Neurophysiology of predator avoidance

Seeing and assessing what is coming.

Last-ditch behavior to avert the threatened strike.

Long-distance detection
Visual information
Alternative strategies or stage responses
Response latency: > 100 ms

Short-distance detection
Mecanosensory information
Highly stereotyped single strategy
Response latency: < 10 ms

Courtesy of Jens Herberholz
7.2 Photographic analysis of tail flips initiated by the giant interneurons

Drawings from high-speed cinematographs. (A) A tactile stimulus to the tail fan at the rear of the animal elicits a tail flip mediated by the lateral giant interneurons that moves the animal upward. (B) The same stimulus to the front of the animal elicits a tail flip mediated by the medial giant interneurons that propels the animal backward. After Wine and Krasne 1972.

Fig. 6.1. Giant interneurons involved in crayfish startle behavior. (a) Crayfish showing the location of the central nervous system (in solid black), which consists of a chain of segmental ganglia linked by paired connectives. Also shown are implanted electrodes for recording neural activity in the freely moving animal. (b) Activity recorded by these electrodes during a startle response: a tap on the abdomen (stimulus) is followed by an impulse in a giant interneuron (lateral giant) and a muscle potential in the abdominal flexor muscles. (c) Transverse section of the connectives between two abdominal ganglia, showing the location of the axons of the two giant interneurons (lateral giant and medial giant) in relation to other large axons. ((a) modified after Schrameck, 1970; (b) redrawn after Krasne & Wine, 1975; (c) redrawn after Krasne & Wine, 1977.)
Natural versus electrical stimulation of the tail flip

7.3 Natural versus electrical stimulation of the tail flip
Electrical stimulation of an MGI elicits a tail flip response very similar (A) to the one elicited by a tactile stimulus to the front of the animal (B). The numbers in parentheses refer to the time (in milliseconds) from the onset of the stimulus. After Wine and Krasne 1982.

7.5 Connectivity patterns of the giant interneurons
The left and right side panels show MGL- and LGI-mediated tail flips, respectively. The schematic wiring diagram illustrates that the MGLs make synaptic contact (solid circles) with the motor giants (MoGs) in every abdominal ganglion, whereas the LGIs connect with the MoGs only in ganglia 1 through 3. (Asterisks indicate the lack of an LGI synapse in ganglia 4 through 7.) The abdominal segments 1 through 6 are illustrated below. The net result of this connectivity is that the MGLs cause all segments to flex, whereas the LGIs activate only the rostral segments. After Wine and Krasne 1982.
7.6 The tail flip circuit

(A) The core LGI circuit (same as in Figure 7.1B). (B) A schematic wiring diagram of the tail flip circuit. The direct inputs to the LGIs via electrical synapses (α) and indirect inputs via chemical synapses (β) are indicated. After Wine and Krasne 1982; data from Krasne 1969, Zucker 1972, and Kennedy, Calabrese, and Wine 1974.

7.10 Input to the LGI

The LGI receives input from both electrical and chemical synapses, as this intracellular recording shows. Activation of the tail afferents (stimulus) gives rise to two components of the complex synaptic input (EPSP) to the LGI: the short-latency alpha (α) component (electrical input), and the longer-latency beta (β) component (chemical input). The net synaptic input produces an action potential (impulse) in the LGI. (The top of the impulse has been cropped in this illustration.) After Krasne 1969.
Fig. 6.4. Neuronal circuit for startle behaviour mediated by the lateral giant interneuron. (a) Schematic representation of the excitatory pathway from the mechanoreceptors to the flexor muscles, showing chemical (---) and electrical (-----) synapses. Labelled circles represent the receptors (R), sensory interneurons (SI), lateral giant (LG), segmental giant (SG), motor giant (MoG) and fast flexor motor neurons (FF). The sensory pathways that generate the two components of the compound EPSP are indicated (α, β, cf. Fig. 6.3(c)). (b) Diagrammatic representation of the arrangement of the above components within the abdominal nervous system. The various components are not drawn to scale and only one segment of the lateral giant is shown; the segmental giant and fast flexor motor neurons are not included. (Modified after Wine & Krasne, 1982.)

Fig. 6.3. Electrical synapses between neurons involved in crayfish startle behaviour. (a) Drawing of an abdominal ganglion to show the relative positions of the lateral giant and motor giant neurons, the synapse between them and the arrangement for recording from each side of this synapse. The segmental synapse between successive lateral giant neurons is also shown. (b) Simultaneous, intracellular recordings from lateral giant and motor giant neurons close to the synapse, demonstrating the negligible delay due to electrical transmission. (c) Intracellular recording from the lateral giant neuron at a point close to the segmental synapse. Following electrical stimulation of the appropriate sensory neurons, a compound EPSP and an impulse are recorded in the lateral giant neuron with a very small delay. The two components of the EPSP (α and β) are produced by separate pathways from the sensory neurons (details in text). (a) and (b) modified after Furshpan & Potter, 1959; (c) redrawn after Krasne, 1969.)
7.11 Testing the necessity of the LGIs for the tail flip

In the experimental setup (A), two intracellular electrodes are placed in the LGI—one for recording the membrane potential ($R_{\text{LG}i}$), the other for passing hyperpolarizing current (Hyp). Two extracellular electrodes are placed on the nerve cord ($R_{\text{NC}}$) and a motor root ($R_{\text{Mot}}$). A stimulating electrode (St) is placed on a nerve root to activate sensory input. (B) When the LGI is not hyperpolarized, the activating stimulus triggers an action potential (AP) in the LGI and output (APs) in the motor nerve. (C) When the LGI is prevented from firing by hyperpolarization, the same sensory input triggers no action potentials in the motor nerves. After Camhi 1984; data from Olson and Krasne 1981.
Fig. 6.6. Inhibition of the abdominal extensor muscles by the lateral giant interneuron. (a) Neuronal circuit generating inhibition of the extensors, showing representative neurons: the lateral giant (LG), fast extensor motor neuron (FE), extensor inhibitor (EI), the muscle receptor organ (MRO) and its accessory cell (AC). Inhibitory neurons are shown in solid black; excitatory (—•—•) and inhibitory (———•) connections are shown but no distinction is made between chemical and electrical synapses. (b) Typical recording used to build the interpretation given in (a). Upper trace is an extracellular record from the motor nerve to the flexors, showing the lateral giant impulse and subsequent compound impulse from flexor motor neurons. Middle and lower traces are intracellular records showing, respectively, an EPSP in the extensor inhibitor and an IPSP in a fast extensor motor neuron; note the short delay of the postsynaptic potentials after the lateral giant impulse. ((b) from Wine, 1977.)
Command-derived inhibition

Inhibition of the abdominal extensor muscles by the LG

Delayed inhibition of the fast flexor muscles by the LG
Location and timing of command-derived inhibition

(A)

- = Chemical
- = Electrical
- = Inhibitory

Mechanosensory neurons → Interneurons → Command and decision neurons → Premotor and motor neurons → Muscles

(B)

Stimulus
- Command
- Flexor motor neurons
- Movement
- Inhibitory actions
  - a 1st synapse pre
  - b 1st synapse post
  - c LGI
  - d MoGs
  - FFs
  - e Muscles

Time (ms) →
¿El nado depende de la LG?

Experimento:
Estimulación mecánica que provoca 100% tailflip también provoca 82% de nado. Estimulación electrica de las LG (i.e. bypassing the sensory system), provoca menos de 1% de nado.

Además…
7.15 Triggering of swimming

Sensory input triggers swimming independently from the tail flip and reextension phases of escape. (A) This schematic diagram shows that a tactile stimulus ($S_1$) triggers swimming ($R_1$) with a delay) in parallel with triggering a tail flip ($R_2$) and a reextension reflex ($R_3$). $S_2$ indicates proprioceptive input (arising from $R_1$) that triggers reextension. (B) The latencies for LGI-mediated tail flips and for nongiant swimming episodes elicited by tactile stimuli are shown. (C) Even when LGI responses do not occur in response to a tactile stimulus, swimming episodes occur with much the same latencies. After Reichert and Wine 1983.
7.16 The complete escape response

This schematic diagram shows the complete escape response (flexion, reextension, and swimming). A tactile stimulus directly activates two sensory processors: One (a) triggers the LGI-mediated tail flip (flexion), and the other (c) triggers a delayed CPG-mediated swimming response. The second phase (reextension) is triggered by a third sensory processor (b), which is activated by input derived from the flexion response. Arrows indicate the flow of information, and numbers aligned with the arrows indicate relative time of occurrence. Solid circles indicate functional inhibition; arrowheads indicate functional excitation. Note that all three phases of escape share common flexion and extension circuitry. After Wine 1984.
Flexibilidad de la respuesta de tail flip

Modulación

La respuesta de escape tiene costos o desventajas:

_energéticamente costosa
_impide otros comportamientos (competing behaviors)
_el movimiento favorece la detectabilidad.
7.18 Feeding inhibits the tail flip response

(A) Gradually increasing the strength of a triggering stimulus finally brings it to threshold for a tail flip (filled circles). While the animal is feeding (trial block II), the threshold for the tail flip further increases (no tail flips occur) from a level (dashed horizontal line) that normally elicits responses in nonfeeding animals (trial blocks I and III). (B, C) The increase in threshold requires that an animal actually be engaged in a feeding response (B), trial block II; simply being in the presence of food (C) does not increase the threshold. (D) When the nerve cord is cut (darker bars) feeding-induced increases in threshold are abolished compared to normal animals (lighter bars). After Krasne and Lee 1988.

7.19 Feeding increases the threshold for LGI activation

(A) Sites in the tail flip circuit where the stimulus (S) was delivered and where the LGI response (R) was recorded intracellularly. (B) As the stimulus strength is increased, the threshold for action potentials in the LGI is reached (the traces are clipped on the top and bottom). (C) By the same technique as in part B, the threshold for spikes in the LGI (filled circles) is determined. During a feeding episode (trial block II) the threshold is increased such that no spikes are elicited (open circles) at a level (dashed horizontal line) that normally elicits spikes in nonfeeding animals (trial blocks I and III). (D) Average data illustrating the dramatic increase in LGI threshold during a feeding episode. A and B after Krasne and Lee 1988.
**Dos tipos de inhibición**

**Rigida:** command derived inhibition

**Flexible:** por sujección, alimentación, etc
Startle behaviour

(a) Diagram of a fish with electrodes attached to it and a speaker nearby. The diagram shows the startle response pathways.

(b) Graph showing muscle potential over time with labels for ipsi and contra. The graph includes markers for On and Off.

(c) Graph showing the stimulus timing with markers for Mauthner and muscle potential. The graph also shows the timeline for Stage 1 and Stage 2.

Time scale: 2 ms

Muscle potential

Stimulus

Mauthner

Muscle potential

Stage 1 begins

Stage 2 begins
Mauthner neurons and the teleost fast start

(a) M
(b) Dorsal
Axon cap
Lateral dendrite
Lateral line
Ventral dendrite
Vestibular
Lateral dendrite
Axon cap
Small dendrites
100 μm

25 μm

(a) SI
R
(b) Impuls 1 ms
command-derived inhibition