# Neuronal Generation of the Leech Swimming Movement

## An oscillatory network of neurons driving a locomotory rhythm has been identified.

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*Summary.* The swimming movement of the leech is produced by an ensemble of bilaterally symmetric, rhythmically active pairs of motor neurons present in each segmental ganglion of the ventral nerve cord. These motor neurons innervate the longitudinal muscles in dorsal or ventral sectors of the segmental body wall. Their duty cycles are phase-locked in a manner such that the dorsal and ventral body wall sectors of any given segment undergo an antiphasic contractile rhythm and that the contractile rhythms of different segments form a rostrocaudal phase progression. This activity rhythm is imposed on the motor neurons by a central swim oscillator, of which four bilaterally symmetric pairs of interneurons present in each segmental ganglion appear to constitute the major component. These interneurons are linked intra- and intersegmentally via inhibitory connections to form a segmentally iterated and intersegmentally concatenated cyclic neuronal network. The network appears to owe its oscillatory activity pattern to the mechanism of recurrent cyclic inhibition.

Generation of the locomotory rhythms of walking, flying, and swimming, whose analysis was begun in the 15th century by Leonardo da Vinci (*1*), came to be generally explained in the first half of this century in terms of oscillatory reflex arcs. According to the oscillatory reflex arc theory of locomotion, motor neurons responsible for execution of one phase of a movement cycle, for example, depression of a wing or protraction of a leg, are driven by proprioceptive feedback from an earlier phase of the movement cycle, for example, elevation of the wing or retraction of the leg (*2*). More recently, it transpired that rhythmic activity patterns of motor neurons in both vertebrate and invertebrate animals are usually generated wholly within the central nervous system, without necessary participation of proprioceptive feedback (*3*, *4*). Thus, locomotory rhythms were inferred to be the products of central nervous oscillators. However, identification of the neural elements of such oscillators and explanation of the mechanism by which they manage to give rise to locomotory rhythms has proved to be very difficult, because of the complexity of and restricted access to most central nervous systems. But, as we will show in this article, the relative simplicity and high accessibility of the nervous system of the leech (Fig. 1), a member of the annelid phylum of segmented worms, permitted the identification of the central nervous oscillator driving its swimming movement.

### The Leech Nervous System

The uncomplicated nature of the leech nervous system made it a favorite material for pioneering neuroanatomical and embryological research in the 19th century (*5*). After decades of neurobiological neglect during the first half of this century, intensive study of the leech resumed during the second half (*6*). The central nervous system of the leech includes two large ganglia, one in the head and the other in the tail. The cerebral and caudal ganglia are linked by a ventral nerve cord consisting of a chain of 21 segmental ganglia and their connectives (Fig. 1a). Each segmental ganglion contains the cell bodies of some 175 pairs of bilaterally symmetrical neurons and innervates, via two bilateral pairs of segmental nerves, one of the 21 abdominal body segments lying between the head sucker and the tail sucker. The gross anatomy of the iterated segmental ganglia is sufficiently stereotyped from segment to segment, and sufficiently invariant from leech to leech, that a large portion of the cell bodies of the central nervous system can be reproducibly identified. It is possible to penetrate these cell bodies with microelectrodes and record action potentials and excitatory and inhibitory synaptic potentials arising from synaptic connections with other neurons. These features make the leech nerve cord an unusually favorable material for the functional analysis of neuronal networks (*7*, *8*).

### The Leech Swimming Rhythm

The leech swims by undulating its extended and flattened body in the dorsoventral plane, forming a body wave that travels rearward, from head to tail (Figs. 1b and 2a). The moving crests of the body wave are produced by progressively phase-delayed contractile rhythms of the ventral body wall of successive segments and the moving troughs by similar, but antiphasic, contractile rhythms of the dorsal body wall. As was noted by Leonardo da Vinci (*1*, p. 595), the forces exerted against the water by these changes in body form provide the propulsion that drives the leech forward through its fluid medium. The period of the segmental contractile rhythm ranges from about 400 milliseconds for fast to about 2000 msec for slow swimming (*9*–*11*). The time taken for the rearward travel of the body wave is about equal to the period of the contractile rhythm, leading to the hydrodynamically favorable result that at all swimming speeds the animal maintains one full wave length between head and tail (*12*).

In one of the first modern physiological studies of leech swimming, von Uexküll (*13*) described the basic activity pattern of the musculature responsible for these changes in body form. First, the flattening of the body, which optimizes the force that moving dorsal and

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ventral body walls exert against the water, is produced by the tonic contraction of dorsoventral muscles. These muscles traverse the body cavity between their points of insertion into the dorsal and ventral body walls. Second, the periodic changes in length of the dorsal and ventral body wall segments that form the troughs and the crests of the wave are produced by the phasic local contraction of longitudinal muscles embedded in the body wall. By exerting a force on the body fluids, the tonic contraction of the dorsoventral muscles generates the antagonistic force necessary for periodic longitudinal distension of the segmental body wall, following relaxation of the segmental longitudinal muscles. Both dorsoventral and longitudinal muscles are innervated by motor neurons in the corresponding segmental ganglion, and the impulse activity of these motor neurons causes the local contraction and distension of the segmental musculature (*14*). These motor neurons are located on the dorsal aspect of the segmental ganglion and are designated according to the numerical system indicated in Fig. 1c.

By the late 1930's the following facts had been established regarding the role of leech nervous system in the generation of the swimming movement. Participation of the head and tail ganglia is not required for production of the body wave, since after decapitation (*13*) or surgical disconnection of these ganglia from the nerve cord (*9*) leeches not only still swim but do so more readily, and for longer episodes, than intact animals. Hence the body wave is generated within the abdominal segments. Moreover, the intersegmental coordination of the contractile rhythm is mediated by the nerve cord connective, since after cutting the connective between any two midbody ganglia, the swimming movements of the body parts anterior and posterior to the cut are no longer phase-locked (*9, 10*). However, the movements of anterior and posterior body remnants do remain coordinated if the connective is left intact while the entire body wall of several midbody segments is removed (*10*). Thus the neuronal activity responsible for

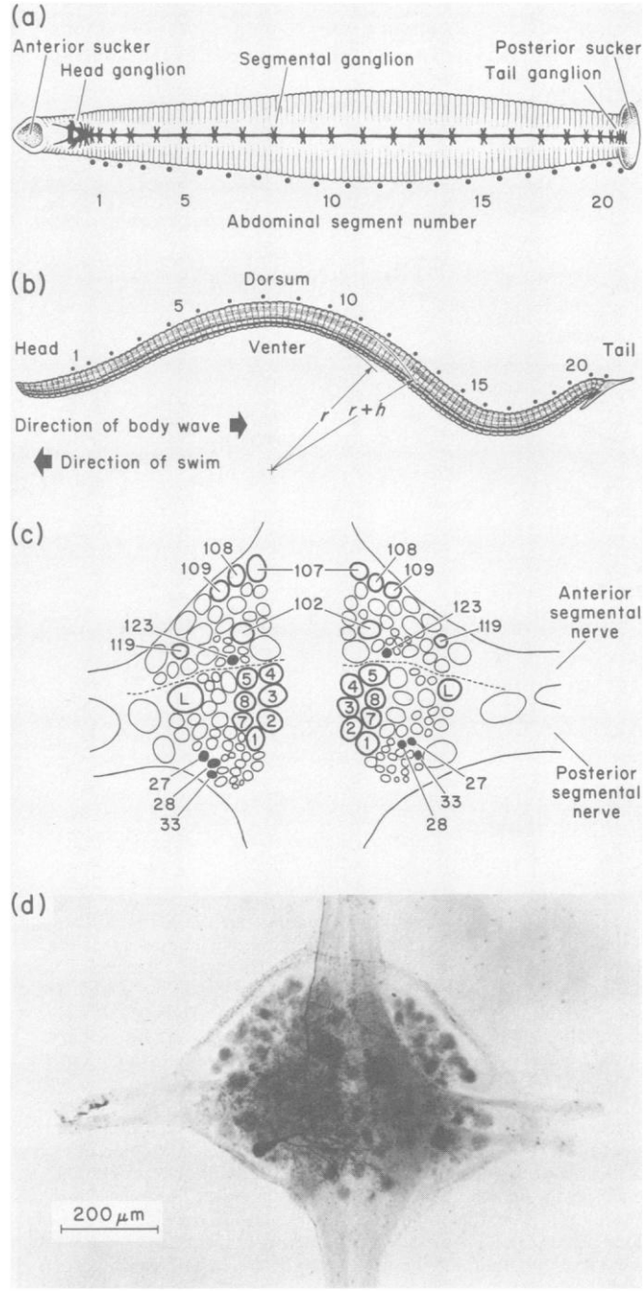


Fig. 1. (a) Schematic view of the segmental body plan of the leech and of its nerve cord, from the ventral aspect. The skin of most abdominal segments is divided into five annuli. The central annulus of each segment contains seven bilateral pairs of sensory organs, or sensillae. [After Nicholls and Van Essen (*6*)] (b) Side view of leech during the swimming movement. The body wave forms a crest in the 8th and a trough in the 16th abdominal segment. If $r$ and $r + h$ are the radii of curvature of ventral and dorsal body walls at a wave crest, then the length ratio of contracted ventral to distended dorsal longitudinal muscles in the 8th segment is $1/(1 + h/r)$, or equal to 0.8 for the body wave shown here. (c) Dorsal aspect of a segmental ganglion of the nerve cord of the medicinal leech, *Hirudo medicinalis*, showing the cell bodies of identified motor neurons (heavy outline) and of interneurons (solid black) related to the generation of the swimming rhythm. The cells are numbered according to the system of Ort *et al.* (*15*). (d) Anatomy of an oscillator interneuron. Photography of the dorsal aspect of a ganglion in which the left cell 33 was stained by intracellular injection of horseradish peroxidase. The axon of the monopolar cell is seen to enter the anterior connective.

coordination of the swimming rhythm travels through ganglia that can neither command contraction of peripheral muscles nor receive sensory input from their own segmental body wall.

### The Semi-Intact Preparation

This last finding provided the experimental point of departure for the work reported herein. It suggested the possibility of developing the semi-intact preparation of the medicinal leech, *Hirudo medicinalis*, shown in Fig. 2b. The front and rear body remnants of this preparation (in which, as in all other preparations to be mentioned herein, the head and tail ganglia have been disconnected surgically from the nerve cord) carry out coordinated swimming movements, while electrophysiological records are taken from exposed and immobilized parts of the peripheral and central nervous system. Figure 2c shows a sample record taken from such a semi-intact preparation, in which the overall swimming movement was monitored by a photocell registering the interruptions of a light beam caused by up and down movements of the front end; the contraction-distension rhythm of the ventral and dorsal sectors of a body wall flap were measured by means of isometric tension transducers; and the efferent impulse activity of the exposed segmental ganglion was monitored by means of a suction electrode attached to the segmental nerve. As can be seen, the dorsal and ventral longitudinal muscles of the exposed body wall flap undergo an antiphasic contraction-relaxation rhythm, whose period of about 1000 msec matches that of the up and down movements of the front end. Moreover, in the dorsal branch of the segmental nerve there occur bursts of impulses of uniform amplitude whose cycle is phase-locked with that of the body wall contractions and the front end movements.

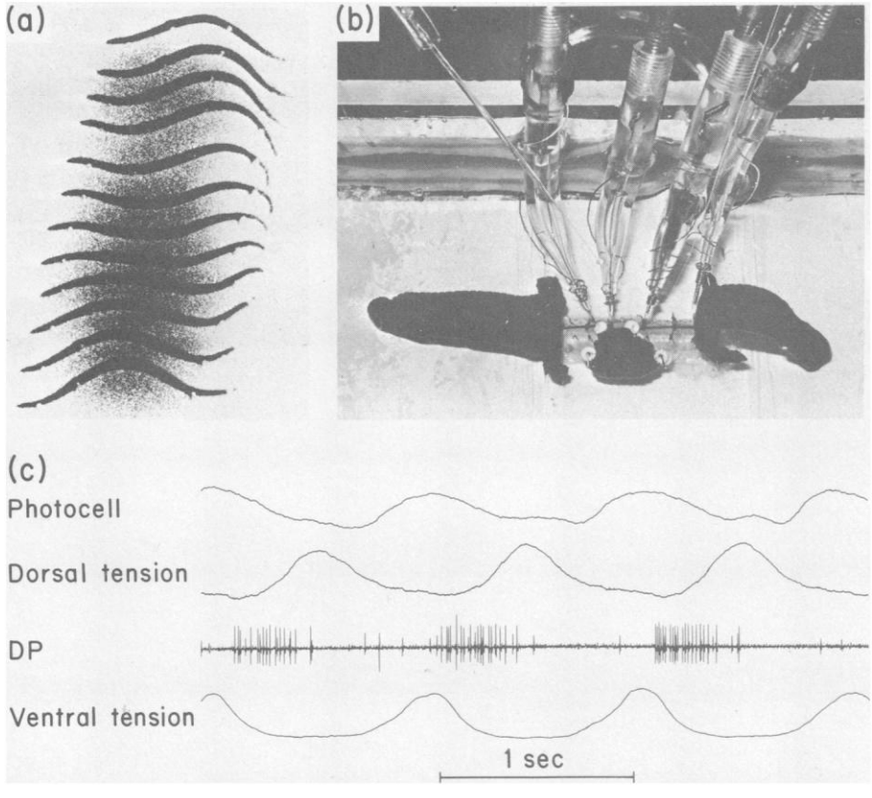


Fig. 2. (a) The body wave of a swimming leech, as seen in this composite print of successive frames of a cinematographic record of a free-swimming specimen, with white reference beads attached to the 1st, 5th, 10th, and 15th abdominal segments. The right-to-left horizontal displacement of the animal depicts its true progress in the water. The time occupied by this episode, which corresponds to one cycle period, is about 400 msec. (b) The semi-intact leech preparation pinned venter upward. Front (right) and rear (left) parts are connected by the exposed ventral nerve cord and its segmental ganglia, of which four are visible. The isolated body wall flap, with sewn-on beads for the attachment of threads leading to tension transducers, is still connected via the left segmental nerves to one of the exposed ganglia. Four glass-tipped suction electrodes for extracellular nerve trunk recordings and two glass capillary microelectrodes for intracellular recordings are shown. (c) Contractile rhythm and segmental nerve activity during a swimming episode of a semi-intact preparation. The trace labeled *Photocell* monitors the up and down movements of the head. The traces labeled *Dorsal tension* and *Ventral tension* represent the output of isometric tension transducers recording the contraction (upward deflection) of the dorsal and ventral sectors of the attached body wall flap. The trace labeled *DP* presents the output of a suction electrode attached to the dorsal branch of the posterior segmental nerve.

In Fig. 3a is another sample record from a semi-intact preparation, in which an intracellular microelectrode was inserted into cell 3 (see Fig. 1c) of the exposed ganglion and a suction electrode was attached to the segmental nerve. While the preparation is at rest, intracellular and segmental nerve recordings show tonic impulse activity, with each intracellularly recorded action potential being followed with constant delay by a large-amplitude impulse in the segmental nerve. Thus these impulses in the segmental nerve are action potentials traveling outward in the axon of cell 3. While the preparation is swimming, the segmental nerve record shows impulse bursts similar to those in Fig. 2c, with the intracellular record showing oscillations in the membrane potential of cell 3 that are phase-locked with the impulse burst rhythm. During the depolarized phase of these potential oscillations of cell 3 there arise action potential bursts that account for the impulse burst rhythm of the segmental nerve branch; during the hyperpolarized phase, cell 3 sustains a burst of inhibitory synaptic potentials. By means of additional intracellular recordings taken from cell 3 it could be shown, moreover, that every impulse traveling outward in the axon of cell 3 is followed by an excitatory synaptic potential in the longitudinal muscle fibers in the dorsal territory of the body wall flap. Hence cell 3 is an excitatory motor neuron of the dorsal longitudinal muscles, a conclusion which is in full accord with the finding manifest in Fig. 2c that in the cycle of the swimming rhythm the cell 3 impulse burst just precedes the contractile phase of the dorsal body wall.

### The Swim Motor Neuron Ensemble

An extensive survey of the cell bodies on the dorsal aspect of segmental ganglia (see Fig. 1c) of semi-intact preparations led to the identification of an ensemble of bilaterally symmetric pairs of motor neurons that take part in the generation of the swimming movement (*15*). This ensemble comprises seven excitatory motor neuron pairs to the segmental longitudinal muscles, of which four, the dorsal excitors (cells 3, 5, 7, and 107), cause contraction of the dorsal and three, the ventral excitors (cells 4, 8, and 108), contraction of the ventral body wall. The en-

semble also includes four inhibitory motor neuron pairs to the segmental longitudinal muscles, of which two dorsal inhibitors (cells 1 and 102) cause distension of the dorsal and two ventral inhibitors (cells 2 and 119) distension of the ventral body wall (*16*). In addition, the ensemble of swim motor neurons includes an excitatory motor neuron pair causing contraction of the right and left dorsoventral muscles, the dorsoventral excitor, cell 109.

During swimming, the dorsoventral excitor, cell 109, is maintained in a depolarized state and produces impulses at a high rate, thus accounting for the flattened body shape and its associated longitudinal stretching force. Meanwhile the membrane potential of the dorsal and ventral excitors and inhibitors oscillates, just as does that of cell 3 in Fig. 3a, between a depolarized and a hyperpolarized phase, with an impulse burst arising during the depolarized phase. Figure 3b summarizes in schematic form the phase relations of the motor neuron activity pattern observed during the swim cycle. With the phase angle 0° assigned arbitrarily to the impulse burst midpoint of the dorsal excitor cell 3, the impulse burst of the ventral inhibitor cell 119 occurs also at about 0° and the impulse bursts of the ventral excitor cell 4 and of the dorsal inhibitor cell 102 occur at phase angles of about 180°. (The other three dorsal excitors are active in the same phase as cell 3, whereas the other two ventral excitors are active in nearly, but not exactly, the same phase as cell 4.) Thus, as would be expected from the antiphasic segmental contractile rhythm evident in Fig. 2c, the dorsal and ventral excitors are active in antiphase, as are the dorsal and ventral inhibitors. However, the impulse bursts of the other two inhibitors do not quite meet that expectation, with the impulse bursts of the dorsal inhibitor cell 1 and of the ventral inhibitor cell 2 occurring at the intermediate (albeit mutually antiphasic) phase angles of about 90° and 270°, respectively (*17*). The dorsoventral excitor cell 109 produces a continuous impulse train, although during swimming its membrane is also subject to a low-amplitude polarization cycle that reaches its peak depolarization at a phase angle of about 0° (*15*).

Comparison of the activity patterns of swim motor neurons in a rostrocaudal sequence of segmental ganglia along the nerve cord shows that the impulse burst cycles of serially homologous motor neurons occur with a progressive intersegmental phase delay, in accord with the rearward travel of the body wave (*18*). Thus the phasic activity pattern of the ensemble of identified motor neurons in the nerve cord accounts for the basic contractile rhythm of the swimming leech. Accordingly, the problem of the neuronal control of the swimming rhythm may be stated in the form of two basic questions: (i) How does the motor neuron ensemble of a given ganglion produce the phasic activity pattern in Fig. 3b, with cycle periods in the range of 400 to 2000 msec? (ii) How are the motor neuron duty cycles of that ganglion coordinated with the duty cycles of serially homologous neurons in other ganglia of the cord, so that the segmental rhythms can run in an appropriate rostrocaudal phase progression?

To ascertain whether the answers to these two questions could be found in terms of a network linking the identified motor neurons, we made a survey of the connections that exist between them. For this purpose, pairs of motor neurons were penetrated simultaneously with microelectrodes and a cell pair was inferred to be connected if passage of current into one cell had an effect on the membrane potential of the other. In this way we found that all the dorsal excitors are interlinked by electrical junctions, as are all the ventral excitors, all the dorsal inhibitors, and all the ventral inhibitors. Furthermore, homologous excitors and inhibitors on the right and left sides of the ganglion are similarly linked. These intraganglionic electrical junctions probably serve to equilibrate the membrane potentials, and thus synchronize the duty cycle phases, of motor neurons that

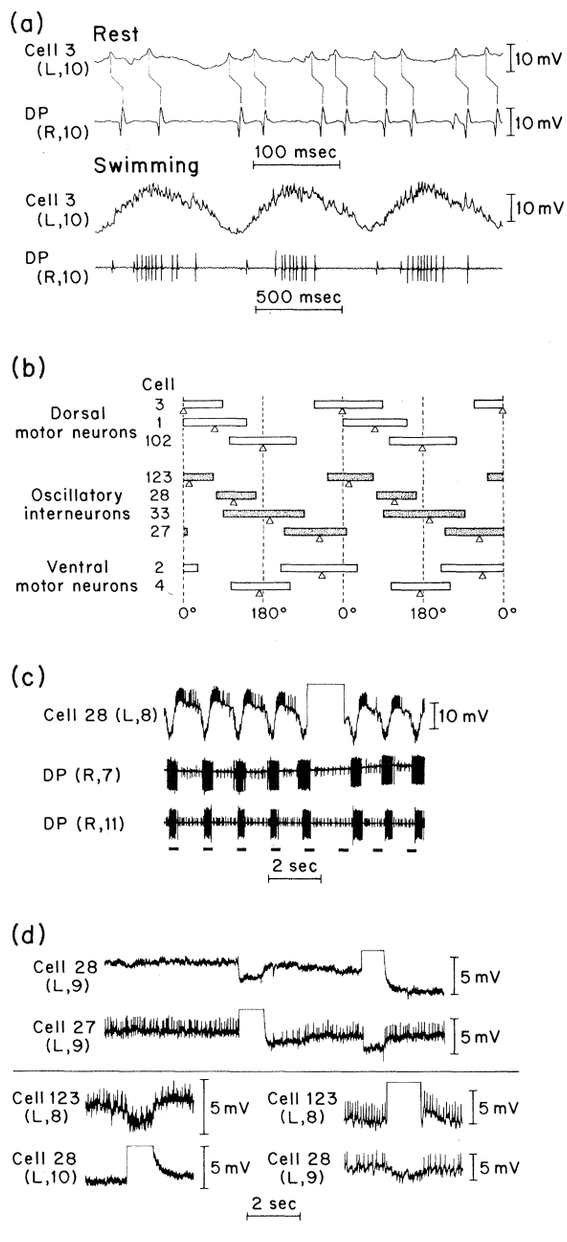


Fig. 3. (a) Extra- and intracellular recordings from a semi-intact preparation. Upper traces: preparation at rest. Lower traces: preparation swimming. *Cell 3 (L,10)*: output of microelectrode inserted into left cell 3 of ganglion 10; *DP (R,10)*: output of suction electrode attached to dorsal branch of right posterior nerve of 10th segment. (b) Summary diagram of the swimming activity cycles of the oscillatory interneurons and of a representative subset of the motor neurons. Each bar indicates the duration of the impulse burst of the cell, and the triangle under each bar points to the burst midpoint. The burst midpoint of cell 3 has been arbitrarily assigned the phase angle 0°. (c) Intracellular microelectrode recording from the left interneuron cell 28 of the 8th abdominal ganglion and suction electrode recordings from the dorsal branch of the right posterior nerve of the 7th [*DP (R,7)*] and 11th [*DP (R,11)*] abdominal ganglia during a swim episode of an isolated ventral nerve cord preparation. In this and the following panel, the sharp upward deflection of the intracellular record marks passage into the interneuron of a pulse of depolarizing current. The bars drawn under the *DP (R,11)* trace indicate the times of occurrence of impulse bursts from cell 3 to be expected if passage of current into the interneuron had *not* shifted the phase of the swimming rhythm. (d) Pairwise intracellular microelectrode recordings from oscillator interneurons within the same ganglion (upper pair of traces) or different ganglia (lower pair of traces).

are to act in concert (*15*). More important, it was found that inhibitory connections link the dorsal inhibitors with the dorsal excitors and the ventral inhibitors with the ventral excitors (homonymous excitors), so that in addition to their direct inhibitory effect on the longitudinal muscles, the inhibitors also cause central inhibition of their homonymous excitors (*15*). Thus the alternating inhibition and disinhibition provided to each excitor by its homonymous inhibitors constitutes one source of the excitor activity rhythm. But the source of the activity rhythm of the inhibitors themselves was not accounted for by these findings.

### Suppression of the Shortening Reflex

Although it did not reveal the source of the motor neuron activity pattern, the survey of motor neuron connections did provide an explanation for the suppression of the "shortening reflex" during swimming. This reflex consists of a bodywide contraction of the longitudinal musculature, and of a transformation of the body shape into a squat ellipsoid, in response to noxious stimuli. The reflex is mediated by a monosynaptic excitatory pathway from cutaneous sensory receptors to the "shortener" motor neuron, cell L, an excitor of both dorsal and ventral longitudinal muscles (*7, 14*). As is to be expected, cell L does not take part in the generation of the swimming rhythm, since simultaneous contraction of all longitudinal muscles would avert up and down bending of the segmental body wall. In agreement with this expectation, cell L does, in fact, become hyperpolarized and its impulse activity is greatly reduced during swimming (*15*). This hyperpolarization of cell L can be accounted for by the connections which link cell L to the swim motor neurons. First, cell L is linked to the dorsal and ventral excitors by rectifying electrical junctions that allow passage of depolarizing current only from cell L to the excitors and passage of hyperpolarizing current only in the reverse direction. Thus cell L receives hyperpolarizing current throughout the swim cycle, from the dorsal excitors during their hyperpolarized phase centering on the phase angle of 180° and from the ventral excitors during their hyperpolarized phase centering on 0°. Second, inhibitory connections lead from both dorsal and ventral inhibitors to cell L. Thus cell L receives inhibitory input throughout the swim cycle, at the phase angles of 90° and 180° from the dorsal inhibitors, and at 270° and 0° from the ventral inhibitors (*15*). Consequently, during swimming cell L cannot subserve the shortening reflex. However, as long as the animal is not swimming, depolarization of cell L attending the shortening reflex can spread via the electrical junctions to the dorsal and ventral excitors and thus recruit them for reflexive contraction of the entire longitudinal musculature.

### The Oscillatory Interneurons

The further quest for the answer to the two questions regarding the neuronal control of the swimming rhythm was greatly advanced by the discovery that the motor neuron ensemble of an isolated nerve cord, severed from all contact with the leech body, can produce sustained episodes of the swimming activity pattern in response to brief electrical shocks delivered to a segmental nerve (*19*). It could be concluded, therefore, that leech swimming provides another instance of a locomotory rhythm produced by a central nervous oscillator capable of giving rise to a motor neuron activity rhythm without necessary participation of proprioceptive feedback. Accordingly, a search was carried out in segmental ganglia of the isolated nerve cord of *H. medicinalis* for neurons other than the swim motor neurons that might constitute the central swim oscillator. For the purpose of this search, a neuron was considered to be a candidate component of the oscillator if, during a swimming episode, (i) its cell membrane underwent a polarization rhythm that was phase-locked with the impulse burst rhythm of the motor neurons, and (ii) passage of current into the neuron shifted the phase of the motor neuron impulse burst rhythm. All previously identified motor neurons that met the first of these two criteria failed the second, and hence did not qualify as candidate components of the central oscillator (*15, 20–22*). After an extensive survey of the segmental ganglia, which included most of the cell bodies on both dorsal and ventral aspects, four bilateral pairs of neurons in each segmental ganglion were found to meet both criteria (*23, 24*). They are the right and left homologs of cells 123, 28, 33, and 27, all having very small cell bodies located on the dorsal aspect (Fig. 1c). Figure 3c shows an intracellular recording taken from the left cell 28 of ganglion 8 during a swimming episode of an isolated nerve cord preparation consisting of a chain of 18 ganglia. The figure presents also the concurrent output of suction electrodes attached to the segmental nerves of ganglia 7 and 11. The initial part of the segmental nerve record shows five cycles of impulse bursts of the dorsal excitor, cell 3, characteristic of the swimming rhythm (see Figs. 2c and 3a). Meanwhile, the intracellularly recorded membrane potential of cell 28 oscillates in a rhythm that is phase-locked with the motor neuron impulse bursts. During its depolarized phase cell 28 produces an impulse burst whose midpoint occurs at a phase angle of about 90° in the swim cycle of its own segment. Transient passage of depolarizing current into cell 28 of ganglion 8 can be seen to arrest the impulse burst rhythms of the cell 3 serial homologs in ganglia 7 and 11. After termination of current passage, the cell 3 impulse bursts resume, but their phase has been shifted relative to the swimming rhythm prior to current passage. Data similar to those in Fig. 3c were obtained also for cells 123, 33, and 27. The intracellularly recorded membrane potentials of all these cells also oscillate in a rhythm phase-locked with that of the swimming rhythm, with the impulse burst midpoint occurring at a phase angle of about 0° for cell 123, 180° for cell 33, and 270° for cell 27 (see Fig. 1c).

Anatomical as well as electrophysiological evidence suggests that the oscillator cells are intersegmental interneurons. First, upon specific staining of cell 28 and cell 33 by intracellular injection of horseradish peroxidase (*25*) both cells can be seen to send an axon into the anterior connective, and neither cell can be seen to send an axon into the segmental nerve roots of its ganglion (Fig. 1d). Second, impulses arising in cell 27, cell 28, or cell 33 can be recorded in the ventral cord connective over a distance of at least five segments to the front, and impulses arising in cell 123 can be similarly recorded over a distance of at least two segments to the rear. No trace of the impulses of any of these four cells can be found in the segmental nerves. Thus cells 27, 28, and 33 project to more anterior ganglia and cell 123 projects to more posterior ganglia of the cord.

### The Oscillator Network

To establish the nature of the network formed by the oscillatory interneurons, we obtained pairwise intracellular recordings from them to ascertain the manner in which they are connected, both intra- as well as interganglionically. Figure 3d shows that passage of depolarizing current into either member of an ipsilateral cell 27–cell 28 pair of the same ganglion hyperpolarizes the other member,

leading to the inference that cell 27 and cell 28 are reciprocally connected intraganglionically via inhibitory links. Moreover, passage of depolarizing current into cell 28 hyperpolarizes cell 123 of a more anterior ganglion and passage of depolarizing current into cell 123 hyperpolarizes cell 28 of a more posterior ganglion. Cells 123 and 28 of different ganglia are thus reciprocally connected interganglionically via inhibitory synaptic links. A diagrammatic summary of the connections that were identified in this way is presented in Fig. 4a. In this diagram the four interneurons of two ganglia—one more anterior and the other more posterior—have been placed at the corner of a square, so that their activity phases progress clockwise. As can be seen, *intra*ganglionically, cells 33, 28, and 123 each make inhibitory connections with that cell that leads it by a phase of 90° in the swim cycle. Furthermore, the antiphasically active cells 27 and 28 are linked via a pair of reciprocally inhibitory connections, and a rectifying electrical junction links cells 33 and 28. *Inter*ganglionically, the three interneurons (cells 28, 33, and 27) whose axons project into the anterior connective make inhibitory connections in more anterior ganglia with the serial homologs of a cell with which they also connect in their own ganglion. The one interneuron (cell 123) whose axon projects into the posterior connective makes inhibitory connections in more posterior ganglia with the serial homologs of a cell (cell 28) which follows it by a phase angle of 90° and with which it does not connect in its own ganglion. Although this is not shown in Fig. 4a, two of the interneurons (cells 28 and 33) have been found to be linked intraganglionically via an electrical junction to their contralateral homologs. Thus the bilateral pair of ganglionic oscillator networks is coupled.

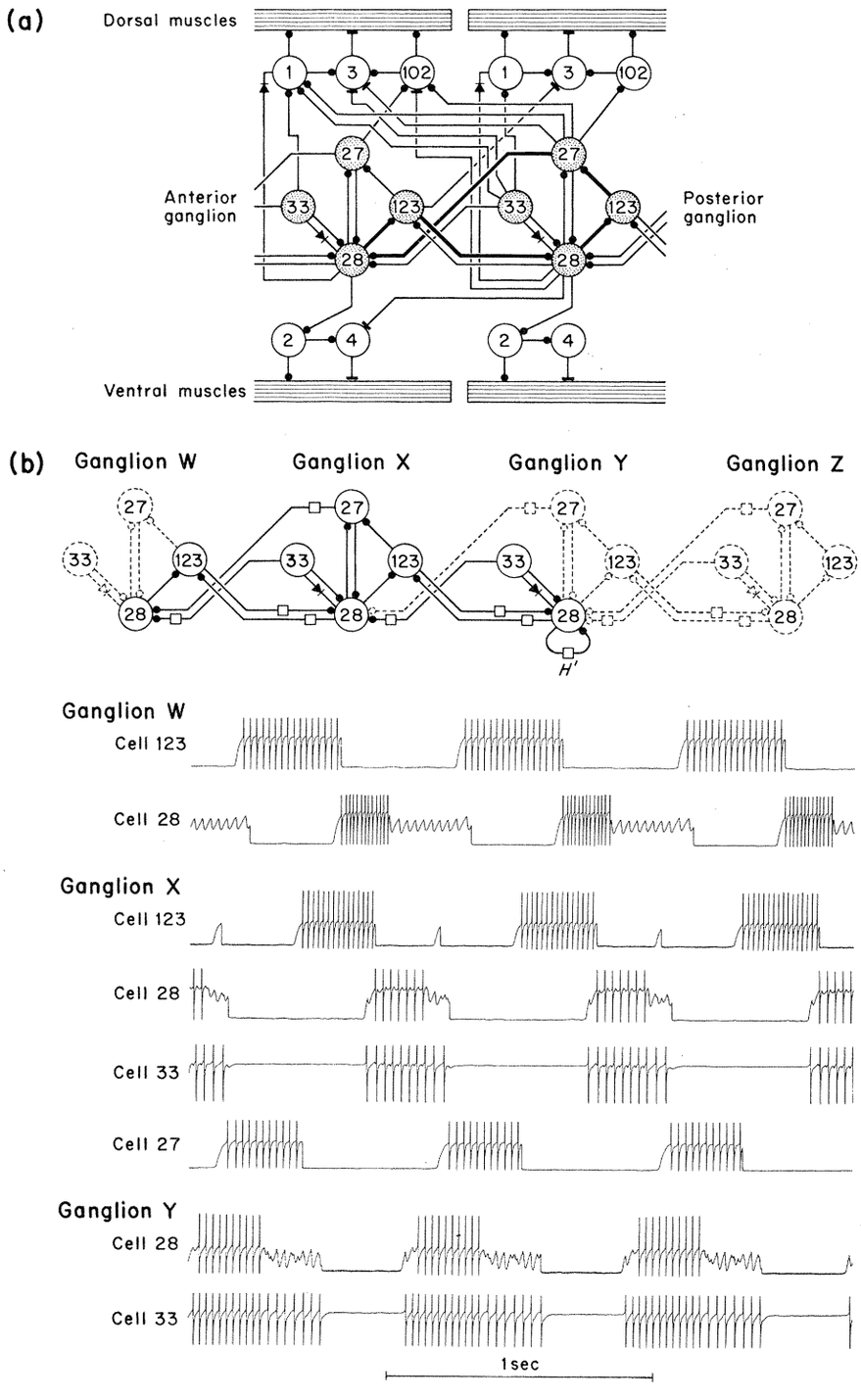


Fig. 4. (a) Summary circuit diagram of identified synaptic connections between interneurons (shown as shaded circles), motor neurons (shown as plain circles), and longitudinal muscles responsible for the swimming rhythm. Meaning of symbols: *T joint* = excitatory synapse; filled circle = inhibitory synapse; diode = rectifying electrical junction. The connections forming the basic five-membered, recurrent cyclic inhibition ring are shown as heavy line. (b) Oscillation of a partial electronic analog model of the network of oscillatory interneurons. Impulse bursts generated by eight electronic neuromimes connected according to the circuit shown at the top. The circuit diagram schematizes the oscillatory interneurons of four ganglia W, X, Y, Z, representing the 1st, 5th, 9th, and last of an isolated chain of 13 ganglia. Cells represented by neuromimes and their modeled connections are shown in solid lines; cell and connections omitted from the model circuit are shown in dashed outline. The boxes designate connections with impulse conduction delays of 80 msec. The self-inhibitory "phantom" connection of cell 28 of ganglion Y incorporating a transmission delay $H' = 250$ msec replaces the presence of cell 123 of ganglion Y and of cells 33 and 28 of ganglion Z. The impulse transmission delays were modeled by means of shift registers. Sufficient tonic excitation was provided to each interneuron analog to produce an impulse frequency of about 80 hertz at the height of its active phase. The details of this model circuit and the justification for use of the "phantom" connection in place of the two posterior cells are described in (*28*).

## Output Connections of the Oscillator Network

A third criterion that must be satisfied if the neurons of the identified network are to qualify as components of the central swim oscillator is that they make appropriate output connections to the previously identified segmental swim motor neurons. To ascertain the existence of such output connections, we took pairwise intracellular recordings from interneurons and motor neurons (*22*). The results of this survey are summarized in Fig. 4a (*26*). As can be seen, the oscillatory interneurons are linked to the motor neurons via both inhibitory and excita-

tory connections, and, in the case of the cell 28–cell 1 pair, also via a rectifying electrical junction. Some of these connections are intraganglionic. Other connections are interganglionic, in the sense that the interneurons whose axons project frontward contact serially homologous motor neurons in several anterior ganglia and that the rearward projecting interneuron (cell 123) contacts serial homologs of the dorsal excitor cell 3 in several posterior ganglia. Theoretical analysis of the network showed that, given the oscillatory interneuron duty cycles, these output connections, in combination with the previously identified inhibitory connections of the inhibitory motor neurons to their homonymous excitatory motor neurons, do give a reasonably close account of the observed motor neuron duty cycles summarized in Fig. 3b (*22*). This analytical conclusion was confirmed by the output of an electronic analog circuit of the motor neurons and their excitatory and inhibitory input connections, driven by an electronically generated simulation of the oscillatory interneuron impulse burst rhythm (*22, 27*). Thus the oscillatory interneurons qualify also by the third criterion for status as central swim oscillator elements.

### Mechanism of the Oscillation

But how do the identified elements of the central oscillator manage to produce the intra- and intersegmentally coordinated swimming rhythm? Analysis of the functional properties of the interneuronal network of Fig. 4a has shown (*28*) that it incorporates the feature of recurrent cyclic inhibition, to whose possible role in the neuronal generation of locomotory rhythms Székely first drew attention (*29*). Recurrent cyclic inhibition gives rise to oscillations whenever an odd number of tonically excited neurons forms a ring network in which each neuron makes inhibitory synaptic contact with and receives inhibitory synaptic input from one other neuron. Accordingly, the basic oscillatory circuit of the central swim oscillator consists of a five-membered ring formed by cells 28 and 123 of an anterior ganglion and cells 28, 123, and 27 of a posterior ganglion. Moreover, since the intersegmental inhibitory connections formed by cells 27 and 123 are repeated in several anterior and posterior ganglia, respectively, the basic circuit can be considered as a series of intersegmentally concatenated, five-membered rings.

To fathom the oscillatory dynamics of one ring (indicated by the heavy lines in Fig. 4a), let us suppose that the anterior and posterior cells 28 happen to be in their depolarized and impulse-generating state. Then the anterior and posterior cells 123, being subject to inhibition from the two cells 28, are in a hyperpolarized, inactive state, while the posterior cell 27 is inactive but recovering from recently terminated inhibition (phase 1). As soon as cell 27 has recovered and reaches its impulse-generating threshold, the anterior cell 28 becomes inhibited, thus disinhibiting and allowing recovery of the anterior cell 123 (phase 2). Once the anterior cell 123 has recovered, it inhibits the posterior cell 28, allowing the posterior cell 123 to recover (phase 3). Upon recovery of the latter, the posterior cell 27 becomes inhibited, allowing recovery of the anterior cell 28 (phase 4). Upon recovery of the anterior cell 28, the anterior cell 123 becomes inhibited, allowing recovery of the posterior cell 28 (phase 5). And upon recovery of the posterior cell 28 and the resulting inhibition of the posterior cell 123, the oscillator has completed one cycle. According to these dynamics, the duty cycles of cell 28 and cell 123 in the same ganglion are nearly antiphasic, whereas the duty cycles of the anterior cells 28 and 123 slightly lead in phase those of their posterior homologs. Thus as long as the cells are provided with a source of tonic excitation, the basic circuit is capable of producing a crude version of the swimming rhythm, with an essentially two-phase, rather than four-phase, segmental duty cycle. The actually identified, topologically more complex network of Fig. 4a can then be viewed as an elaboration of the basic five-membered intersegmental ring, in the sense that the addition of cell 33, of reciprocal inhibitory connections between cells 27 and 28, and of frontward intersegmental inhibitory connections of cells 28 and 33 create a set of subsidiary rings that generate the actually observed four-phase segmental duty cycle. The cycle period of this network can be shown to depend on two parameters: (i) the intersegmental travel time taken by impulses conducted from ganglion to ganglion in the axons of the oscillatory interneurons, and (ii) the recovery time taken by each interneuron to reach action potential threshold upon its release from inhibition (*28*).

In order to test the theoretically predicted oscillatory properties of this complex cyclic network, an electronic analog model of the interneurons and their intra- and interganglionic connections was constructed, according to the schema of the insert of Fig. 4b. This model consisted of eight interconnected electronic "neuromime" elements (*30*). Each such element mimics an excitable nerve cell membrane, in that it gives rise to an electrical impulse once membrane polarization has fallen below threshold level. The cell membrane analogs also provide for the simulation of both excitatory and inhibitory synaptic currents, whose summed effects determine whether the membrane is polarized above or below threshold level. Figure 4b shows the four oscillatory interneurons, cells 123, 28, 33, and 27 of one ganglion (ganglion X) embedded in a chain of 13 ganglia, of which ganglia W and Z are the fore- and rearmost, and ganglia X and Y are the fifth and ninth in the chain, respectively. The model includes also cells 123 and 28 of ganglion W and cells 28 and 33 of ganglion Y. The output of this analog model is presented in Fig. 4b. As can be seen, the model oscillator runs with a realistic swim cycle period of about 840 msec while incorporating the actually observed interganglionic impulse conduction time of about 20 msec per segment and the realistic recovery time of about 50 msec. Moreover, the model yields for the four interneurons of ganglion X a good approximation of the observed interneuronal impulse burst relations shown in Fig. 3b, and gives rise to an appropriate rostrocaudal phase progression of the cycle phases of the serial homologs of cells 123 and 28 in ganglia W, X, and Y (*31*). This high degree of verisimilitude of the model output makes it appear that the network of Fig. 4a constitutes the major component of the central swim oscillator.

### Proprioceptive Feedback

The preponderance of central nervous oscillators as generators of basic locomotory rhythms must not be taken to mean that sensory feedback plays no role at all in the realization of such rhythms. On the contrary, in most cases the centrally generated basic rhythm is subject to influence by proprioceptors whose afferent signals serve to modulate period phase and amplitude of the movement (*2, 12, 32, 33*). This is the case also for the leech swimming movement (*10, 34*), as shown by the following experiment (*21*). A leech (whose head and tail ganglia have been severed from the nerve cord) is suspended, dorsum up, in a water-filled dish, by pinning the 6th and 7th (denervated) abdominal segments to one hard-rubber pillar and the 15th and 16th (denervated) segments to another such pillar. The preparation carries out

swimming movements, which are recorded by cinematography of the lateral body silhouette. The cinematographic records are evaluated by measuring, frame by frame, the elevation of the head, midbody, and tail segments, above or below the plane of support provided by the rubber pillars.

The results of two sets of such measurements—the two support pillars were separated by 39 millimeters in one experiment and by 31 mm in the other—are presented in Fig. 5a. As long as the pillars are separated by 39 mm, the head, tail, and midbody sections undergo a regular rhythm of phase-locked up and down movements, whose cycle period gradually increases from about 600 to 1000 msec. However, when the separation of the pillars is reduced to 31 mm, a distance so short that the midbody segments encounter mechanical resistance during their transition from wave crest to wave trough, the undulatory body movements are no longer regular. In some abnormal cycles, the midbody segments do not manage to complete formation of the trough and precociously initiate formation of the next crest, whereas in other cycles formation of the trough is completed only after an initial delay of the midbody downward movement. These mechanically induced dynamic irregularities of the midbody segments are accompanied by phase-locked irregularities of the movement of the head and tail segments, whose undulations are not subject to any mechanical restrictions. Thus the leech possesses a proprioceptive feedback system that monitors the realization of the rhythmic changes in body shape of the swimming movement commanded by the central oscillator. In case the movement is not properly executed somewhere along the length of the animal, the perturbation is signaled to both frontward and rearward segments to effect a body-wide adjustment of the swimming rhythm.

Since the radius of curvature of a given leech body segment, or the degree to which it is bent upward or downward, is given by the length ratio of the dorsal and ventral segmental body walls (Fig. 1b), it appeared likely that the proprioceptive feedback operating in the experiment of Fig. 5a has its basis in the sensory detection of longitudinal body wall stretch. In order to study the effect of body wall stretch on the segmental swimming rhythm, records were taken from a semi-intact preparation to which a body wall flap had been left attached by its segmental nerve to an exposed midbody ganglion. Figure 5b shows the result of transient longitudinal stretch of the dorsal sector of the body flap during a swimming episode of such a preparation (*19*). In the ganglion that innervates the body wall flap, stretch causes an increase in the duration and impulse number of the dorsal excitor impulse bursts and shortens, or even abolishes, the impulse bursts of the ventral excitors. Weaker but opposite effects occur in the dorsal impulse bursts in a more anterior ganglion. Other experiments, not presented here, have shown that ventral

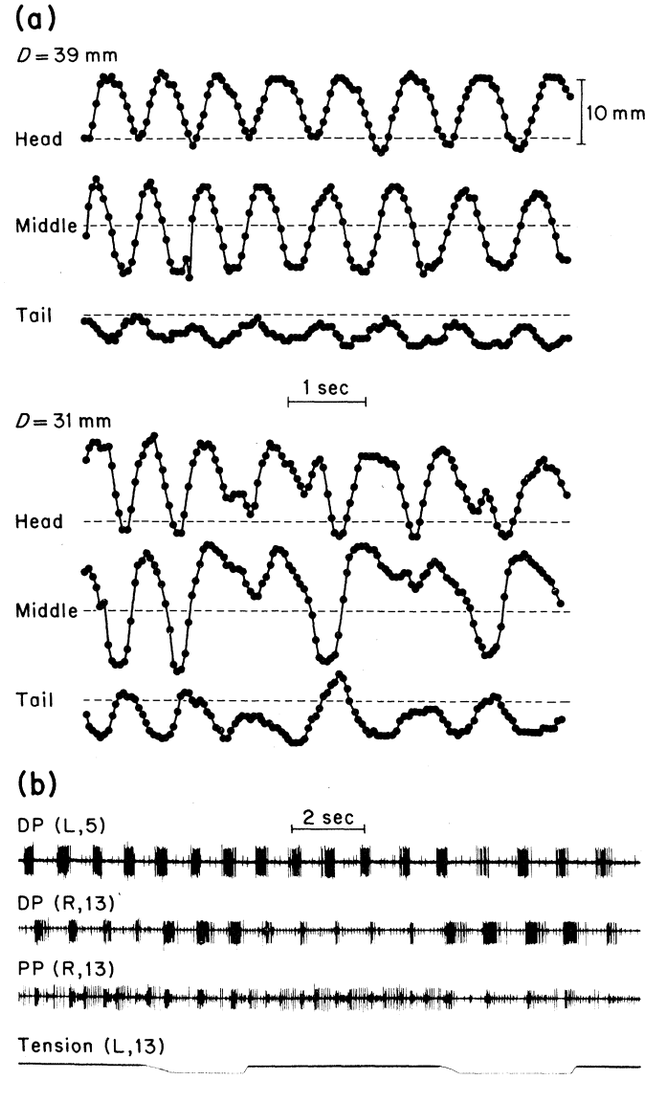


Fig. 5. (a) Upward and downward movement of the beads sewn to the head, middle, and tail segments of a partially restrained, intact leech preparation supported by two pillars separated by distance *D*. The dashed horizontal lines indicate the plane of support provided by the pillars. The measurements were made on magnified, frame-by-frame projections of a film on which the movements of the lateral body silhouette had been recorded. Top: unrestricted swim; bottom: restricted swim. (b) Effect on the swimming rhythm of longitudinal stretch of the dorsal sector of the body wall flap belonging to the 13th abdominal segment of a semi-intact preparation. The downward deflection of the trace labeled *Tension (L,13)* indicates the time at which stretch was applied. The other traces represent the output of suction electrodes attached to the dorsal branch (*DP*) or the posterior branch (*PP*) of posterior segmental nerve. The impulse bursts seen in the *DP* records represent the activity of the dorsal excitor, cell 3, and those in the *PP* record the activity of both dorsal excitors (in-phase bursts of impulses) and ventral excitors (out-of-phase bursts of impulses).

body wall stretch has the opposite effect from dorsal stretch, in that it increases the duration and impulse number of the ventral excitor impulse bursts (and of the dorsal inhibitor bursts), while shortening, or abolishing, the impulse bursts of the dorsal excitors (*21*). Additionally, in a "nearly isolated" preparation, consisting of an isolated nerve cord to which a body wall flap has been left attached by its segmental nerve to a single midbody ganglion, transient body wall stretch shifts the phase of the swimming rhythm in the entire nerve cord (*19*).

These results thus reveal the existence of a segmental reflex loop with negative feedback: from motor neurons to longitudinal muscles to stretch receptors back to motor neurons. This loop opposes longitudinal stretch of the dorsal or ventral segmental body wall. The identity of the stretch receptors in the segmental reflex loop remains unknown. Possible candidates for the receptors are the peripheral nerve cell bodies located at branch points of the segmental nerves (*35*). In the disposition of their cell bodies and the anatomy of their processes, these peripheral neurons resemble some abdominal stretch receptors that detect distensions of the insect gut by stretch of the gastric nerves in which they are located (*36*). In any case, the fact that body wall stretch affects not only the segmental motor neurons but also causes a system-wide shift in the phase of the swimming rhythm indicates that the reflex pathway leads from stretch receptors to motor neurons via the elements of the central swim oscillator.

Although the operation of this reflex loop is clearly not necessary for the generation of the swimming rhythm, it should be noted that the loop could be an oscillatory circuit in its own right, capable of giving rise to a motor neuron impulse burst cycle with appropriate dynamic parameters. This oscillatory potential is given by the inertia provided by the constant delay of about 100 msec that elapses between the time of occurrence of an impulse in a motor neuron and the change in muscle tension, and hence activity of the correspondent stretch receptor, produced by that impulse (*18*). The overshoots produced by this inertial delay would cause the lengths of the muscles forming part of the reflex loop to oscillate about an average, or "resting" value. As long as the inherent dynamics of the central swim oscillator are matched with those of the reflex loop, the rhythmic sensory afference generated by execution of the swimming movement would not normally exert a significant phasing effect on the central oscillator duty cycle. But the stretch reflexes would affect the centrally generated rhythm as soon as the actual body movements do not match those caused by the central oscillator. Just such monitoring effect has been shown to occur also for the phasing of wingbeat in the locust by input from proprioceptors associated with each wing (*33*). It would appear, moreover, that this interaction between the central oscillator and the peripheral reflex loops not only permits modulation of the swimming movement in response to external stresses but also exerts a general stabilizing influence on the centrally generated rhythm (*19*).

## Conclusions

At the phenomenological level, leech swimming shares many features in common with other vertebrate (*37*) and invertebrate (*38*) locomotory rhythms. For instance, all of these locomotory rhythms seem to be generated by a central oscillator, whose period can vary over a broad range; each cycle of muscle contraction is caused by motor neuron impulse bursts that occur in two or more distinct phases of the cycle; sensory input affects both the period and the pattern of the motor neuron activity; and the movement of homologous limbs or body segments is coordinated in a characteristic manner.

Whether these phenomenological resemblances are attributable to similarities in underlying neuronal networks is not known. Since the basic motor neuronal activity pattern underlying swimming in the leech is quite similar to that which produces walking in cat (*39*) and cockroach (*4*), as well as swimming in fish (*40*) and turtle (*41*), it is possible that recurrent cyclic inhibition networks are responsible for rhythm generation in all these locomotory systems. Nevertheless, it must be noted that leech swimming is a rather inflexible locomotory rhythm. Leeches can swim only in the forward direction, and the relative timing of the movements of successive body segments is rigidly fixed. By contrast, other animals can move forward, backward, and even sideways (*42*), and can even move different legs at different rates (*37, 39*). Hence, here the central oscillator network controlling interlimb coordination must be more flexible than that of the leech swim generator.

Since the circuitry of central nervous oscillatory networks is known only in a very few neurophysiologically favorable (invertebrate) preparations (*43*), the question of whether the neuronal circuitry described here generates locomotion also in higher groups, particularly in our own vertebrate subphylum, cannot yet be answered. But it seems significant that the current list of fundamentally different and theoretically plausible types of neuronal oscillators is not only brief but also of long standing (*44*). Moreover, it is worthy of note that the mechanism of recurrent cyclic inhibition evidently responsible for the activity rhythm of the leech central swim oscillator was originally put forward by Székely to account for the walking rhythm of amphibia (*29*). Thus on these grounds it seems reasonable to expect that the applicability of the locomotory circuitry identified in this work transcends the generation of the leech swimming movement.

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# Cognitive Development and Social Policy

## The contribution of parental occupation and education to mental performance in 11-year-olds in Warsaw.

Anna Firkowska, Antonina Ostrowska, Magdalena Sokolowska, Zena Stein, Mervyn Susser, Ignacy Wald

In this study we asked what effect directed social change has had on the distribution of mental performance among school children. We made our observations in a society committed a generation ago to a new political system and a defined social policy.

The question posed was broad. We sought to make the data more specific by differentiating among domains of childhood environment that may influence cognitive development and, thereby, the distribution of mental performance in school children. Certain factors intrinsic to family social structure and position are separated from factors extrinsic to family social structure and position. Factors here termed intrinsic reside in the personal attributes of family members; children experience them primarily within the family milieu. Although such factors also determine experiences outside that milieu, they can exert a direct influence on cognitive development through the mediation of the family socialization process. Parental occupation and education, as well as birth order, family size, and other determinants of family microculture belong in this class (*1*). Factors we term extrinsic reside in the environment external to family social life; children experience them primarily outside the family milieu. Although such factors may influence that milieu, they can exert a direct influence on cognitive development through extrafamilial experience. Schools, housing, health and welfare services, recreation, and criminality and employment rates belong in this class.

The separation of intrinsic from extrinsic factors could be accomplished by research design together with analysis because of the special situation in Warsaw. In that city there had been, it was believed, redress of inequalities of habitat among its people. We aimed to test whether this equalization of extrinsic factors in the wider environment had altered or reduced the gradients in mental performance typically found with social class in other environments.

In Western societies numerous studies have demonstrated the association of social class with mental performance and mild mental retardation. Although defined by occupation, education, income, or social status criteria inherent in family members that we have classed as intrinsic, social class is composed of a complex of factors without and within the family. Families of manual and unskilled laborers, in comparison with those of nonmanual and better educated workers, have generally been poorly housed and poorly schooled, have had inferior health care, and have often suffered from discrimination in social and public life. Given this complex in observed associations of social class with mental performance, extrinsic factors are readily confounded with such intrinsic factors as parental occupation and education.

The family milieu and socialization process is influential in cognitive development. This one may infer from studies of adoptees (*2*), of family types (*3*), of birth order and family size (*4*), and also from the average mental performance of surviving singletons of twin pairs compared with the below-average performance of surviving pairs (*5*). While by now this influence is hardly in doubt, the size of the contribution of intrinsic factors relative to extrinsic factors must remain an open question as long as they have not been clearly separated from each other. Attempts have been made to separate them by multivariate analytical techniques (*6–9*). Such studies are inherently limited by the intimate covariation of the social class complex. Families of the upper social classes who are exposed to the environment of poverty are few and, by

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