Neural control of the hearts in the leech, *Hirudo medicinalis*

II. Myogenic activity and its control by heart motor neurons

Anthony R. Maranto and Ronald L. Calabrese*

The Biological Laboratories, Harvard University, 16 Divinity Avenue Cambridge, Massachusetts 02138, USA

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Summary. 1. The electrical activity of the heart in *Hirudo medicinalis* is correlated with the rhythmic discharge of segmental heart motor neurons (HE cells). Excitatory junctional potentials from the HE motor neurons summate in the heart muscle cells and give rise to large plateau-like potentials with associated spikes called bursts.

2. Individual heart muscle cells isolated by enzymatic dissociation of the heart are capable of producing a myogenic polarization rhythm.

3. The peripheral branches of the HE motor neurons are capable of producing antidromic burst activity (peripheral neurogenic rhythm) independently of the heart's myogenic rhythm when central activity in the HE cells is experimentally suppressed. HE motor neurons synaptically interact with one another in the periphery: their peripheral bursts can be coordinated and orthodromic activity in an HE cell can elicit antidromic activity in other ipsilateral HE cells whose central activity is suppressed experimentally. Antidromic bursting in HE cells is not normally observed when they are expressing their normal central activity rhythm. These observations indicate that there is a peripheral nerve plexus comprising the HE cells' peripheral branches that is capable of spreading the HE cells' activity along the heart tube.

4. The heart produces a myogenic contractile rhythm as well as its polarization rhythm, and these two activities appear to be associated.

5. The HE motor neurons reset and entrain the heart's electrical and contractile myogenic rhythms. Since the HE cells' activity is the output of the heartbeat central pattern generator, the pattern generator must be the governing oscillator determining the animal's heartbeat.

* To whom offprint requests should be sent

Introduction

The leech *Hirudo medicinalis* has two hearts: muscular tube-like vessels that extend the entire length of the animal on either side. Each heart is innervated along its length by heart motor neurons (HE motor neurons), which occur as bilateral pairs in the third through eighteenth segmental ganglia of the ventral nerve cord (Thompson and Stent 1976a). The HE motor neurons extend out to the hearts through a bilateral set of anastomosing segmental vascular nerves which branch off the anterior ganglionic roots (Thompson and Stent 1976a). Associated with the vascular nerves is a net or plexus of fine nerves which branch extensively on the heart and surrounding tissues (Zerbst-Boroffka et al. 1982). The impulse activity of the HE cells is easily recorded from the vascular nerves with extracellular electrodes. This activity takes the form of rhythmic bursts, which are coordinated both along the length of the nerve cord and from side to side (Thompson and Stent 1976a, b; Calabrese and Peterson 1983). The activity pattern of the HE motor neurons has been shown to be the output of a heartbeat central pattern generator comprising a set of segmental heart interneurons (HN cells) (Thompson and Stent 1976b, c; Calabrese and Peterson 1983). The HN neurons control the activity of the HE motor neurons through rhythmic inhibition. The constriction patterns of the hearts match closely the activity pattern of the HE motor neurons, indicating that the heartbeat central pattern generator ultimately controls the constriction pattern of the hearts.

While the mechanism of action of the central pattern generator and its role in determining the constriction pattern of the heart have received much attention, the properties of the heart and its associated plexus have been largely ignored. As
early as 1914 Gaskell concluded that the hearts must be myogenic because they continued to beat rhythmically when all connections to the central nervous system (CNS) were severed. This seemingly clear result was complicated by the discovery that the peripheral branches of the HE motor neurons, recorded in the vascular nerves, produced bursts of impulses when all connections to the CNS were severed (Thompson and Stent 1976a). Thus it seems possible that the activity observed by Gaskell (1914) might arise from a peripheral neurogenic rhythm. In this study we establish that both myogenic and peripheral neurogenic rhythms exist, and we attempt to identify their role in generating the animal's heartbeat. Moreover, we show that the role of the central pattern generator is to entrain the myogenic rhythm of the hearts through the agency of the HE motor neurons.

Methods

Animals, physiological solutions, dissections, and intracellular recording were described in the previous paper (Maranto and Calabrese 1984).

Recording in situ. An in situ preparation was used to obtain simultaneous intracellular recordings from the heart motor neurons and the heart muscle cells. This preparation consists of a single segment of the leech body wall with the hearts still attached and innervated by a single ganglion (Fig. 1). The fourth segment, by the numbering system described in the following paper (Calabrese and Maranto 1984), was always used. Before cutting the segment away from the rest of the animal, nylon monofilament fishing line (2 pound test) was inserted into the lumens of both hearts. The connective tissue along the hearts' dorsal surfaces was carefully removed to permit impalement of the muscle cells with microelectrodes while preserving innervation. Next, the blood sinus surrounding the fourth ganglion was cut away and a thin strip of body wall beneath the ganglion was removed to allow transillumination. Once the connectives between the second and third and the fifth and sixth ganglia had been cut, the fourth segment was removed and pinned ventral side down in a Sylgard-coated dish. The fourth ganglion was twisted about its roots and pinned ventral side up. The hearts were then stretched along the monofilament that had been inserted into their lumens and the ends of the hearts were securely pinned. In a few experiments additional attached segments were similarly prepared. When heart muscle tension was monitored (method described in Calabrese and Maranto 1984) no monofilament inserts were used.

Isolation of muscle cells. Both heart tubes were dissected relatively cleanly from a leech and placed in 2 ml of a saline containing 5 mg protease (Sigma Type XIV from Streptomyces griseus). The tissue was agitated on a shaker table for 30–60 min at room temperature until the cells could be separated by gentle trituration. Longer enzymatic treatment was preferable to more vigorous trituration because the latter tended to decrease the yield of intact cells. The resulting suspension of cells was pelleted in a clinical centrifuge, washed, and resuspended three times with physiological saline to a final volume of 0.5 ml. To this volume was added 0.5 ml of a solution of low melting point agarose (BRL, Inc.; 10 mg ml in saline) at 30 °C. The mixture was then divided equally among four 35 mm culture dishes and allowed to gel at 4 °C. The dishes were then topped with L-15 culture medium (Maranto and Calabrese 1984) and stored in a humid chamber at 4 °C for up to a week before use. L-15 medium was replaced with three changes of saline before physiological recordings were made.

Results

Electrical activity of the heart

Simultaneous intracellular recordings from an HE motor neuron and a muscle cell in the ipsilateral heart are shown in Fig. 2. Each spike in an HE cell elicits a unitary excitatory junctional potential in the muscle cell (Thompson and Stent 1976; Maranto and Calabrese 1984). The junctional potentials normally summate and give rise to slow plateau-like regenerative potentials such as those illustrated in Fig. 2. Often, as was the case in this preparation, discrete action potentials were resolvable on the slow potentials. Amplitudes of the slow potentials ranged between 10–20 mV, and the action potentials rose as much as another 10–20 mV above this level to a membrane potential of approximately 0 mV. These slow potentials with their superimposed action potentials will be referred to as bursts. The bursts in the leech heart resemble spontaneous bursting pacemaker potentials in neurons (Gorman et al. 1980), smooth muscle (Prosser 1974), and other invertebrate hearts (Maye et al. 1974; Miller 1968).

Myogenicity of the heart

To determine whether the electrical activity rhythm recorded in the leech heart has a myogenic compo-
ment, muscle cells were isolated by enzymatically dissociating the heart. When recordings were made from individual muscle cells in culture, we found that the cells were capable of producing rhythmic bursts (Fig. 3A). Each burst consisted of a 10–15 mV slow potential with superimposed 5–10 mV action potentials. The interburst period could be altered by injecting steps of current (not shown), or it could be altered continuously with ramps of current (Fig. 3B). The longest period observed was approximately 15 s while the shortest period, corresponding to bursts consisting of one action potential, was about 200 ms. These cultured muscle cells do not normally display a visible contractile rhythm but are capable of contracting if stimulated with prolonged depolarizing currents or injured upon penetration with a microelectrode.

The ability to burst rhythmically seems to be a property of healthy cells rather than an artifact of damaged cells. Upon penetration with a microelectrode, an isolated cell was usually quiescent and inexcitable. It had a membrane potential near −20 mV and an input resistance of less than 40 megohms. These initial levels appear to result from injury, because with time the membrane spontaneously hyperpolarized to approximately −40 mV and the resistance increased to 70–150 megohms. Recovery could be hastened by injection of steady hyperpolarizing current. When cells were repolarized, many produced rhythmic bursts spontaneously, while many others did not. These latter cells could often be induced to burst with steady depolarizing currents of less than 0.1 nA. We will demonstrate in the following paper of this series that the myogenic properties of the heart are subject to neuromodulation (Calabrese and Maranto 1984). Despite our efforts to isolate the heart muscle cells from all neural input, it is still possible that there is tonic release of neuromodulatory substances from neuronal terminals, which remain attached to the isolated muscle cells, and that these substances are necessary for the expression of the myogenic bursts, which we have observed. Regardless of this possibility, our observations with isolated muscle cells demonstrate clearly that phasic neural drive is not necessary.
for the expression of rhythmic muscle bursts. Thus the rhythmic activity of heart muscle cells can be properly termed myogenic.

Since the muscle cells are electrically coupled in the intact heart (Maranto and Calabrese 1984), we expected that the myogenic bursting of individual cells would give rise to synchronous rhythmic activity in the intact heart. Indeed, it is possible to observe myogenic activity in the heart by recording from a muscle cell while simultaneously suppressing activity in the HE motor neuron with hyperpolarizing current. However, these observations are often complicated by the spontaneous appearance of antidromic action potential bursts in the HE cell body, which coincide with bursts in the heart. Therefore, before proceeding with a description of heart activity, we will describe in the following two sections the origin of the antidromic bursts in the HE cell.

Peripheral neurogenic rhythm

Thompson and Stent (1976a) observed that the bursts of action potentials recorded in the soma of a hyperpolarized HE cell originate near the terminals of the HE cell. This observation was made by recording simultaneously from vascular nerves near the heart and from the HE cell body. To determine whether this antidromic activity originates neurally or myogenically through coupling of the HE cell's terminals with the heart muscle, we recorded from HE cell bodies in the in situ preparation from which all muscular heart vessels were removed. Figure 4 shows that when the HE cell was hyperpolarized, antidromic bursts of action potentials appeared in the HE cell body even in the absence of heart muscle. The onset of these bursts was always delayed relative to the start of the hyperpolarization. In this case, the first burst occurred after 40 s. Once peripheral bursting had begun, it continued as long as the HE cell was hyperpolarized centrally. Typically, the interburst period was variable as in this recording, although in some preparations it was relatively uniform. Since this rhythm appears to arise in the periphery of the HE cell rather than in the heart or its side branches, we call it the peripheral neurogenic rhythm. Isolated sections of heart tube also show evidence of peripheral neurogenesis in the branches of HE cells that adhere to the heart. Thus there may be two sites at which bursts can be generated in the HE cell's periphery, one in its branches which run on the heart and one in more proximal branches. In all subsequent experiments involving peripheral neurogenesis in HE cells, the heart tube was not removed. Therefore, we were not able to distinguish the site at which the HE cell's periphery was active.

Peripheral interactions between HE cells

Further experiments aimed at studying the peripheral neurogenic rhythm resulted in the discovery of additional features of the HE cell's periphery, which are normally masked by its central activity. Specifically, we found that the nerve plexus on the heart and in the tissues surrounding the heart (Zerbst-Boroffka et al. 1982) can coordinate the peripheral neurogenic rhythm over several segments. Figure 5A illustrates the preparation from which these observations were made. Since the central connections between ganglia 4 and 7 were destroyed by removing ganglion 6, the centrally controlled bursting of the ipsilateral HE cells in ganglia 4 and 7 were uncoordinated (Fig. 5B). However, when both cells were hyperpolarized, antidromic bursts of action potentials from the peripheral neurogenic rhythm were coordinated in both cells (Fig. 5C). Thompson (1976) observed the spread of peripheral action potentials across a single segmental boundary using vascular nerve recordings. He also demonstrated, by severing the
heart, that neither the heart nor the nerve plexus around the heart was required to conduct peripheral action potentials into the vascular nerves of adjacent segments. From his observations it appears that the vascular nerve is sufficient to coordinate peripheral action potential bursts when the heart and its associated plexus is present, though severed. However, his experiment does not imply that peripheral bursts would be coordinated if the heart and its associated plexus were absent, because the vascular nerves could be serving simply as a conduction pathway for peripheral interactions that occur at the heart.

Conversely, we wanted to test whether the nerve plexus surrounding the heart is sufficient to coordinate peripheral neurogenic activity. Therefore, we severed the vascular nerves by cutting through the body wall in segment 6 in preparations such as the one in Fig. 5A. The hearts were left intact and constituted the only tissue bridges between ganglia 4 and 7. When ipsilateral HE cells in ganglia 4 and 7 were hyperpolarized, coordinated bursts of antidromic spikes indistinguishable from the bursts in Fig. 5C were recorded (not shown). As expected, this coordination was also abolished by cutting the ipsilateral heart. Thus, it appears that the nerve plexus around the heart is sufficient to coordinate peripheral action potential bursts between segments and is a site where peripheral interactions between HE cells occur. At this time it is not possible to conclude whether peripheral interactions between HE cells also occur at the level of the vascular nerve.

The preparation in Fig. 5A was also used to examine the effects of centrally generated HE cell bursts on peripheral neurogenic bursts. When cell
HE(R.4) was allowed to burst centrally while cell HE(R.7) was hyperpolarized, the bursts of antidiromic spikes in cell HE(R.7) was phase-locked to the bursts in cell HE(R.4). Additionally, as in Fig. 5B, no antidiromic spikes invaded cell HE(R.4), which was bursting centrally. Figure 5E illustrates that a current-induced burst in cell HE(R.7) was also capable of eliciting an antidiromic burst in cell HE(R.4) while cell HE(R.4) was hyperpolarized. In summary, central bursts in HE cells are able to entrain or suppress peripheral bursts in their own segment while entraining or eliciting peripheral bursts in adjacent segments.

Heart activity when the central pattern generator is suppressed

Four forms of heart activity may be distinguished when the HE motor neuron's central activity is suppressed by hyperpolarization. These forms can be explained in terms of the presence or absence of myogenic and peripheral neurogenic rhythms. Three of these forms were encountered in this study and are illustrated in Fig. 6.

Figure 6A illustrates one form of heart activity characterized by rhythmic bursting in the heart without associated activity in the ipsilateral HE cell. Although the frequency of muscle bursts was similar to the frequency of inhibitory bursts in the HE cell, the two oscillations were independent and gradually drifted apart. Because of the similarity of the heart’s bursting pattern to the bursting pattern seen in isolated muscle cells (Fig. 3A) and because of the absence of correlated activity in the HE cell, we conclude that the electrical activity of the heart in such preparations is purely myogenic.

In the second form of activity, ongoing bursts were occasionally reset by larger bursts (Fig. 6B) which were correlated with bursts of antidiromic spikes recorded in the HE cell body. Apparently, peripheral bursts in the HE cell drive the larger muscle bursts, and the smaller bursts are myogenic. There was no phasic relationship between the bursts driven by the HE cell's peripheral rhythm and either the myogenic bursts or the central pattern generator's rhythmic inhibition of the HE cell. This observation indicates that the peripheral neu-
myogenic rhythm was acting independently of these other rhythms. We conclude that both myogenic and peripheral neurogenic rhythms are active in such preparations, and that the myogenic rhythm can be reset by bursts driven by the peripheral neurogenic rhythm.

The third form of activity was characterized by the presence of rhythmic antidromic bursts of spikes in the HE cell and corresponding depolarizations in the muscle (Fig. 6C). No other potentials were present in the muscle. As before, the antidromic spikes were not phasically related to the central pattern generator's activity. In such preparations the myogenic rhythm appears to be absent. All activity in the muscle is driven by the peripheral neurogenic rhythm.

Of the seven preparations we used for this aspect of the present study two displayed the first form of activity, two the second form and three the third form. The fourth form of activity was observed rarely in the study described in the following paper (Calabrese and Maranto 1984). It was characterized by quiescence in the heart and the HE cell (not shown). In these preparations both the myogenic and peripheral neurogenic rhythms are absent. It will be shown in the following paper, however, that the myogenic rhythm can be induced in such quiescent preparations (Calabrese and Maranto 1984).

Resetting of the myogenic and peripheral neurogenic rhythms by central activity in the HE cells

Since the electrical activity of the heart matches the electrical activity of the central pattern generator (Fig. 2) the central pattern generator must interact with the myogenic and peripheral neurogenic rhythms to entrain and coordinate them. The HE motor neuron is the primary agent by
Fig. 8. Entrainment of the myogenic and peripheral neurogenic rhythms by the HE cell. The heart's polarization rhythm is normally-phase-locked with the HE cell's activity rhythm which is, in turn, determined by rhythmic inhibition from the heart's central pattern generator. When the HE cell was hyperpolarized, the heart continued to burst rhythmically but at a slightly longer period than previously. After approximately 40 s of hyperpolarization, antidromic action potentials appeared in the HE cell as the peripheral neurogenic rhythm began to be expressed (clear arrows). At the solid arrow a relatively strong neurogenic burst reset the myogenic rhythm. When the HE cell is released from hyperpolarization, it again entrained the muscle to its own period and the peripheral neurogenic rhythm was no longer evident. Solid dots indicate the expected time of myogenic bursts. Hollow dots indicate the actual time of the bursts entrained by the HE cell.

which this interaction is achieved. A necessary prerequisite for entrainment is that rhythmic activity in the governed oscillator can be reset by a single burst of activity in the governing oscillator. The ability of HE cell activity to reset the myogenic rhythm is illustrated in Fig. 7A. In this preparation the heart was allowed to burst myogenically by suppressing activity in the HE cell with steady hyperpolarizing current. When the HE cell was made to fire with a brief pulse of depolarizing current, it evoked a burst in the heart, which reset the myogenic rhythm to a new phase.

Resetting of the peripheral neurogenic rhythm by HE cell activity was demonstrated in the same preparation used in the experiment of Fig. 5A. This preparation exhibited a regular peripheral neurogenic rhythm without any spontaneous myogenic bursts, when a pair of ipsilateral HE cells in ganglia 4 and 7 were held hyperpolarized (Fig. 7B). Releasing cell HE(R,7) from hyperpolarization reset the peripheral neurogenic rhythm in the same segment [cell HE(R,7)] and in distant segments [cell HE(R,4)]. The electrical activity of the innervated heart followed the peripheral neurogenic rhythm throughout the experiment.

Entrainment of the myogenic and peripheral neurogenic rhythms by central activity in the HE cells

Figure 8 illustrates that the central activity rhythm of the HE motor neuron entrains both the myogenic rhythms of the heart and the HE cell's own peripheral neurogenic rhythm. At the beginning of the experiment the HE cell was allowed to fire normally. The polarization rhythm of the heart was phase-locked to the HE cell's activity, and no peripheral neurogenic rhythm was apparent. When the HE cell was hyperpolarized to suppress its central activity, the heart continued to burst rhythmically but at a slightly longer period than previously. After several seconds the HE cell began to express an irregular peripheral neurogenic rhythm. As soon as the HE was released from hyperpolarization the myogenic rhythm phase-locked to its activity and peripheral neurogenic bursts ceased.

Resetting and entrainment of the heart's contractile rhythm by the HE cell's central activity

Until now we have not addressed the issue of the relation of the electrical activity recorded from the heart and the heart's contractile activity. Thompson and Stent (1976a) observed that visually recorded constrictions of the heart were correlated with central activity in HE cells. Figure 9A shows that the frequency and duration of central activity in an HE cell determines the amplitude and duration of bursts recorded in heart muscle. Figure 9B shows there is a similar relation between central activity in an HE cell and the amplitude and duration of heart contractions. Because we found it necessary to immobilize the heart to record its electrical activity, we did not record electrical activity
Fig. 9A, B. Relation between electrical and contractile activity of the heart. Two separate preparations were used in A and B. In both preparations there was no myogenic activity and the peripheral neurogenic activity was weak and irregular. The HE cell's central activity was suppressed with a steady hyperpolarizing current, and bursts of impulses of different impulse frequency and burst duration were elicited in the HE cell by depolarizing current pulses of varying amplitudes and durations.

A. Burst amplitudes and durations recorded in the heart muscle followed HE impulse frequency within limits. (Short duration and or low impulse frequency bursts elicited only passive responses in the muscle [e.g., 4 and 6].

B. Tension amplitude and duration of heart muscle contraction followed HE impulse frequency and duration within limits. The saturation characteristics of tension amplitude and duration seem to parallel those of electrical activity.

Fig. 10A, B. Resetting and entrainment of heart's myogenic contractile rhythm by the HE cell. This preparation displayed a myogenic contractile rhythm but no peripheral neurogenic rhythm when HE cell's central activity was suppressed with hyperpolarizing current. A. When the HE cell was released from hyperpolarization for a brief time, it fired a burst that induced a single contraction and reset the heart's myogenic contractile rhythm. Solid dots indicate the expected times of the myogenic contractions. B. At beginning of the trace the heart's contractile rhythm was phase-locked to the HE cell's activity rhythm. When the HE cell was hyperpolarized, the myogenic contractile rhythm persisted, but its period no longer matched that of the HE cell's rhythm. When the HE cell was released from hyperpolarization, it quickly reentrained the myogenic contractile rhythm. Solid dots indicate the expected times of the myogenic contractions. Hollow dots indicate the actual time of contractions entrained by the HE cell.
and muscle tension simultaneously from the same section of the heart. Simultaneous records were made of electrical activity and muscle tension in adjacent sections of the heart tube in a few preparations, and the two were correlated. Thus, we have evidence that the two activities are related. Moreover, the HE cell's central activity resets and entrains the heart's myogenic contractile rhythm (Fig. 10). These observations strengthen the case for a relation between electrical activity and contraction and firmly establish the role of the HE motor neuron as the agent that entrains the leech's heartbeat.

Discussion

Sources of rhythmicity in the heartbeat system

There are three principal sources of rhythmicity within the heartbeat system of the leech. First, the individual heart muscle cells are capable of rhythmic activity. In the heart they form a linear array of independent oscillators, which are probably held together by electrical coupling (Maranto and Calabrese 1984). Their oscillatory capabilities are often expressed as coordinated myogenic bursting of the heart. However, this is not always the case: as will be shown in the following paper (Calabrese and Maranto 1984), their myogenic capabilities are subject to modulatory influences.

The second source of rhythmic activity is the peripheral branches of the HE cells. It is not clear whether this activity is inherent to the HE cell's periphery, driven by unidentified peripheral neurons, or the result of peripheral synaptic interactions between HE cells. Our results indicate peripheral synaptic interactions between the HE cells but do not rule out the other mechanisms proposed. The role of this peripheral activity in the system remains obscure. Peripheral bursting in the HE cells does not occur normally but is only observed when the HE cell's central activity is experimentally suppressed. Moreover, it takes several seconds to develop after the HE cell's central activity is suppressed. It is not expressed in all preparations, and is often highly irregular. All these observations indicate that the peripheral neurogenic activity is normally suppressed by central activity in the HE cell and that it is not the output of a robust oscillator. However, the peripheral synaptic interaction between the HE cells uncovered here could serve a useful function in insuring the spread of synaptic excitation from the HE cell along the heart. Nevertheless, the peripheral bursting of the HE cells is not unique, since, other cells within the heartbeat system express this property (Calabrese 1980).

The third source of rhythmic activity is the heartbeat central pattern generator (Calabrese and Peterson 1983) which, as will be argued below, is the governing oscillator in the system.

Overview

The results presented here indicate that the leech's heart is myogenic. However, this statement should not be construed to mean that the heart acts independently of neural control. Indeed, the resetting and entrainment experiments presented here show that the myogenic oscillator is normally entrained by a central oscillator (the heartbeat central pattern generator) through the agency of the HE motor neurons. Thus, the leech's heart stands midway on the spectrum between the purely neurogenic hearts of crustaceans and the mostly myogenic hearts of molluscs (Stent et al. 1979).

Normally the leech's two hearts beat in a well coordinated but different longitudinal pattern (Thompson and Stent 1976a). One constricts synchronously along its length, the other in a rear-to-front peristaltic wave. The two hearts are coordinated with one another: they beat at the same period and in a stable phase relation. With remarkable regularity the hearts switch roles (Krahl and Zerbst-Boroffka 1983). It is difficult to imagine how such an elaborate metastable pattern of constriction could be generated by two independent linear arrays of oscillatory muscle cells each held together only by electrical coupling (Maranto and Calabrese 1984) and with no side-to-side interaction. Some central mechanism seems necessary to accomplish the task. This mechanism is provided by the heartbeat central pattern generator comprising a set of segmental heart interneurons that ultimately control the firing pattern of the segmental heart motor neurons through rhythmic inhibition (Thompson and Stent 1976a, b, c; Calabrese 1977; Calabrese and Peterson 1983). The HE motor neurons fire synchronously on one side of the animal and in a rear-to-front progression on the other, and they display precise side-to-side coordination. This asymmetry in the HE cells' firing pattern is metastable: these patterns regularly switch sides. Ultimately the HE motor neurons impose the central pattern generator's rhythm upon the hearts by entraining their myogenic rhythms in each segment through rhythmic excitation. Thus, the central pattern generator establishes the precisely coordinated metastable constriction pattern observed, and its role in the system seems clear.
The role of the myogenic oscillators seems less clear because the central pattern generator could act by driving otherwise passive muscle. Perhaps, the myogenic oscillators provide a safety mechanism through redundancy, or increase the system’s efficiency through amplification of the HE cells’ output, or perhaps, as will be suggested in the following paper (Calabrese and Maranto 1984), they provide an important point at which the system may be modulated.

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