by the production of occult seizures (*8*).

Puromycin's effect on memory is not confined to mice or goldfish, is reliable in naive and trained animals, and is extended to include discrimination as well as avoidance and escape behavior. Furthermore, the deficits produced by puromycin may not be attributed to sensory, motor, or motivational factors (*9*). However, puromycin's effect does not appear to be attributable to inhibition of protein synthesis, because AXM had no effect on memory.

To test the latter conclusion, the biochemical actions of these inhibitors on protein and RNA synthesis were investigated with a dual tracer method to study the incorporation of radioactive precursors into RNA and proteins precipitable by trichloroacetic acid (TCA). Five groups of adult male quail (Table 3), maintained at 85 percent of free-feed weight, were injected intracerebrally at each of four sites with either puromycin (45, 90, or 180 μg/site), AXM, or saline. One hour after intracerebral injection, each animal received an intramuscular injection of RNA precursor orotic acid-6-C^{14} (400 μc/kg; 25 mc/mmole; crystals were dissolved in 0.9 percent saline and titrated to *p*H 9 with 0.1*N* KOH to a concentration of 0.14 mc/ml) in the first quadrant of the breast. Four hours later, an intramuscular injection of protein precursor L-leucine-4,5-H^3 hydrochloride (270 μc/kg; 5 c/mmole; stock diluted in 0.9 percent saline to a concentration of 51 μc/ml) was made into the same region. One hour later, the animals were killed, and their cerebral hemispheres were removed, weighed, and homogenized by sonification in ice-cold water (1:1, volume to weight). The RNA and protein supernatants and precipitates were prepared, and the radioactivity was measured in a liquid-scintillation counter by a modification (the elimination of the heat step after the initial TCA treatment of portions of the homogenate) of the method of Brink, Davis, and Agranoff (*10*) (Table 3).

As the concentration of puromycin is increased, inhibition of RNA and protein synthesis is indicated by an increase in radioactivity in the supernatant and the concomitant decrease in the precipitate compared to control values (*11*). Quantitative inhibition of protein or RNA synthesis apparently is not responsible for the memory deficits observed with puromycin. Puromycin (45 μg) caused less inhibition of protein synthesis than AXM, but still inhibited memory; therefore puromycin apparently acts in a qualitatively different fashion from AXM in producing this memory deficit. The lack of effect of puromycin at this concentration (45 μg) on RNA synthesis, but not on protein or memory inhibition, suggests that the qualitative differences in action between puromycin and AXM on memory are related to puromycin's mechanism of action on protein synthesis.

Two possible modes of action for puromycin are either that it destroys memory by inhibiting the formation of specific proteins which make up the engram or that it blocks the expression of the engram by forming a peptidyl-puromycin-engram complex. The second alternative is supported by the report that intracerebral injections of saline restore memory previously lost after the intracerebral injection of puromycin (*12*) and also by the report that peptidyl-puromycin is stable for at least 58 days after initial injection in mouse brain (*13*).

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4. Ten uninjected subjects were eliminated on day 1 (six for lack of response and four for bias). Six subjects were eliminated from the following groups: (i) puromycin (180 μg), one eliminated (no response on day 4); (ii) AXM, three eliminated (two made no responses on day 4 and one died immediately after the intracerebral injections); and (iii) controls, two eliminated (no responses on day 4).
5. A fifth experimental group ($n = 22$) was injected with puromycin (90 μg/site). This group was not included in the analysis because their percentage of free-feed weight was held at 75 percent, 10 percent lower than that of the other groups. Performance of this group, however, was significantly lower than that of the controls on day 4 (t, 4:38; $P < .005$) and on the days to criterion (t, 4.03; $P < .005$).
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15. I thank Dr. J. Zolman in whose laboratory the behavioral work was performed and Dr. D. Knapp for assistance with this project. I also thank Dr. L. Nelson and R. H. Defran for their critiques of this manuscript.

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Central Neuron Initiation of Periodic Gill Movements

Abstract. *In* Aplysia *periodic spontaneous gill movements are controlled by activity endogenous to the abdominal ganglion. These movements were still observed when only the ctenidio-genital nerve was left intact between the ganglion and the gill. One kind of spontaneous gill movement (one per 5 minutes at 15°C) was correlated with the expression of activity of interneuron II; others were not. With reference to this kind of spontaneous gill movement, four types of central neurons in the ganglion send processes to the gill via the nerve. Two cell types (ii, iii) are inhibited and the other two (i, iv) are excited. Two types (i, ii) elicited gill movement—one type activating large gill areas elicited spontaneous gill movements, and the other activating specific gill regions did not participate in the spontaneous gill movements. The value of this preparation in studying the role of central neurons eliciting specific patterned movements and the temporal organization of their activity is shown.*

The neuronal organization of behavior is more amenable to detailed analysis with identification of the same neurons from animal to animal. Techniques are available to study directly neural events which initiate discrete behavior patterns especially in invertebrates. Individual neurons have been identified and their function described in several species. (*1*).

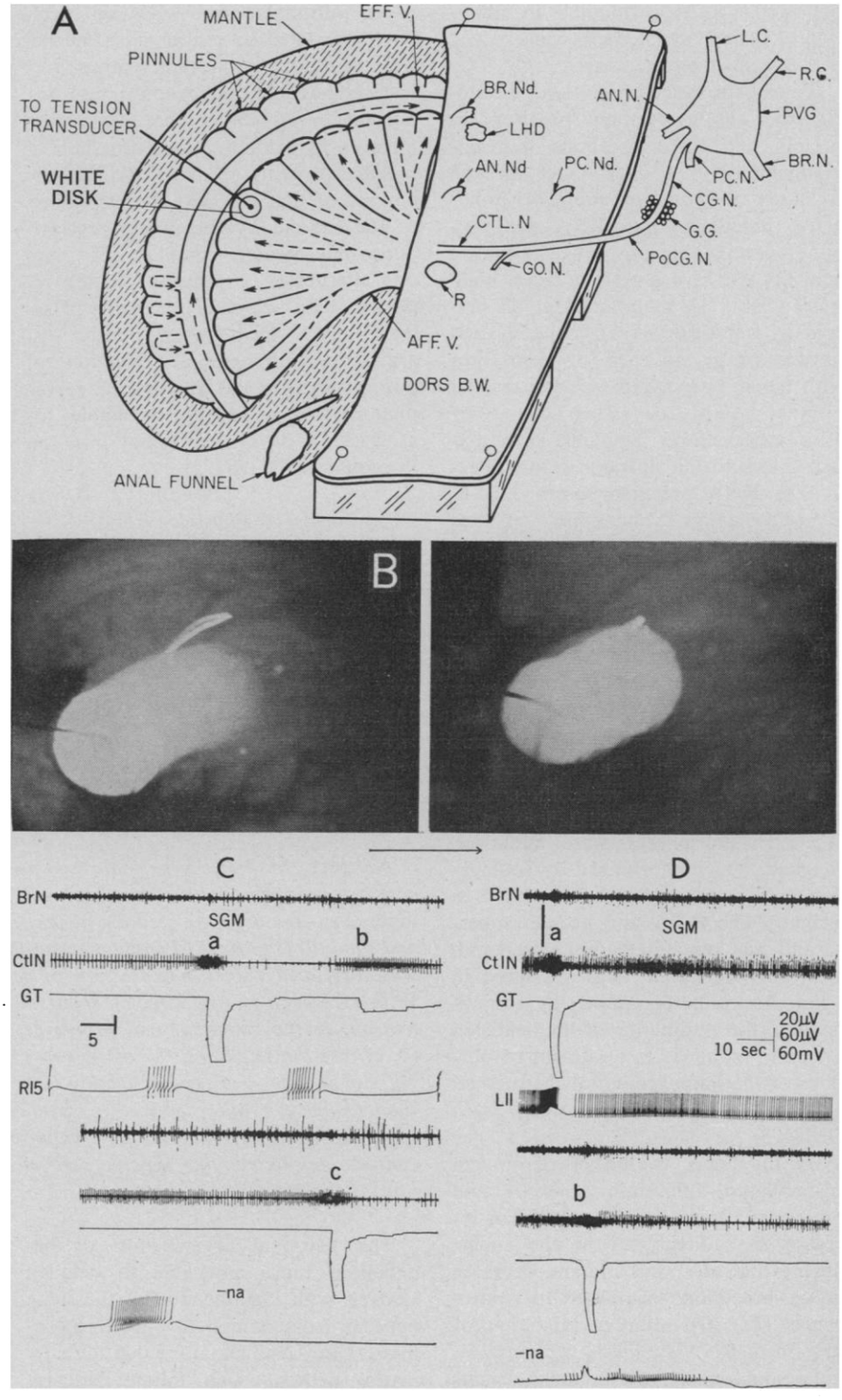
I now describe the properties of central neurons which elicit a specific

periodic pattern of movements in the gill of the sea hare *Aplysia*. Thirty neurons in the abdominal ganglion have been identified, and their discharge patterns and intraganglionic connections have been studied (*2*). The axons of these cells are distributed in the major nerve trunks emerging from the ganglion. However, little is known about the function of these neurons (*3*, *4*) unlike the related *Tritonia* in which particular central neurons are known to elicit specific movements (*1*). If the *Aplysia* nervous system is to be a useful preparation for studying the neural correlates of behavior, the relation between the major ganglia (buccal, cerebral, pedal, pleural, and abdominal) and the periphery must be established.

In freely moving *Aplysia* periodic spontaneous movements of the mantle structures (anal funnel, mantle, and gill) were observed (Fig. 1A). After the animal was pinned out, with its intact central nervous system exposed ventrally, these structures continued to show periodic movements (*4*). Spontaneous gill movements (SGM) persisted after all nerve trunks except the ctenidio-genital nerve [(*5*) and Fig. 1A] were severed from the abdominal ganglion. The gill movements continued as long as this nerve connected the gill to the ganglion, although the other mantle structures no longer showed periodic movements. Thus, neural control of periodic SGM is in part mediated by this nerve (*6*).

Other experiments were carried out in semi-intact preparations from both sexually mature and immature animals (Fig. 1A). The midsection, containing mantle structures, reproductive tract, and the abdominal ganglion, was removed. The ganglion was connected to the gill by only the ctenidio-genital nerve. All the other nerves were cut. The preparation, in sea water at 15°C, was suspended on blocks so that the gill and other mantle structures moved freely and could be observed. A white disk was attached to the outer margins of a pinnule with a thread which connected the gill to a tension transducer. When photographed, the disk traced a path during gill movement (Fig. 1, A and B). The ganglion was fixed to a silastic pedestal containing a light source to illuminate cells from which intracellular recordings were made with micropipettes (filled with 0.6*M* K_2SO_4). A nerve cuff was used to record extracellular activity *en passant* from the ctenidial nerve [(*5*) and Fig. 1A]. Extracellular activity from the proximal cut ends of other nerves, in particular the branchial nerve also going to the gill (*7*), was recorded with suction electrodes (Figs. 1 and 2).

Neurons were stimulated intracellularly, through the micropipette by means of a bridge circuit, to observe direct effects on gill movements. They were also tested as to whether they had processes in the ctenidial nerve (Fig. 2). Extracellular activity in the ctenidial and branchial nerves was synchronized to intracellular recordings from neurons in the ganglion and these were fed to a computer to be averaged (*8*). An extracellular event time-locked to the intracellular activity would indicate the presence of a process in



either nerve or the effect of synchronous postsynaptic potentials. In order to eliminate the latter possibility, each nerve was stimulated individually, and antidromic invasion was observed in the intracellular record while the neuron was hyperpolarized. If increased hyperpolarization of the cell reduced the response and increased its latency, a cell was considered to have a process in the nerve [(*8*) and Table 1].

Though cell morphology and position (*2*) within the ganglion provided initial cues for cell identification, these properties by themselves were insufficient. Size, shape, pigmentation, and location varied. During this study the discharge pattern, especially during SGM, and the nerve trunks by which the cells' processes innervate the periphery were more reliable cell signatures. Four cells, close together, are described below. They are similar in appearance, have very similar discharge patterns, and have processes in the same two trunks. Each had a particular motor field in the gill which differentiated it.

At least two types of gill movements were observed and correlated with distinguishable bursting patterns in the ctenidial nerve (Figs. 1B and 2A). The SGM is correlated with the effects of, as yet unidentified, interneuron II (*3*, *4*), and it occurred on the average of one per 5 minutes at 15°C (*4*). Kandel *et al.* (*2*) have indicated the manner in which interneuron II's activity can be assayed, for example, inhibition of long-duration action in neuron R15 (Fig. 1B). Spontaneous gill movements have been observed which appear to reflect intraganglionic activity other than that of interneuron II (*9*).

Of the 18 cells studied on both dorsal and ventral surfaces, 11 have processes in the ctenidial nerve. Ten of the neurons which were located on the dorsal surface of the ganglion were classified into four cell types. An eleventh cell did not fit any of the types (Table 1). Classification was based on whether a cell was excited or inhibited during SGM correlated with the effects of interneuron II and whether it elicited gill movements when stimulated intracellularly (Table 1).

Type i cells, SGM elicitors. These cells were active prior to and during SGM and elicited gill movements. One cell of this type, LD1, increased its firing rate about 3 seconds prior to initiation of SGM with the rate going from approximately 3 to 8 impulses per second (Fig. 2A). Gill movements appeared to be sensitive to the discharge rate as no movements were noticeable at 3 impulses per second, but they did occur at 8 impulses per second (Table 1, Fig. 2A). Upon the cell's activation, the two rows of pinnules bent away from the midline ("flaring out") exposing the efferent vessel [(*6*) and Fig. 1A]. The movement, seen over the entire gill, was similar to that observed during the SGM. When the cell was hyperpolarized, the extent of movement during SGM was considerably reduced and flaring out was not observed (Fig. 2A). Also, ctenidial nerve and intracellular records suggested that interneuron II's rate of periodic expression was increased during hyperpolarization. Thus, this cell type may be involved in modulating interneuron II's activity.

Type ii cells, neurons eliciting movements subsequent to SGM. These cells were inhibited during the initiation of SGM, yet they elicited gill movements when stimulated intracellularly. Each of four cells [L7, LC1, LC2 (L9), and LC3], found on the left side of the dorsal surface, activated a particular motor field in the gill (Table 1). Inhibition of these cells was due to a high rate of inhibitory postsynaptic potentials during interneuron II's activity (*2*). Type ii cells discharged in bursts upon recovery from inhibition while the gill was still moving (Fig. 2B) and exerted their influence after SGM were initiated. Movements elicited by these neurons are also sensitive to impulse rate (Fig. 2B). Impulse rate and response latencies in the gill are directly related. Sensitivity of gill movements to impulse rate of type i and ii cells may be due to a facilitatory influence at the axon terminals in the gill rather than in the ganglion; this has already been described at motor fiber terminals in crayfish (*10*). Type ii cells have a more restricted peripheral effect than LD1 by way of the ctenidio-genital nerve and, unlike LD1, they have processes in the branchial nerve.

Type iii cells. These cells, inhibited during SGM, did not have direct effects on gill movements. Bursting neurons, L4, L6, and R15, characterize this type. When SGM occurred, interburst interval of R15 lengthened reflecting the effects of interneuron II [inhibition of long duration preceded by an excitatory phase (*2*)]. Other neurons in the ganglion also modulate the interburst interval [(*2*) and Fig. 1C]. Neuron R15 (*11*) is particularly interesting because its discharge rate reflects a circadian rhythm (*8*), and the animal's locomotor activity also shows a daily rhythm (*12*).

Type iv cells. These cells did not have direct effects on gill movements, yet they had increased discharge rates during SGM. Neurons L11 and LD2

Fig. 1. (A) Semi-intact preparation showing the abdominal ganglion connected to the gill by only the ctenidio-genital nerve (*6*). The genital ganglion straddles the ctenidio-genital nerve on the ventral surface of the reproductive tract. Eales (*7*) has described, in part, the innervation of this nerve with reference to the reproductive tract. The arrows indicate the flow pattern of the blood [see also (*6*, *7*)]. *AFF.V.*, afferent vessel; *AN.N.* and *AN.Nd.*, anal nerve; *BR.N.* and *BR.Nd.*, branchial nerve; *CG.N.*, ctenidio-genital nerve (*5*); *CTL.N*, ctenidial nerve; *DORS.B.W.*, inner surface of dorsal body wall; *EFF.V.*, efferent vessel; *G.G.*, genital ganglion; *GO.N.*, gonadal nerve; *L.C.*, left connective; *LHD*, large hermaphrodite duct; *PC.N.* and *PC.Nd.*, pericardial nerve; *PoCG.N.*, postctenidio-genital nerve; *PVG*, parieto-visceral (abdominal) ganglion; *R*, rectum; *R.C.*, right connective. (B) Path of white disk during SGM correlated with interneuron II and gill movement elicited by a single neuron. (Left) SGM; (right) gill movements elicited by L7; both from same preparation. Arrow indicates direction of movement. (C) Simultaneous recordings from neuron R15 (type iii) intracellularly and of gill tension during SGM. Branchial and ctenidial nerve activity was recorded extracellularly. (Upper) Two SGM; *a* is correlated with the activity of interneuron II as seen in the intracellular record of R15 [inhibition of long duration preceded by excitation (*1*)]; gill movement at *b* is correlated with another intraganglionic event. The interburst interval at *a* and *b* is longer than when no gill movement occurs. No evidence of increased activity in branchial nerve at *b*. (Lower) SGM, *c*, occurs with no noticeable changes either in amplitude or rate when R15 is hyperpolarized. Vertical scale: 30 μv, *BrN*; 100 μv, *CtlN*; 60 mv, R15; time, 5 seconds; *na*, onset of hyperpolarization; *GT*, gill tension. (D) Simultaneous recordings from L11 (type iv) intracellularly and of gill tension during SGM. Branchial and ctenidial nerve extracellular records (C, above). Notice at both *a* and *b* (due to interneuron II) the burst in the *CtlN* precedes that in the *BrN*, see vertical line. (Upper) High rate of discharge in L11 precedes the SGM at *a*. Small gill movements occur following *a* as L11 discharge rate is increased. (Lower) When L11 is hyperpolarized, —*na*, SGM still occurred, *b*. There was no apparent change in amplitude of gill tension. Notice two distinct postsynaptic potentials during hyperpolarization—one correlated with activity of interneuron II, the other occurs before and after SGM.

Table 1. Neurons of the abdominal ganglion (PVG) of *Aplysia* with processes in the ctenidial nerve. Movement A, posterior pinnule rows come together, constriction of efferent vessel is inferred; movement B, bunching of pinnules on posterior gill (whole gill moves thusly with only the branchial nerve intact), contraction of efferent vessel is inferred; movement C, contraction of afferent vessel; movement D, outward bending of posterior pinnules; movement E, pinnules "flare out" over entire gill; ips, impulses per second.

Cell (*1*)	Type	Activity during SGM*	Direct effects†	Nature of movement	Range (ips)	Movement latency (sec)	Process in branchial nerve	Sensory input‡
L4	iii	−	N.O.				Yes	
L6	iii	−	N.O.				N.O.	
L7§	ii	−	Yes	A	8–18	1.1–2.4	Yes	Yes
L11§	iv	+	N.O.				N.O.	
LC1	ii	−	Yes	B	5–17	0.8–3.4	Yes	Yes
LC2 (L9)§	ii	−	Yes	C	5–12	1.6–3.2	Yes	Yes
LC3	ii	−	Yes	D	4–17	0.7–3.6	Yes	Yes
LD1	i	+	Yes	E	7–12	0.9–1.8	N.O.	
LD2	iv	+	N.O.				N.O.	
R15	iii	±	N.O.				N.O.	N.O.
RB1		0					Yes	N.O.

* Postsynaptic potential action by interneuron II: +, excitatory; −, inhibitory; 0, none. See text. † N.O., none observed. ‡ Tactile stimulation of the gill initiated spiking in cell. § Comparable cells have been found in *Aplysia dactylomela* (*16*).

are of this type and had discharge patterns similar to LD1 (Fig. 1D) and also had no processes in the branchial nerve (Table 1). No noticeable movements were observed in response to high discharge rates during depolarization. Hyperpolarizing L11 did not suppress SGM (*11*).

Though both the branchial and ctenidial nerves innervate the gill, no type i cells have been found with processes in the former. The burst in the ctenidial nerve precedes that in the branchial nerve (Fig. 1D), yet the conduction distance to the recording site is twice as long in the branchial nerve. Type ii cells, active subsequent to SGM initiation, have processes in the branchial nerve. Branchial nerve activity may be controlling the next pattern of movements after the SGM. The dual innervation, in part, may be to transport blood through the gill by sequential pumping movements [(*6*) and Table 1].

Temporal organization of spontaneous patterned gill movements resides in the abdominal ganglion. Effects of interneuron II's activity, also seen in intracellular recordings from the isolated ganglion (*2*), appear to involve a subset of neurons in the ganglion (RB1 in Table 1). The resulting output elicits gill movements. This does not rule out modulatory effects by head ganglia or by afferent inflow from the periphery, especially the gill. Extracellular recordings from the anal and pericardial (*13*) nerves in addition to the branchial and ctenidio-genital nerves show bursts correlated with interneuron II's activity. A reasonable explanation for multiple paths of this expression is that functions at the innervation sites of the nerves, that is, heart, kidney, and gill, are synchronized with respect to blood circulation and are under central control (*6*).

An invertebrate preparation for study of the neural organization of centrally initiated behavior has been described. It can lead to greater understanding of periodic behaviors in vertebrates such as rapid eye movements associated with activated sleep (*14*) which are also centrally initiated. Identified neurons

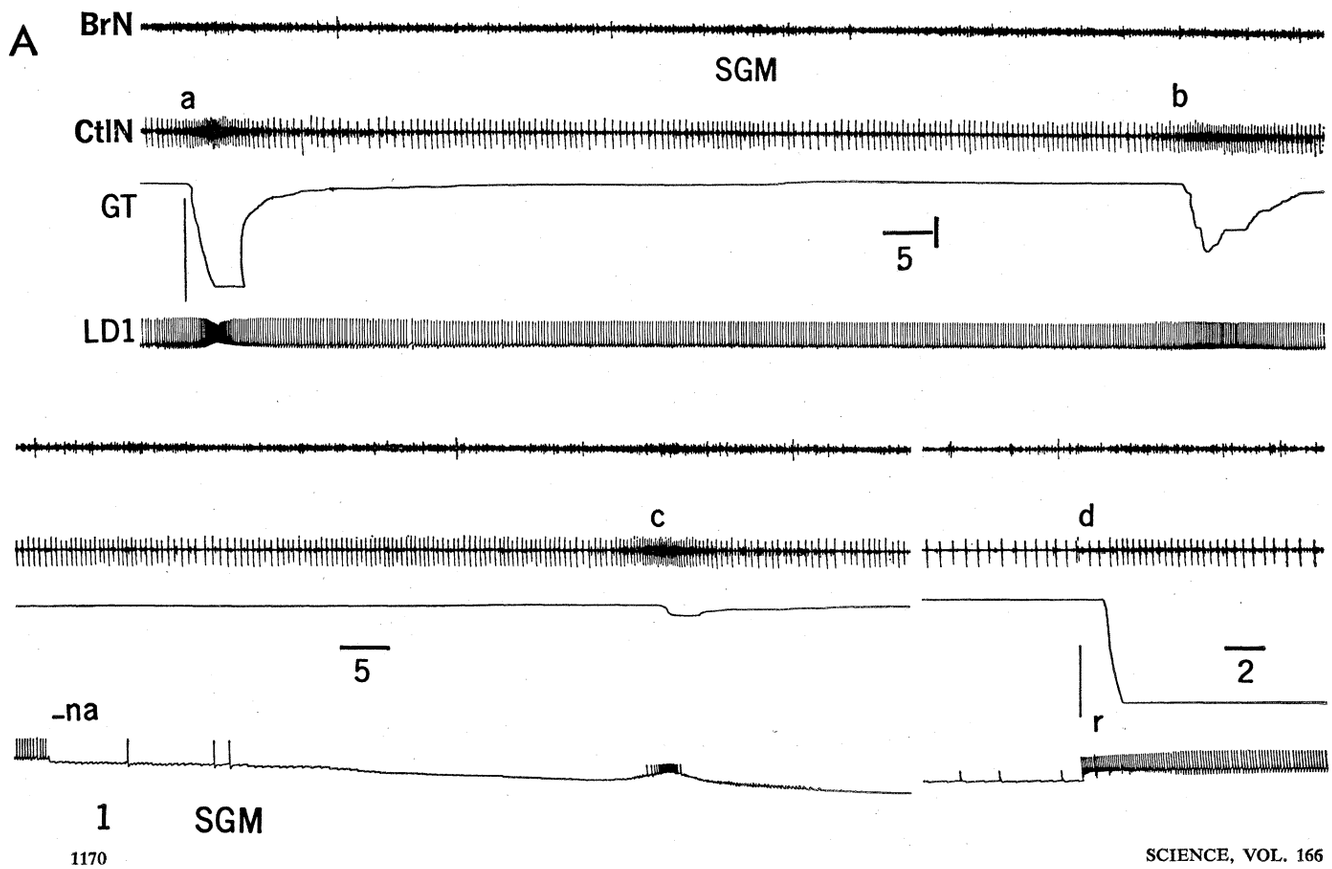
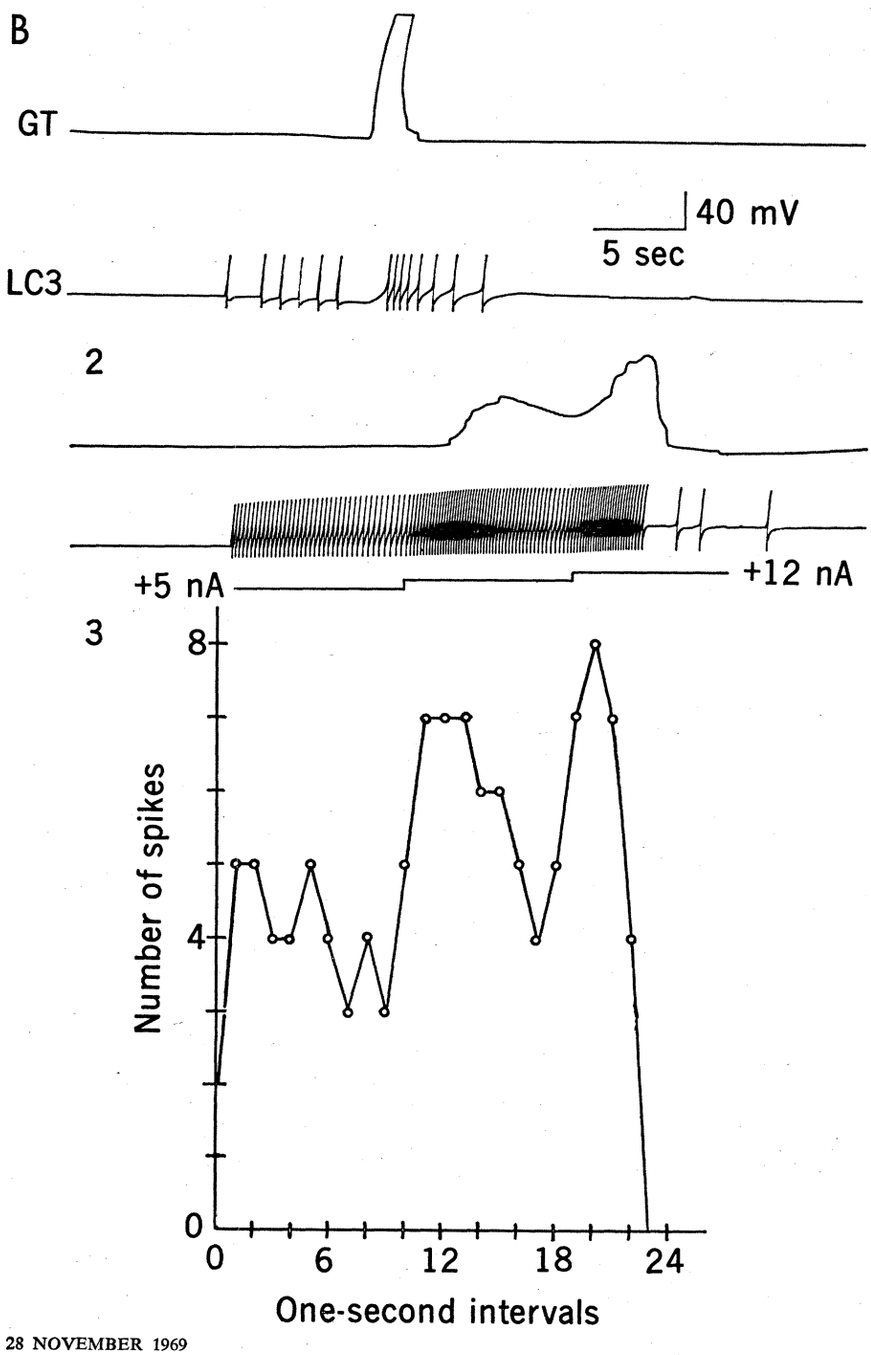


Fig. 2. (A) Simultaneous recordings from LD1 (type i) intracellularly and of gill tension during SGM. Branchial and ctenidial nerve extracellular records (Fig. 1C). (Upper) SGM and other gill movement about 2 minutes apart at *a* and *b; a* is correlated with interneuron II's activity; *b* is not (see text). The discharge rate of LD1 increases about 3 seconds before SGM occurred at *a*, from 3 to 8 impulses per second. Between *a* and *b* the rate was about 3 impulses per second. (Lower) At *—na* LD1 was hyperpolarized. SGM still occurred, *c*, but the amplitude was markedly reduced. When the cell was released from inhibition, *d*, the gill pinnules bent outward and remained in that position during the high discharge rate, 14 impulses per second at *r* (release). There was no noticeable recruitment in the abdominal ganglion as reflected in the *CtlN* and *BrN* records (see text). Scale: 30 μv, *BrN*; 150 μv, *CtlN*; 60 mv, *LD1*; time in seconds. *GT*, gill tension. (B) Simultaneous record of gill tension and from LC3 (type iv) intracellularly. [1] LC3 is inhibited at the initiation of SGM; during the movement the cell discharges. [2] The effects of incremental depolarization on gill tension. Decrease of discharge rate indicated cell adapted to applied current level. As the cell adapted to 12 μa the rate decreased; yet the gill held the tension even as the rate fell off. [3] The graph indicates the number of impulses which occurred during each second while the cell was stimulated, from the record in [2]. The threshold for eliciting gill movement was between 5 to 7 impulses per second with a latency of 2 to 3 seconds. The movement was clearly rate-sensitive. *GT*, gill tension.



in the *Aplysia* abdominal ganglion, periodically activated, elicit stereotyped movement patterns in the gill; and each neuron's influence is projected to a specific region. These nerve cells control movements in an organ vital for the animal's physiological maintenance; and in this sense they are comparable to central neurons controlling respiration and correlated cardiovascular activities in vertebrates. They are not merely participating in reflexive behavior of peripheral structures. It is not known whether the *Aplysia* central neurons which have processes in the gill actually innervate the musculature. Their level in the neuronal hierarchy [compare, the command fibers in crustaceans (*1*), the executive cells in *Tritonia* (*1*), and pyramidal tract neurons in primates (*15*)] is yet to be determined. This report shows that a neuron's impulse rate regulates the extent and latency of movement (Table 1). Similarly, pyramidal tract neurons in the unanesthetized monkey show firing rates proportional to the force developed at the wrist (*15*). In *Aplysia* an individual neuron's role in the motor periphery as well as the functional significance of its discharge pattern can be determined.

Note added in proof: A recent report by Kupfermann and Kandel (*17*) listed four neurons in the abdominal ganglion involved in spontaneous and reflexive gill withdrawal. Only one cell with certainty, L7, is common to both reports. The effects of its activity in the gill, as well as that of two other neurons (L9-1, L9-2), were not indicated by the authors. The innervation of the gill was not given. In contrast, the present paper reports the neuron complement involved in gill movements which are respiratory and possibly circulatory in function. The path of each identified neuron's process is determined.

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tegration, F. D. Carlson, Ed. (Prentice-Hall, Englewood Cliffs, N.J., 1968), p. 17. The cell nomenclature by the above authors has been used throughout this paper. The modifications used here are an extension of the nomenclature, for example, LC1 and LD1 cells.
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6. During SGM the afferent vessel contracts, moving the entire gill toward the dorsal surface of the body wall (Fig. 1A). Two rows of pinnules, fingerlike projections, which are sites of gaseous exchange between the blood and sea water, separate. The pinnules bend away from the midline of the gill and expose the efferent vessel which carries the blood toward the auricle of the heart (*7*). As the gill returns to the rest position, the efferent vessel appears to contract. The branchial nerve also innervates the gill (*7*). When it is the only nerve connecting the ganglion to the periphery, it elicits gill movements clearly distinguishable from those described above; the pinnules clump together, similar to a fan being closed. The entire gill then moves rostrad, pivoting about the afferent vessel.
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9. Interneuron I, L10 (*1*), was inhibited during SGM; its activity did not contribute to the movements. This cell does not have a process in the ctenidio-genital nerve but does have one in the pericardial nerve (*13*).
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11. Neuron R15, presumed to be neurosecretory (*2*) and having no observable effects on the gill, may have long-term influence, for example, metabolic effects on muscle in the gill which could regulate gaseous exchange over circadian intervals. Neuron L11, a non-secretory cell, could control the activity of cilia in the gill.
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13. Anatomical descriptions of this ganglion have treated the genital and pericardial nerves as separate trunks [see Eales (*7*) and Strumwasser (*8*)]. Frazier *et al.* (*2*) have considered the nerves as a single trunk. These nerves do have overlapping constituents, for example R15. However, there are cells, for example L12 and L13, each of which has a process in only one of the nerves; L12 has one in the genital nerve, and L13 has one in the pericardial nerve. The pericardial nerve, when stimulated, did not elicit gill movements.
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18. I thank F. Strumwasser for encouragement, support, and comments. I also thank J. J. Gilliam for technical assistance. Supported in part by PHS special fellowship 7 FO3 GM-32, 521-03, grant NB 07071, and NASA grant NGR 05-002-031.
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Ultraviolet Video-Viewing: The Television Camera as an Insect Eye

Abstract. *A television camera, like the eyes of some insects, is sensitive to ultraviolet light. When equipped with an appropriate ultraviolet-transmitting lens, such a camera can be used for the direct examination of ultraviolet reflection patterns (for example, on flowers, butterflies) that are invisible to us, but visible to insects.*

Honeybees and certain other insects, unlike man and possibly all vertebrates, are visually sensitive and behaviorally responsive to ultraviolet light (*1*). Objects such as flowers may possess ultraviolet reflection patterns, invisible to us, but visible and significant to pollinating insects (*2*). The demonstration of such "hidden" ultraviolet patterns, whether on flowers or sometimes, as in the case of butterflies (*3*), on the insects themselves, has hitherto relied on the use of special photographic techniques, all somewhat elaborate, and unsuited for direct examination of subjects in motion (*2–5*). An ordinary television camera, by virtue of its intrinsic sensitivity to ultraviolet light (*6*), providing only that it is equipped with an ultraviolet-transmitting lens, can serve as an appropriate "eye" by which the ultraviolet patterns of nature may be directly observed. Portable video units are particularly suitable since both video camera and tape recorder can be carried by one individual in the field (Fig. 1A). We have used a Sony