1895) and Starling (1918). As has long been known, stretch of the isolated lobster heart (i.e., with cardiac ganglion attached) causes an increase in both the rate and force of heart contraction (Maynard 1960). Aortic pressure also increases (Kuramoto and Ebara 1984*a;* see fig. 9), presumably as the result of increased cardiac output. Stretch of the dendritic tree of the pacemaker system neurons may be the effective factor (Alexandrowicz 1932; Maynard 1960), although different responses to stretch, and to application of 5-hydroxytryptamine (5-HT), have been observed in each of three classes of pacemaker cells in *Panulirus* (Kuramoto and Ebara 1988; Kuramoto 1990), suggesting that the control mechanisms may be much more complex.

A potential problem associated with these observations is that the crustacean heart is normally stretched by the alary ligaments, which are, however, disconnected in these experiments, and it is conceivable that the results reported by Kuramoto and Ebara (1984*a;* see fig. 9) simply reflect a return to normal tension. Ignoring this, if we assume that within physiological limits an increase in cardiac dimension is associated with an increase in rate and/or force of systole (Kuramoto and Ebara 1984*a;* fig. 9), then increased cardiac filling resulting from depression of the ventral septum would result in increased cardiac output automatically by this apparent Starling principle. Wilkens (1987) suggests that the supplemental (double) cardiac contractions occasionally seen at low heart frequencies may also serve to enhance cardiac output at low heart rates.

Adjustment of cardiac output via the CNS is also likely, and could occur at several levels. Both excitatory and inhibitory nerves run from the cardiac control centers in the CNS to the heart (Bullock and Horridge 1965; Wilkens, Wilkens, and McMahon 1974; Young 1978). These both contact the cardiac ganglion and innervate muscle fibers directly (Delalue and Holley 1976; Yazawa and Kuwasawa 1984). This innervation could clearly regulate cardiac performance in the short term either chronotropically, via the pace-

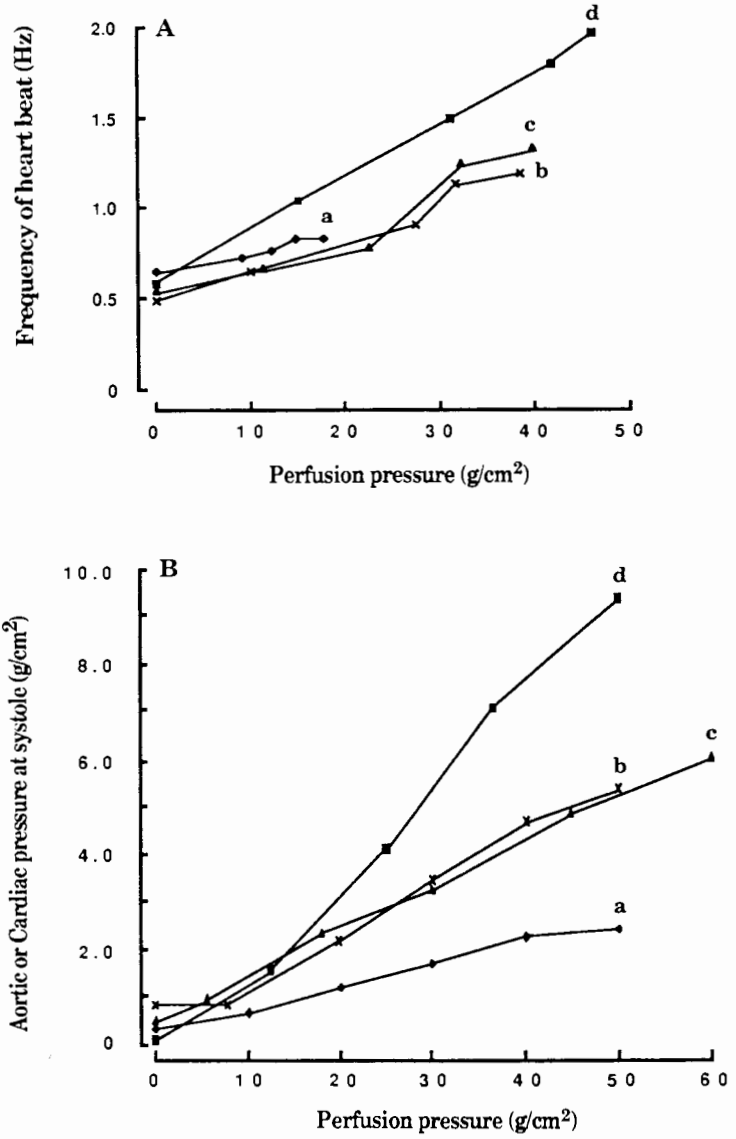

Fig. 9. Variation in (A) *heartbeat frequency (animals* a, b, c, d*) and in* (B) *pressure developed in the heart (animal* c*) and sternal artery (animals* a, b, d*) with variation in perfusion pressure (distension) in the perfused, isolated heart system of the lobster* Panulirus japonicus. *(Modified, with permission, from Kuramoto and Ebara [1984a].)*

maker system, or inotropically, via an effect either on the pacemaker system or on the myocardium directly. Longer-term regulation of the heart could also occur via the influence of the pericardial organ, which is innervated,

known to contain many cardioactive substances (Cooke and Sullivan 1982), and located strategically on the venous return directly before the heart.

Given the presence of innervated muscle in the aortic outflow valves (discussed below), it is possible that crustaceans may also be able to control the output resistance of the heart by variation of valvular tonus. Simultaneous contraction of the muscle of all valves could provide an increase in output resistance and thus limit outflow into the arterial system generally. Although this particular situation would appear to be clearly maladaptive, possible adaptive effects of occluding specific sets of valves are discussed below.

It seems likely from the above that cardiac output in crustaceans can be adjusted by a balance of neural and local influences involving control of heart rate and both end diastolic volume and systolic ejection. Indirect measurements of cardiac output in crustaceans (see McMahon and Wilkens [1983] for review) indicate that cardiac output may vary up to 10-fold. Measurements of cardiac performance under various conditions (discussed below) suggest that changes in stroke volume may be the more important regulator of cardiac output in the crustacean open circulatory system. Additional work is, however, urgently needed in this area before we can evaluate the roles played by any of these suggested mechanisms in controlling this variation.

Control of the Distribution of Cardiac Output

The distribution vessels of crustaceans do not contain smooth muscle and thus lack the regulatory capability of vertebrate arterioles. This has been taken to indicate that crustaceans have little or no control over the distribution of hemolymph flow. To the contrary, recent work demonstrates that flow can vary dramatically between arterial outputs in the crab *C. magister* (Bourne and McMahon 1990). Figure 10 shows flow velocities recorded simultaneously from ophthalmic and left antennary arteries and the posterior arterial complex by pulsed Doppler transducers in an unrestrained crab. In both panels *A* and *B* the animal was quiescent, but a few seconds before the measurements shown in panel *B* the animal had been disturbed and responded with a brief period of activity. Panel *B* clearly shows marked depression of flow through the antennary artery and enhancement of flow through both the ophthalmic artery and through the posterior arterial complex, clear evidence for the redistribution of cardiac output. Several possible control mechanisms can be postulated.

Role of the Cardiac Valves

Several lines of evidence point to use of valvular mechanisms in regulation of hemolymph distribution. Recently, Kihara and Kuwasawa (1984) and Ki-

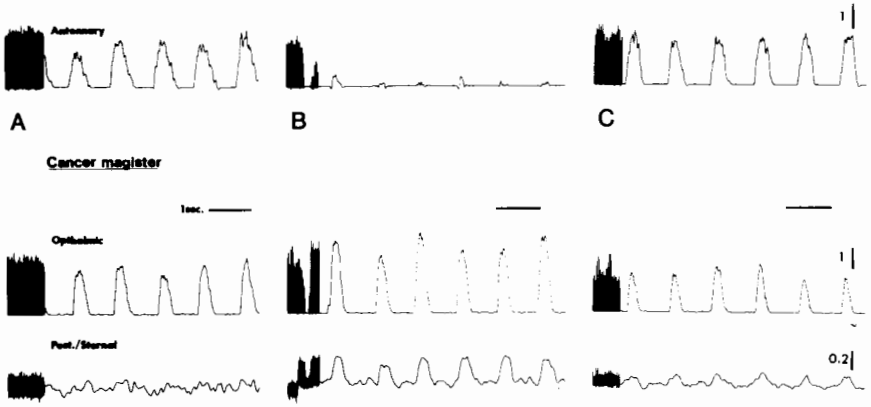

Fig. 10. Velocity of hemolymph flow recorded simultaneously in the left antennary and ophthalmic arteries and in the posterior arterial complex of Cancer magister. *Panel* A *shows the velocity before, panel* B *30 s after, and panel* C *12 min after a brief period of movement and struggling. Note the temporary redistribution of flow between the arterial outflows. Vertical calibration bars represent voltage output from a pulsed Doppler flow system. In the antennary artery 1 V is equivalent to 3.03 mL/min mean blood flow.*

hara, Kuwasawa, and Yazawa (1985) have demonstrated complex innervation of the aortic valves of the giant isopod *Bathynomus doederleini* (fig. 11). The arterial system of this isopod is characterized by a single anterior median artery, a pair of anterior lateral arteries arising from the anterior margin of the heart, and three or more pairs of lateral arteries arising from the lateral aspect of an elongated heart (figs. 1, 11). These authors demonstrate both excitatory and inhibitory nerve supply to the valves of the anterior arteries and show that the stimulation of the excitatory innervation is associated with contraction of the valve and attenuation of the pulse recorded in the associated artery, while stimulation of the inhibitory axons diliates some valves and is associated with an increase in the arterial pulse (fig. 12; Kihara and Kuwasawa 1984). Kihara et al. (1985) detail the innervation of the valves of both the anterior three arteries and the five pairs of lateral arteries. Whereas the structure of the valves seems similar in each case, there are differences in innervation of the three anterior valves. The anterior median valve receives only excitatory innervation. The paired anterolateral artery valves receive both excitatory and inhibitory innervation, while the valves of all the five pairs of lateral arteries apparently receive only inhibitory innervation (fig. 11). It is interesting that the muscles of the lateral valves can

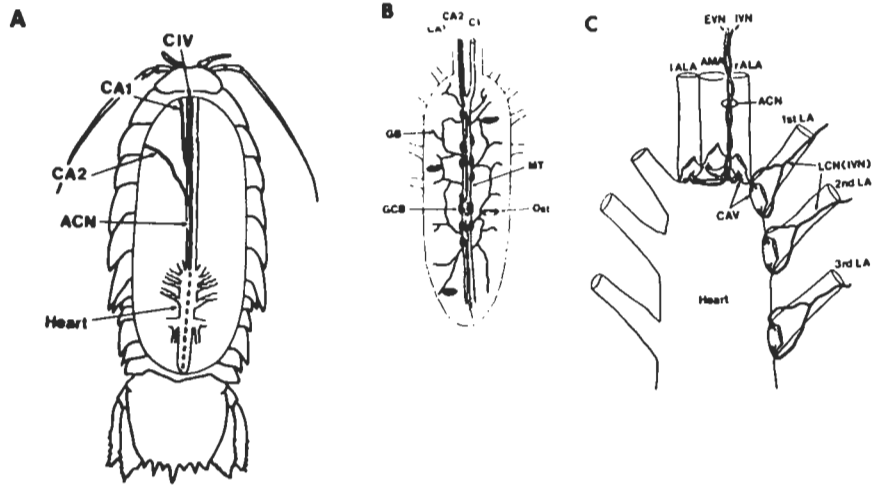

Fig. 11. Circulatory anatomy and cardiac neuroanatomy in Bathynomus doederleini. A, *Heart location and innervation;* B, *anatomy of the heart and cardiac ganglion;* C, *valve structure and innervation;* ACN, *anterior cardiac nerve;* ALA, *anterior lateral artery (right and left);* AMA, *anterior median artery;* CAV, *cardioarterial valves;* CIV, *cardioinhibitor and valve nerve;* CA1 *and* CA2, *first and second cardioaccelerator nerves;* EVN, *excitatory valve nerve;* GB, *branch of cardiac ganglion;* GCB, *cell body of cardiac ganglion;* IVN, *inhibitory valve nerve;* LA, *lateral arteries;* LCN, *lateral cardiac nerve—from* IVN; MT, *main trunk of cardiac ganglion;* Ost., *ostium. (Diagrams* A *and* B *are from Kihara and Kuwasawa* [*1984*]. *Diagram* C *is from Kihara et al.* [*1985*]. *Reproduced with permission.)*

be excited and show increased contraction in response to both 5-HT and octopamine (Fujiwara and Kuwasawa 1987).

It is thus clearly possible that the distribution of cardiac output in the isopod can be independently controlled by central variation of the neural and/or neurohormonal output to these sets of valve muscles. If we take into account the differences in nerve supply to the separate arterial outputs, the system appears sufficiently complex to effectively control the fractional distribution of the cardiac output to the three major networks of the isopod circulation. The three networks are shown diagrammatically in figure 1*C* as follows: first, via the anterior median artery to the esophagus, cerebral ganglion, eyes, and antennae; second, via the anterolateral arteries to the skeletal muscle of the cephalon; and third, via the lateral arteries to the thoracopods, the pleopods (swimmerets, respiratory surface), and the telson. The potential importance of these control mechanisms in redistribution of

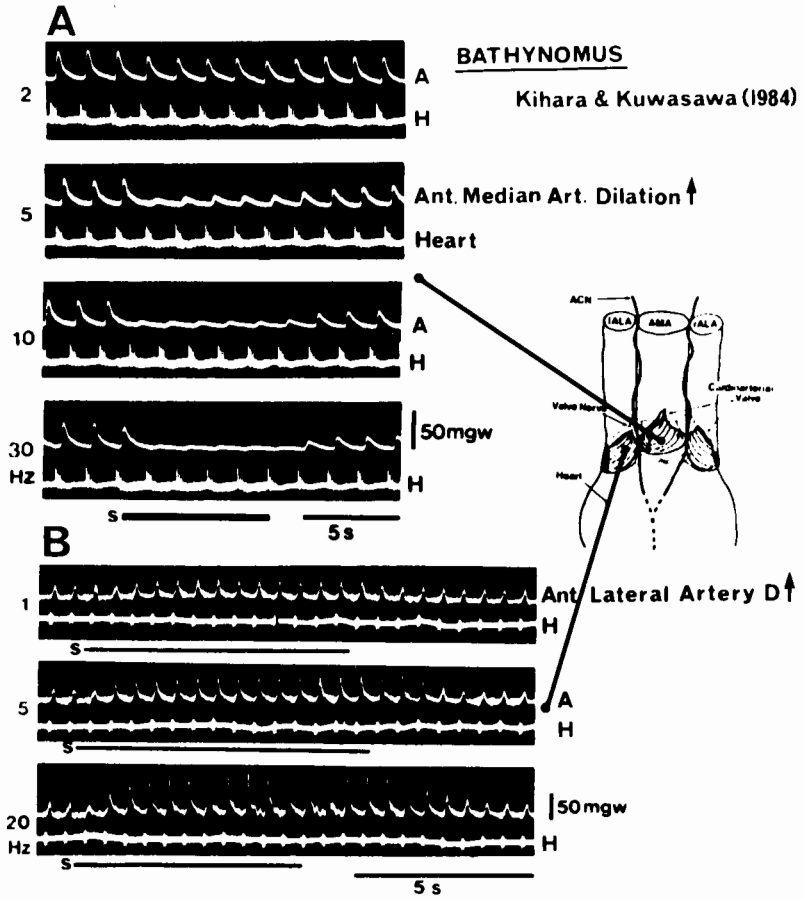

Fig. 12. Alteration of the mechanogram of hemolymph flow in (panel A*) the anterior median and (panel* B*) left anterior lateral arteries of* Bathynomus doederleini *during stimulation of nerves to the arterial valves. The upper trace (*A *at right of graph) in each pair is a mechanogram of the artery. Dilation of the artery is an upward deflection. The lower trace (*H*) is an extracellular recording of heart electrical activity. Numbers at left of each trace are the frequencies of stimulation applied. Activation of the nervous system of the valve in the anterior median artery clearly causes valve contraction (flow suppression) (panel* A*). Valve relaxation (flow enhancement) accompanies stimulation of the valves of the anterior lateral arteries (panel* B*). (Reproduced, with permission, from Kihara and Kuwasawa [1984].)*

hemolymph flow during locomotor activity, or in diverting hemolymph through the respiratory pathways, is discussed below.

Similar differential innervation of the valves also occurs in some decapod

crustaceans, in which the anterior cardioarterial valve is innervated separately from the lateral and posterior valves (Alexandrowicz 1932). Details of the excitatory and inhibitory fiber innervation to each valve have not been worked out. Evidence gathered from recent pharmacological experiments, however, suggests that a system similar to that shown in the isopods may also function in the decapods. Kuramoto and Ebara (1984*b*) confirmed the muscular nature of the semilunar valves present at the origin of each of the major arterial outputs of the heart of the lobster *Panulirus japonicus.* Rhythmic contractions of, for instance, the valve musculature of the posterior abdominal artery were observed, but, at least in the isolated heart, these were not in phase with systole. The first evidence to implicate lobster aortic valves in distribution of hemolymph flow was Kuramoto and Ebara's (1984*b*) observation that valve muscles of the anterior and posterior arteries differ in their response to neurohormonal modulator substances. Octopamine causes contraction of the posterior valves but may relax anterior valves, whereas dopamine, noradrenaline, acetylcholine, and the pentapeptide proctolin all cause contraction of both sets of valves. More recently, Kuramoto (personal communication) has shown that contraction of the valves at the posterior abdominal artery causes diminution of the arterial pressure but an increase in ventricular force (fig. 13), providing further evidence of a role for these valves in distribution of cardiac output. The potential multiple action of these neurohormones on circulatory function is demonstrated by further recent studies (T. Kuramoto, personal communication) demonstrating that 5-HT increases the rate and ventricular pressure of cardiac beat while inhibiting valve closure. This combination could produce a rapid increase in hemolymph pressure and suggests that this could be involved in the "baroreceptor" responses proposed by Burggren et al. (1990). It is important to note here that the action of these neurohormonal agents may differ between species. Acetylcholine and octopamine, at least, appear to have opposite actions on the heart valves of *Panulirus* and *Bathynomus.*

There is thus clear evidence for neurohormonal control of arterial valve tonus in *Panulirus* that could function to divert hemolymph away from the posterior abdominal artery and towards the head and other anterior circuits or vice versa. Active closure of the posterior valves might be important in preventing reflux of hemolymph from the abdominal aorta during the massive abdominal flexions that occur in escape responses of the lobster (Kuramoto and Ebara 1984*b*) but might also allow the control of hemolymph flow between various arterial systems as outlined for *Bathynomus* elsewhere. Active control of valve aperture appears to be a prime candidate for the redistribution of cardiac output reported above for the brachyuran crab *C. magister* (fig. 10).

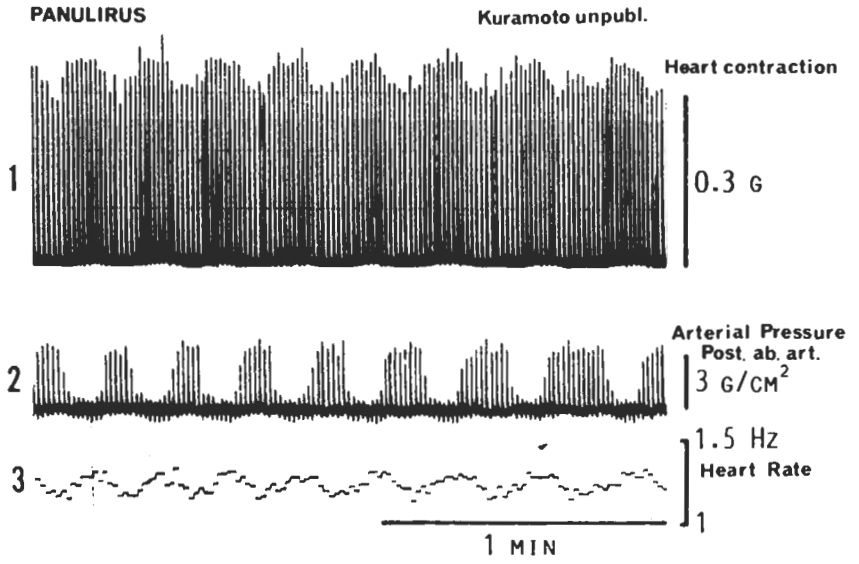

Fig. 13. Simultaneous effects of contraction of the cardioarterial valve on heart tonus and pressure recorded in the abdominal artery in the lobster Panulirus japonicus. 1, *Force of heart contraction;* 2, *abdominal artery pressure;* 3, *heart rate (measured by cardiotachometer). Rhythmic contraction of the cardioarterial valve was induced by perfusion with 10 nmol* $\times L^{-1}$ *octopamine. Periods of valve contraction can be inferred from the associated decrease in the arterial pulse (see text). (Previously unpublished material reproduced with permission from T. Kuramoto.)*

Local Distribution of Hemolymph Flow

Following this demonstration of redistribution of cardiac output between arterial systems, we now turn to the possibility of mechanisms that can distribute hemolymph flow within an arterial unit. These include both further valvular systems and more specialized structures.

As an illustration of the latter, the cor frontale is a structure positioned on the anterior aorta immediately before the extensive hemolymph supply to the cerebral ganglion. This organ has innervated muscle, whose contractility is facilitated by 5-HT and a peptide fraction from the pericardial organ (Steinacker 1979). Steinacker (1978) describes similar structures downstream in this circulation prior to the optic lobe circulation and suggests that all function as booster pumps for those areas that have dense capillary beds (i.e., have a high resistance to hemolymph flow) within that perfusion unit (fig. 3). Clearly, they could also function to further redistribute the output within this arterial circulation.

Valvular structures have also been reported at other areas in the circulation. Within the arterial system, valves are located at the entrances of the lateral (segmental) arteries leaving the posterior abdominal artery in Malacostraca (Alexandrowicz [1913], as cited in Maynard [1960]). These may function to restrict the back flow of hemolymph during the powerful tail flexions that occur during escape swimming, or they may have some regulatory function at the segmental level.

One-way (rectifying) valves have also been demonstrated in the gill filaments of the crab *C. maenas* (Taylor and Taylor 1986). These apparently passive structures limit the back flow of hemolymph at the efferent outflow of each lamella. This may be important in ensuring that pressure changes occurring in the branchial cavity and elsewhere (i.e., those associated with reversed ventilatory pumping in aquatic forms, or with movement generally; see below) act only to enhance the flow of hemolymph returning to the heart. No muscle has yet been reported in these valvular structures, but it is interesting to note here that Maynard (1960) cites Parrot (1938) as observing that administration of tissue extracts increases hemolymph flow through the isolated crab gill. This suggestion that hemolymph regulation might occur at this level also is intriguing, but the present authors have not been able to confirm this reference. Should further investigation reveal muscle tissue in the valves reported by Taylor and Taylor (1986), this would provide a possible locus for such regulation.

Facilitating Effects of External Pressure Fluctuations

Pressure changes associated with noncardiac events such as ventilatory pumping are transferred to the circulatory system (Blatchford 1971; Burggren et al. 1985; fig. 14). We have recently used external pressure changes to measure compliance in the isolated perfused gill of the crab *Cancer anthonyi* (fig 15). The internal volume of the gill can be estimated from measurements of gill surface area (method from Greenaway [1984]) and estimates of the average diameter of the hemolymph luminal space (60 μm, taken from Aldridge and Cameron 1979). Thus:

$$\text{Gill hemolymph volume} = \tfrac{1}{2} \times \text{surface area} \times \text{lumen thickness}.$$

This calculation yields an individual gill volume of approximately 1 mL (gill no. 7 of crabs of 610–645 g wet weight). This volume is very sensitive to change in the transmural pressure difference between hemolymph and branchial fluid spaces (fig. 15). The volume change is greatest when the external (branchial) pressure changes from "negative" (subambient) to

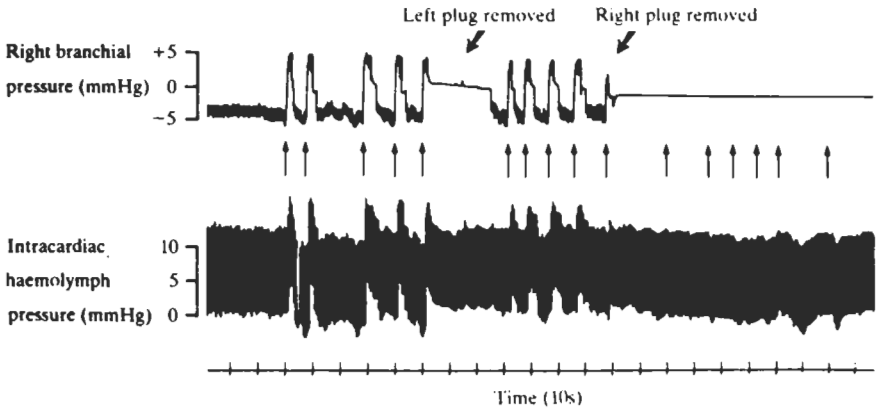

Fig. 14. Effects of ventilatory pumping pressures on hemolymph pressures recorded in the circulatory system of Cardisoma guanhumi. *Upward deflections in the branchial cavity pressure trace are bouts of reversed scaphognathite pumping. These deflections are transmitted to the circulatory system as shown. Opening of the branchial cavities to air removes the pressure pulse from both systems. (Reproduced, with permission, from Burggren et al. [1985].)*

"positive" (above ambient). Essentially similar effects of decrease in transmural pressure on perfused gill conductance are presented by Taylor (1990). Thus, the transmural pressure difference resulting from reversed ventilatory pumping periods occurring commonly in crabs (fig. 16) may reduce gill hemolymph volume. In fact, a reversed pumping bout inducing a change in transmural pressure of approximately 3–4 cmH_2O (2.2–3 Pa; fig. 16) would practically empty the gill of contained hemolymph (fig. 15). This reasoning assumes that hemolymph pressure in the gills approaches zero during reversed pumping. This is likely, since cardiac arrest often accompanies periods of reversed pumping in crabs (McMahon and Wilkens 1977, 1983). Explanations of the function of these periods of reversed pumping remain speculative (McMahon and Wilkens 1983), but the evidence above suggests that one possibility could be to reduce temporarily any physiological dead space resulting from stagnant hemolymph pools within the gill. Taylor (1990), however, reasons that effects on branchial hemolymph flow are likely to be muted by pressure equilibration across the branchiostegal wall, at least during bilateral reversed pumping. Periods of unilateral reversed pumping, such as are commonly observed in crabs (McMahon and Wilkens 1983), however, might still cause marked effects on branchial hemolymph flow.

McMahon and Wilkens (1983) further reasoned that the usually negative

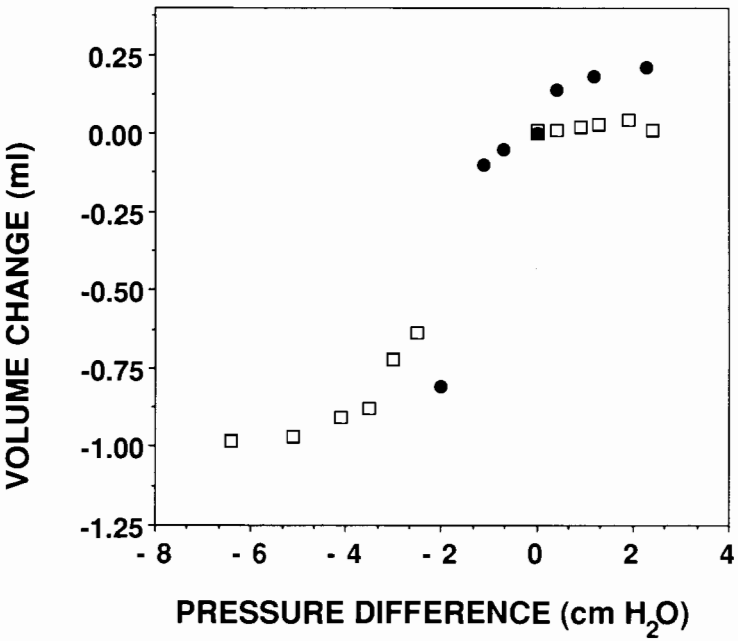

Fig. 15. The relation between transmural pressure (hemolymph space − branchial water space) vs. volume within the hemolymph space in the isolated perfused gill of Cancer anthonyi. *Volume (the ordinate) is expressed as the volume above or below the residual volume (= zero) which occurs when the pressure difference across the gills is zero. Gills were perfused by the method of Burnett (1984) and Burnett and McMahon (1985). During the experiment the compliance of individual gills (no. 7) at 15° C from two individual crabs (represented by different symbols) was measured by stopping the internal perfusate flow and attaching a glass pipette to the efferent cannula to record volume changes within the gill. Pressure differences across the gills were created by passing a stream of seawater across the gills in the normal direction (countercurrent to the flow of perfusate and blood) under a slight positive pressure (see Burnett [1984] for details). Fine pressure adjustments were obtained with a needle valve.*

pressure created by the beating of the scaphognathites within the branchial cavity may act on the very fine membranes of the gills to increase the diameter of the lamellar hemolymph pathways so as to decrease resistance and thus increase gill hemolymph flow. This conclusion is only partly supported by the present data for *C. anthonyi.* Both the decrease in branchial chamber pressure (these pressures are normally negative) and the increase in branchial (pericardial) hemolymph pressure that occur at this time (fig. 16) would increase the transepithelial pressure across the gills. The resultant

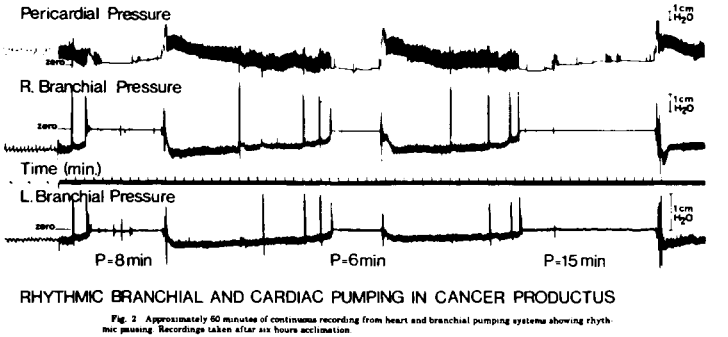

Fig. 16. Correlated cardiovascular and ventilatory pumping pressures during ventilatory patterning in Cancer productus, *showing fluctuations in pericardial pressure during periods of linked respiratory and cardiovascular arrest. (From McMahon and Wilkens [1977].)*

gill inflation (fig. 15) must facilitate hemolymph flow initially. Further increases in branchial pumping, resulting in more negative branchial chamber pressures, are not associated with large changes in gill volume and may not greatly enhance conditions for branchial hemolymph flow, at least for *C. anthonyi* under the perfusion conditions used.

Movements of the limbs, as for instance in locomotion, produce internal pressure pulses. These have been shown to affect hemolymph flow measured in the posterior abdominal artery of the lobster (Jorgensen et al. 1989). Generally, enhanced flow could result directly (i.e., via the "muscle pump") or from actions elsewhere in the system to provide increased return flow and thus increased cardiac output.

Strong evidence for a controlled ischemia involving two alternate respiratory-exchange circuits has been published for the amphibious crab *Holthuisana transversa* by Taylor and Greenaway (1984). When this animal is submerged the gills are the primary gas exchangers; however, an alternate exchange organ in the walls of the branchial cavity predominates when these animals breathe air. Using microsphere injection these authors showed that the percentage of hemolymph flow diverted to the gills (away from the alternate gas exchanger in the lining of the branchial cavity wall) decreased from 87% ± 6% to 20% ± 7% in transition between water and air, indicating extensive control over flow to these two vascular beds. Clearly, several of the mechanisms outlined above could be involved in this diversion. Taylor and Greenaway (1984) suggest that scaphognathite activity in water could act to decrease gill resistance and increase gill hemolymph flow as outlined above. However, the subambient pressure would seem equally likely to cause deflection of the thinner branchiostegal membranes and thus

potentiate flow through the alternate circuit. Clearly, more work is needed to elucidate the extent to which the above mechanisms might be involved.

Neural Coordination between Ventilatory and Cardiac Pumping

The functional correlation between circulatory and ventilatory pumping performance proposed for the decapod crustaceans above suggests that complex interactions between heart and cardiac performance might occur, as has been suggested for fish (Randall and Smith 1967). In aquatic crustaceans, interactions may occur on a beat-to-beat basis (Young and Coyer 1979) or over a longer time span (Young 1978; see McMahon and Wilkens [1983] for review). In the latter case the periods of ventilatory shutdown (pauses) commonly seen in decapod crustaceans are normally correlated with periods of cardiac arrest (fig. 16). Cardiac arrest is also commonly, but not always, associated with periods of reversed pumping, especially those of shorter duration. Clearly there is a tight correlation between the outputs of the central pattern generators associated with ventilation and heart performance such that the heart does not continue to beat during periods of apnea or while reflux of exhalant water over the gills can occur. Interestingly, these correlations are apparently not seen in air-breathing decapod crustaceans (McMahon and Burggren 1979; McMahon and Wilkens 1983); perhaps the presence of air in the branchial chambers allows sustained gas exchange and thus sustained cardiac performance during the periods of intermittent ventilation typical of this group.

The System at Work

In a majority of animals, although not in insects, a principal function of the circulatory system is to transfer O_2 and CO_2 between the respiratory organs and the tissues. Thus, the abilities of a circulatory system might best be tested by an examination of its capacity to deliver additional oxygen to exercising tissues during activity, or in maintaining oxygen delivery during acclimation to environmental stresses, such as hypoxic exposure.

The potential importance of change in hemolymph distribution during activity in isopods may be seen in the recent work of Tanaka, Kuwasawa, and Fujiwara (1986), who show that initiation of locomotor activity in the swimmerets (branchiae) of *B. doederleini* is accompanied by (1) activity in the cardioaccelerator nerve associated with increased heart pumping and (2) activity in the excitatory valve nerve that would divert hemolymph away from the three anterior arteries and toward the swimmerets. Cessation of swimmeret activity is associated with decreased cardioaccelerator activity

and activity in the inhibitory valve nerve that would divert hemolymph to the anterior lateral arteries. It is thus clear that an increase in swimmeret activity is associated with an increase in cardiac output and redistribution of that output to preferentially supply the swimmeret area. In these experiments this is clearly a short-term diversion, organized by the CNS via nerve supply to the cardiac ganglion, heart muscle, and valve muscle systems. Other work by Kuwasawa and co-workers, however, has shown that these valvular systems also react to neurohormones such as 5-HT and octopamine, suggesting a route for longer-term control. It is important to point out here that, at least in *Bathynomus,* the swimmerets are not only locomotor structures but also bear the gills. Thus, this diversion also increases hemolymph supply to the respiratory structures. Particularly in long-term activity this may be equally as important as the increase to the locomotor muscles.

A similar situation may also occur during activity in decapod crustaceans. Fick estimates of cardiac output indicate that a marked increase accompanies activity. Although increases in rate are involved in some cases, increases in stroke volume may play the more important role (see McMahon [1981] for review). In *C. magister* strenuous enforced pedestrian (and pugilistic) activity was associated with a 2.5-fold increase in cardiac output while heart rate increased only 38% (McMahon, McDonald, and Wood 1979). In sustained swimming in *Callinectes sapidus,* heart rate increased by 61%, but, nonetheless, an increase in stroke volume played an important role in increased cardiac output (Booth, McMahon, and Pinder 1982). No studies have yet investigated whether redistribution of hemolymph flow is associated with sustained locomotor activity in decapod crustaceans, but recent work of Bourne and McMahon (1990; see fig. 10) suggests that this may occur, at least over the short term.

During hypoxic exposure, adjustments to many physiological systems are necessary to maintain O_2 delivery to the tissues (see McMahon [1988] for review). One of the adjustments used by crustaceans is to increase the hemolymph supply to the gills to load additional O_2 for transfer to the tissues. The required increase in cardiac output rarely involves an increase in heart rate, which either remains constant or declines (McMahon and Wilkens 1975; Taylor 1976; Butler, Taylor, and McMahon 1978). An increase in cardiac output, nonetheless, occurs during hypoxia, as suggested by Fick calculations in *Homarus* (McMahon and Wilkens 1975) and confirmed by direct measurements of cardiac output via the thermal dilution method (Jorgensen et al. 1982). The latter study clearly shows (fig. 17) that, in the crab *C. magister,* the increased cardiac output occurring in progressive hypoxia results almost entirely from an increase in stroke volume. This figure also shows that in very severe hypoxia (ambient $P_{O_2} < 30$ Torr) both heart rate and

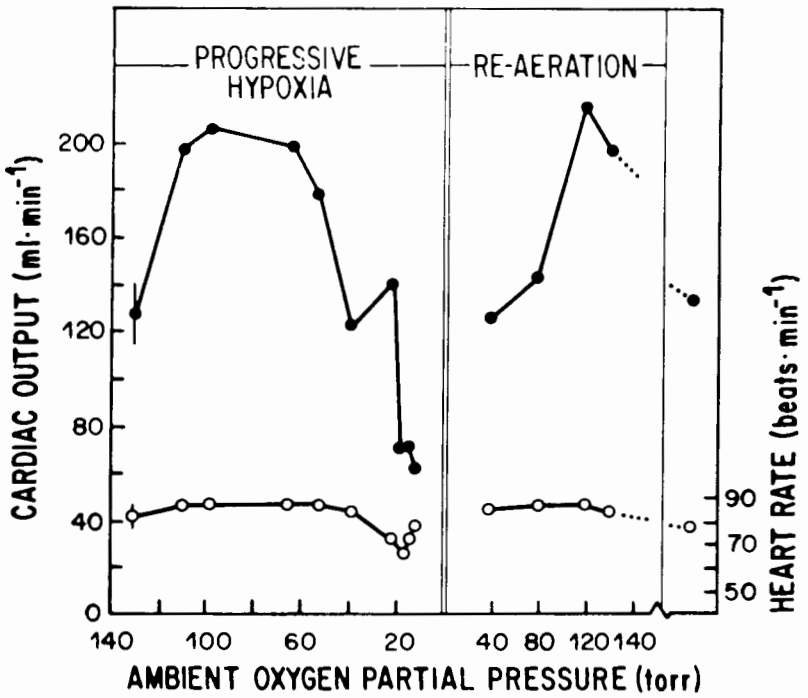

Fig. 17. Variation in measured cardiac output (closed symbols) and heart rate (open symbols) during progressive hypoxia and recovery in the crab Cancer magister. *(From McMahon [1988]; data from Jorgensen et al. [1982].)*

stroke volume decrease. This result presumably occurs as the compensatory mechanisms, both at the level of the heart and at higher centers, become impaired.

Increases in rate and depth of ventilation occurring both during activity and during exposure to hypoxia result in decreased pressures (more negative) in the branchial chambers. The suggestion of McMahon and Wilkens (1983) that this might enhance branchial circulation is, however, apparently not supported by the available data (see fig. 15 and discussion above).

Conclusion

This review of recent work on the open circulatory systems of several advanced crustaceans reveals that these are complex, highly efficient, and tightly regulated systems capable of a degree of tissue perfusion that rivals that of vertebrate closed systems. The heart consists of a muscular chamber

(ventricle) suspended in a second chamber (pericardial cavity), which acts as a primer chamber of variable volume. The neurogenic excitation system provides a much higher degree of local control over heart performance than occurs in the all-or-none hearts of the vertebrates. A considerable capacity for autoregulation may also occur at the level of the cardiac ganglion. This, together with complex neural control over both heart rate and stroke volume, may allow wide control over cardiac output. Despite the absence of muscle in the peripheral vascular system, these open circulatory systems apparently exhibit a high degree of control over the distribution of cardiac output between the various arterial outflow systems and perhaps at the organ level. This review suggests that the commonly expressed view of the crustacean open circulatory systems as poorly designed structures capable of only sluggish performance is incorrect, at least for the more advanced decapod crustaceans. In fact, these crustacean open circulatory systems appear to be functionally equivalent to the closed systems of the vertebrates despite the radical difference in design.

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Literature Cited

ALDRIDGE, J. B., and J. N. CAMERON. 1979. Carbon dioxide exchange in the blue crab *Callinectes sapidus* (Rathbun). J. Exp. Zool. 207:321–328.

ALEXANDROWICZ, J. S. 1913. Zur Kenntnis des sympathetischen Nervensystems einiger Wirbellosen Z. Allg. Physiol. 14:358–376.

———. 1932. The innervation of the heart of the Crustacea. I. Decapoda. Q. J. Microsc. Sci. 75:181–249.

BAUMANN, H. 1921. Das Gefaßsystem von *Astacus fluviatilis (Potamobius astacus)* Z. Wiss. Zool. 118:246–342.

BELMAN, B. W. 1975. Some aspects of the circulatory physiology of the spiny lobster *Panulirus interruptus*. Mar. Biol. 29:295–305.

———. 1976. New observations on blood pressure in marine Crustacea. J. Exp. Zool. 196:71–78.

BELMAN, B. W., and J. J. CHILDRESS. 1976. Circulatory adaptations to the oxygen mini-

mum layer in the bathypelagic mysid *Gnathophausia ingens*. Biol. Bull. 150:15–37.

BLATCHFORD, J. G. 1971. Hemodynamics of *Carcinus maenas* (L). Comp. Biochem. Physiol. 39A:193–202.

BOOTH, C. E., B. R. MCMAHON, and A. W. PINDER. 1982. Oxygen uptake and the potentiating effects of hemolymph lactate on oxygen transport during exercise in the blue crab, *Callinectes sapidus*. J. Comp. Physiol. 148:111–121.

BOURNE, G. B., D. D. JORGENSON, B. R. MCMAHON, L. E. BURNETT, and P. L. DEFUR. 1988. An examination of cardiac performance in the Dungeness crab *Cancer magister*. In Society for Experimental Biology. Proceedings, Lancaster meeting, 1988. (Abstr.)

BOURNE, G. B., and B. R. MCMAHON. 1990. Control of cardiac output and its distribution in crustacean open circulatory systems. J. Physiol. (in press).

BULLOCK, T. H., and G. A. HORRIDGE. 1965. Structure and function in the nervous systems of invertebrates. W. H. Freeman, San Francisco.

BURGGREN, W. W., A. J. PINDER, B. R. MCMAHON, M. DOYLE, and M. G. WHEATLY. 1990. Heart rate and hemolymph pressure responses to hemolymph volume changes in the land crab *Cardisoma guanbumi:* evidence for "baroreflex" regulation. Physiol. Zool. 63:167–181.

BURGGREN, W. W., A. J. PINDER, B. R. MCMAHON, M. G. WHEATLY, and M. DOYLE. 1985. Ventilation, circulation and their interactions in the land crab, *Cardisoma guanhumi*. J. Exp. Biol. 117:133–154.

BURNETT, L. E. 1979. The respiratory function of hemocyanin in the spider crab *Libinia emarginata* and the ghost crab *Ocypode quadrata* in normoxia and hypoxia. J. Exp. Zool. 210:289–300.

———. 1984. Carbon dioxide excretion across perfused crab gills: facilitation by carbonic anhydrase. Am. Zool. 24:253–264.

BURNETT, L. E., P. L. DEFUR, and D. D. JORGENSEN. 1981. Application of the thermal dilution technique for measuring cardiac output and assessing stroke volume in crabs. J. Exp. Zool. 218:165–173.

BURNETT, L. E., and B. R. MCMAHON. 1985. Facilitation of CO_2 excretion by carbonic anhydrase located on the luminal surface of the basal membrane of crab gill epithelium. Respir. Physiol. 62:341–348.

BUTLER, P. J., E. W. TAYLOR, and B. R. MCMAHON. 1978. Respiratory and circulatory changes in the lobster (*Homarus vulgaris*) during long term exposure to moderate hypoxia. J. Exp. Biol. 73:131–146.

CAMERON, J. N., and T. A. MECKLENBERG. 1973. Aerial gas exchange in the coconut crab *Birgus latro* with some notes on *Gecardoidea lallandii*. Respir. Physiol. 19: 245–261.

COOKE, I. M., and R. E. SULLIVAN. 1982. Hormones and neurosecretion. Pages 206–290 in H. L. ATWOOD and D. C. SANDEMAN, eds. Neurobiology: structure and function. Vol. 3 of D. E. BLISS, ed. The biology of Crustacea. Academic Press, New York.

DELALUE, J. C., and A. HOLLEY. 1976. Neural regulation of the heart muscle in an isopod crustacean: acceleration and peripheral inhibition. J. Exp. Biol. 64:345–356.

FRANK, O. 1895. Zur Dynamik des Hertzmuskels. Z. Biol. Munich 32:370. Trans. C. R. CHAPMAN and E. WASSERMAN. Am. Heart J. 58 (1959):282–317, 467–478.

FUJIWARA, Y., and K. KUWASAWA. 1987. Neurophysiological and pharmacological studies on the cardioarterial valve of *Bathynomus doerderleini.* Zool. Sci. 4:984.

GEORGE, G. C., K. K. NAIR, and P. T. MUTHE. 1955. The pericardial membrane and its role in crustacean circulation. J. Anim. Morph. Physiol. 2:73–78.

GIBBONS, C. A., and R. E. SHADWICK. 1988. Mechanical design in the arteries of lower vertebrates. Presented at 2d International Congress of Comparative Physiological Biochemistry, Baton Rouge, La., August.

GREENAWAY, P. 1984. The relative importance of the gills and lungs in the gas exchange of amphibious crabs of the genus *Holthuisana.* Aust. J. Zool. 32:1–6.

GREENAWAY, P., and C. A. FARRELLY. 1990. Vasculature of the gas-exchange organs in air-breathing brachyurans. Physiol. Zool. 63:117–139.

HARTLINE, D. K. 1979. Integrative physiology of the lobster cardiac ganglion. Am. Zool. 19:53–65.

JORGENSON, D. D., C. ASAY, T. L. DANIEL, and A. P. FARRELL. 1990. Circulatory responses to pedal locomotion in the lobster. Am. Zool. (in press).

JORGENSON, D., G. B. BOURNE, L. E. BURNETT, P. L. DEFUR, and B. MCMAHON. 1982. Circulatory function during hypoxia in the Dungeness crab *Cancer magister.* Am. Zool. 22:958A.

KIHARA, A., and K. KUWASAWA. 1984. A neuroanatomical and electrophysiological analysis of nervous regulation in the heart of an isopod crustacean *Bathynomus doerderleini.* J. Comp. Physiol. 154:883–894.

KIHARA, A., K. KUWASAWA, and T. YAZAWA. 1985. Neural control of the cardioarterial valves in an isopod crustacean *Bathynomus doerderleini:* excitatory and inhibitory junction potentials. J. Comp. Physiol. 157:529–536.

KURAMOTO, T., and A. EBARA. 1984*a.* Effects of perfusion pressure on the isolated heart of the lobster, *Panulirus japonicus.* J. Exp. Biol. 109:121–140.

———. 1984*b.* Neurohormonal modulation of the cardiac outflow through the cardioarterial valve in the lobster. J. Exp. Biol. 111:123–130.

———. 1988. Combined effects of 5-hydroxytryptamine and filling pressure on the isolated heart of the lobster, *Panulirus japonicus.* J. Comp. Physiol. 158:403–412.

KURAMOTO, T., and K. KUWASAWA. 1980. Ganglionic activation of the myocardium of the lobster *Panulirus japonicus.* J. Comp. Physiol. 139:67–76.

KURAMOTO, T., and H. YAMAGISHI. 1990. Physiological anatomy, burst formation, and burst frequency of the cardiac ganglion of crustaceans. Physiol. Zool. 63:102–116.

MCLAUGHLIN, P. A. 1983. Internal anatomy. Pages 1–53 in L. MANTEL, ed. Internal anatomy and physiological regulation. Vol. 5 of D. E. BLISS, ed. Biology of Crustacea. Academic Press, New York.

MCMAHON, B. R. 1981. Oxygen uptake and acid-base balance during activity in decapod crustaceans. Pages 299–335 in C. F. HERRIED and C. R. FORTNER, eds. Locomotion and energetics in arthropods. Plenum, New York.

———. 1985. Functions and functioning of crustacean hemocyanin. Pages 35–58 in J. LAMY, J.-P. TRUCHOT, and R. GILLES, eds. Respiratory pigments in animals: relation structure-function. Springer, Berlin.

———. 1988. Physiological responses to oxygen depletion in intertidal animals. Am. Zool. 28:39–53.

MCMAHON, B. R., and W. W. BURGGREN. 1979. Respiration and adaptation to the ter-

restrial habitat in the land hermit crab *Coenobita clypeatus*. J. Exp. Biol. 79:265–281.

McMahon, B. R., D. G. McDonald, and C. M. Wood. 1979. Ventilation, oxygen uptake and hemolymph oxygen transport following enforced exhaustive activity in the Dungeness crab *Cancer magister*. J. Exp. Biol. 80:271–285.

McMahon, B. R., and J. L. Wilkens. 1975. Respiratory and circulatory responses to hypoxia in the lobster *Homarus americanus*. J. Exp. Biol. 62:637–655.

———. 1977. Periodic respiratory and circulatory performance in the red rock crab *Cancer productus*. J. Exp. Zool. 202:363–374.

———. 1983. Ventilation, perfusion and oxygen uptake. Pages 289–372 in L. Mantel, ed. Internal anatomy and physiological regulation. Vol. 5 of D. E. Bliss, ed. Biology of Crustacea. Academic Press, New York.

Mangold, E. 1925. Weitere Studien zur Physiologie des Krebsherzens. II. Wärmestillstand und Wärmestarre des Herzens mariner Decapoden. Z. Vergleichende Physiol. 3:512–520.

Maynard, D. M. 1960. Circulation and heart function. Pages 161–226 in T. H. Waterman, ed. The physiology of Crustacea. Vol. 1. Metabolism and growth. Academic Press, New York.

Miller, W. S. 1895. The anatomy of the heart of *Cambarus*. Trans. Wis. Acad. Sci. 10: 327–338.

O'Mahoney-Damon, P. 1984. Heart rate of the land crab *Gecarcinus lateralis* during aquatic and aerial respiration. Comp. Biochem. Physiol. 79A:621–624.

Parrot, J. L. 1938. Propriétés des extraits alcooliques de système nerveux et de certains viscères de *Maia squinada*. C. R. Soc. Biol. 129:188–190.

Pearson, J. 1908. Cancer. Liverpool Mar. Biol. Comm. Mem. 16:1–209.

Randall, D. G., and J. C. Smith. 1967. The regulation of cardiac activity of fish in a hypoxic environment. Physiol. Zool. 40:104–113.

Shadwick, R. E., and J. M. Gosline. 1985. Mechanical properties of the *Octopus* aorta. J. Exp. Biol. 114:259–284.

Shadwick, R. E., C. A. Pollock, and S. A. Stricker. 1990. Structure and biomechanical properties of crustacean blood vessels. Physiol. Zool. 63:90–101.

Starling, E. H. 1918. The Linacre lecture on the law of the heart. Longmans Green, New York.

Steinacker, A. 1978. The anatomy of the decapod auxiliary heart. Biol. Bull. 154: 497–507.

———. 1979. Neural and neurosecretory control of the decapod crustacean auxiliary heart. Am. Zool. 19:67–75.

Tanaka, K., K. Kuwasawa, and Y. Fujiwara. 1986. Inhibitory and acceleratory cardiac reflex in *Bathynomus doerderleini*. Zool. Sci. 3:968.

Taylor, A. C. 1976. The respiratory responses of *Carcinus maenas* to declining O_2 tension. J. Exp. Biol. 65:309–322.

Taylor, A. C., and P. S. Davies. 1981. Respiration in the land crab *Gecarcinus lateralis*. J. Exp. Biol. 93:197–208.

Taylor, E. W., and P. J. Butler. 1978. Aquatic and aerial respiration in the shore crab *Carcinus maenas* (L) acclimated to 15°C. J. Comp. Physiol. 127:315–323.

Taylor, H. H. 1990. Pressure-flow characteristics of crab gills: implications for regulation of hemolymph pressure. Physiol. Zool. 63:000–000.

TAYLOR, H. H., and P. GREENAWAY. 1984. The role of gills and branchiostegites in gas exchange in a bimodally breathing crab, *Holthuisana transversa:* evidence for a facultative change in the distribution of the respiratory circulation. J. Exp. Biol. 111:103–121.

TAYLOR, H. H., and E. W. TAYLOR. 1986. Observations of valve-like structures and evidence for rectification of flow within the gill lamellae of the crab *Carcinus maenas* (Crustacea, Decapoda). Zoomorphology 106:1–11.

VOLK, L. 1988. The role of suspensory ligaments in modifying cardiac output in crustaceans. M.S. thesis. University of Calgary.

WILKENS, J. L. 1987. Cardiac and circulatory control in decapod Crustacea with comparisons to molluscs. Experientia 43:990–994.

WILKENS, J. L., L. A. WILKENS, and B. R. MCMAHON. 1974. Central control of cardiac and ventilatory pacemakers in the crab *Cancer magister.* J. Comp. Physiol. 90:89–104.

WOOD, C. M., and D. J. RANDALL. 1981. Hemolymph gas transport, acid-base balance, and anaerobic metabolism during exercise in the land crab (*Cardisoma guanhumi*) J. Exp. Zool. 218:23–35.

YAZAWA, T., and K. KUWASAWA. 1984. The cardioregulator nerves of hermit crabs: anatomical and electrophysiological identification of their distribution inside the heart. J. Comp. Physiol. 154A:871–881.

YOUNG, R. E. 1978. Correlated activities in the cardioregulator nerves and ventilatory system in the Norwegian lobster *Nephrops norvegicus* (L). Comp. Biochem. Physiol. 61A:387–394.

YOUNG, R. E., and P. E. COYER. 1979. Phase coordination in the ventilatory and cardiac rhythms of the lobster *Homarus americanus.* J. Exp. Biol. 82:53–74.