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THE STRUCTURAL BASIS OF AN INNATE BEHAVIOURAL PATTERN

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SUMMARY

The abdominal nervous system of the crayfish contains six serially homologous ganglia, each containing approximately 650 neurones. No two ganglia are identical, and the ganglia interact extensively. Studies confined to intraganglionic interactions thus yield limited and sometimes misleading information. Each ganglion contains intrinsic (local) interneurones, motor neurones and projecting interneurones in roughly equal numbers, except in the specialized terminal ganglion where the ratio of these cells is approximately 3:2:1. Although the number of nerve cell bodies in a ganglion is small enough to be tractable, integration occurs in the neuropile, which contains terminals from interneurones and afferents that outnumber the neurones originating in the ganglion by at least ten to one. The abdominal nervous system responds almost exclusively to a variety of mechanosensory stimuli. It has very limited light sensitivity. Other modalities, notably chemosensitivity, are undescribed and may be lacking. The effectors of the abdomen consist of fast axial muscles (used for tailflip-powered escape), slow axial muscles (for setting abdominal posture), appendage muscles (for swimmeret beating), and slow muscles of the intestine and rectum (that control gut emptying). The fast and slow muscles of the tailfan are specialized homologues of the axial and appendage muscles.

The abdominal nervous system represents only 3–4% of the 100000 neurones within the crayfish central nervous system (CNS). Most sensory information gathered in the abdomen is transmitted to the rostral CNS for processing, and many abdominal motor programmes are activated by descending commands. Nevertheless, a surprising degree of autonomy is present, and at least some motor programmes of every motor system can be activated in isolated abdomens.

Tailflip escape behaviour illustrates the integrative properties of the crayfish nervous system. Ninety pairs of efferents and eighteen pairs of interneurones have been identified within the abdominal portion of the escape circuit. A cell-by-cell analysis has so far provided neurophysiological explanations, in varying states of completeness, for ethological concepts such as innate releasing mechanisms, spatial patterning of movement, serial order in behaviour, and alterations in responsiveness to a constant stimulus.

INTRODUCTION

'. . . is it not possible that beneath all the variations of individual behavior there lies an inner structure of inherited behavior which characterizes all the members of a

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given species, genus or larger taxonomic group – just as the skeleton of a primordial ancestor characterizes the form and structure of all mammals today? Yes, it is possible!'

Konrad Lorenz (1958)

Recent work, mainly on invertebrates, has established beyond doubt the reality of an inner structure for inherited behaviour. Ethological concepts like innate releasing mechanisms and fixed action patterns have been shown to be the products of specific neural networks, whose operations can account for many of the salient features of behaviour (e.g. Kandel, 1976). The explanation of behavioural concepts in terms of integrative neurophysiology requires, as an essential step in the analysis, the fractionation of exceedingly interactive networks in ways that are informative rather than merely destructive. There are two general approaches to fractionating nervous systems. One is to focus on a particular behaviour pattern (e.g. Kennedy, 1975). Focus on a particular pattern can be accomplished by using a stimulus that evokes a repeatable behavioural response; the neural systems that are activated by the stimulus can then be studied. The other approach is to focus on a particular neuroanatomical entity (e.g. Maynard, 1972). This is especially useful in segmented animals, where it is inviting to cut through the cables of pure axons that string neural ganglia together and then analyse the isolated portions. Discrete anatomical units containing from 10 to approximately 10^5 neurones have been studied in this way.

Each approach has inherent limitations. Kennedy (1975) has advocated the former approach in the following terms:

'. . . behaviors, not anatomical entities, are the elements on which natural selection works in shaping neuronal organization. Thus the system we must complete is the behavioral act, not the anatomy of the control center. Elements in the neuronal ensemble may participate in other behaviors as well; or the anatomical unit may contain behaviorally irrelevant elements. Neither should matter as long as our inventory of the neurons and connections that participate in the behavior is complete.'

We have found it useful to use both strategies by concentrating on those aspects of crayfish escape behaviour that can be studied in the isolated abdomen. In this review, I give a brief overview of the main features of the abdominal nervous system of the crayfish, *Procambarus clarkii*, as revealed by the work of numerous investigators, and then focus on recent advances in the analysis of the escape response, made primarily in F. B. Krasne's laboratory and in my own. A key concept that now guides our work is that arthropod nervous systems and behaviour have evolved, in large part, by the gradual modification of individual body segments and that much can be learned by intersegmental comparisons. (For a brief summary of studies along these same lines see Miller, Hagiwara & Wine, 1984.)

MAJOR STRUCTURAL FEATURES

Gross anatomy

Anatomically, the crayfish abdomen is simply the posterior portion of the body, and as such contains the posterior homologues of gut, axial muscles and appendages (Fig. 1). Functionally, however, the abdomen is analogous to an appendage, and for this

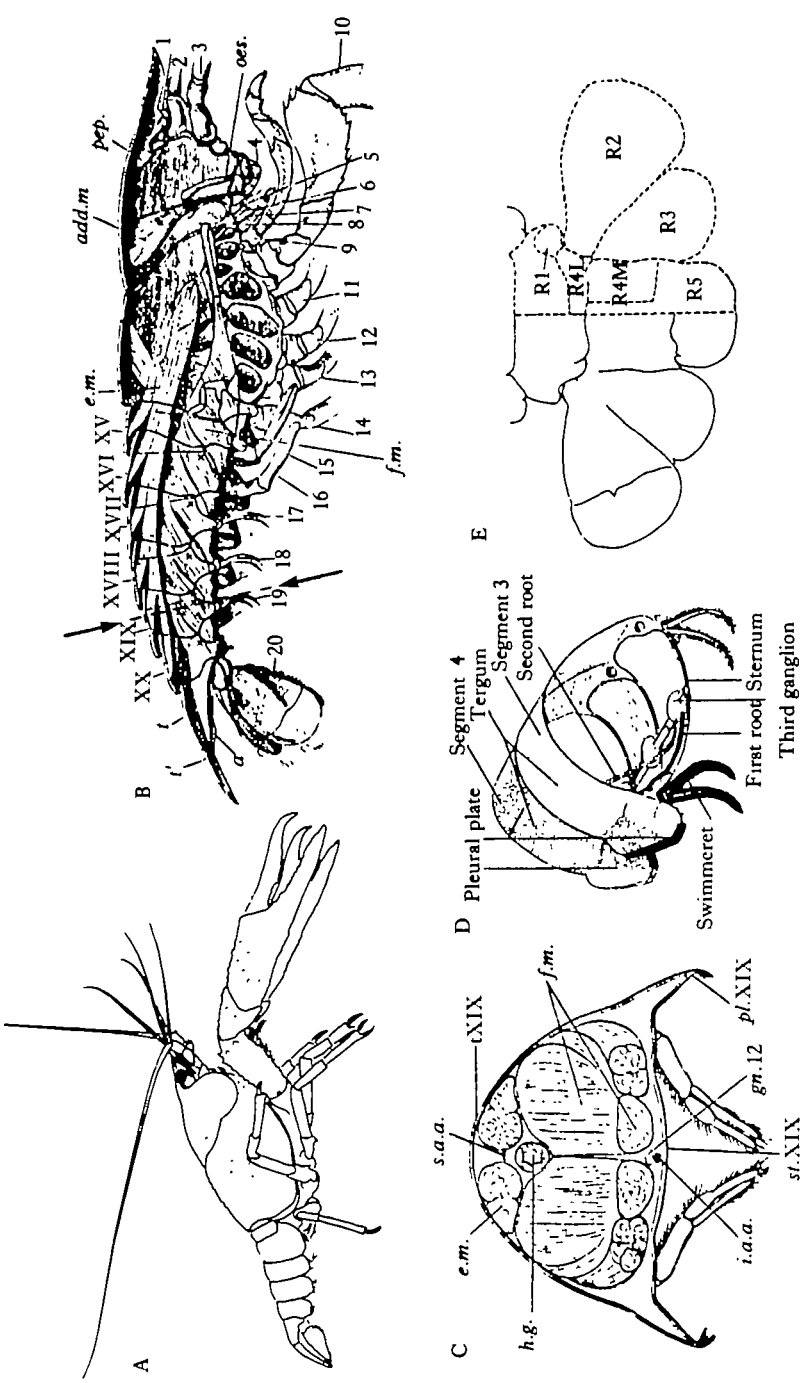


Fig. 1. Major anatomical features of the crayfish abdomen. (A) Alert, adult crayfish with extended abdomen. (B) Sagittal section (Huxley, 1880). Roman numerals refer to body segment and arabic numerals to appendages. *a*, vent; *add.m.*, adductor muscle of mandible; *pep.*, procephalic process; *oes.*, oesophagus; *t* and *t'*, two segments of telson. (C) Cross section of the 5th abdominal segment (arrows on B). *e.m.*, phasic extensor muscles; *s.a.a.*, superior abdominal artery; *pl.* XIX, 19th tergum; *h.g.*, hindgut; *f.m.*, flexor muscles; *gn.* 12, 12th ganglion (5th abdominal ganglion); *i.a.a.*, inferior abdominal artery (ventral artery); *st.*, stergite; *t.*, tergum; (Huxley, 1880). (D) Sensory fields of 1st and 2nd roots of the 3rd abdominal ganglion (Wiersma & Hughes, 1961). (E) Innervation of the tailfan. Each number is the sensory receptive field of that root of the terminal ganglion. R6 is purely motor and R7 innervates the intestine (Calabrese, 1976).

reason is often referred to as the tail. Crayfish have two distinct modes of locomotion, both of which involve the abdomen. During walking, which in adults constitutes more than 99% of the time spent in motion, the extended abdomen (Fig. 1A) is used for balance. It can also help steer the animal, by acting like a rudder, while the pitch and stroke frequency of the swimmerets can be varied to correct rolls and to aid in yawing movements (Davis, 1968). In backward walking, especially up an inclined plane, the curled tail is rhythmically applied to the substrate to help the animal move (Kovac, 1974). The abdomen has five similar segments and two terminal ones which have been modified to form the tailfan. Each anterior segment contains a massive set of axial phasic flexor and extensor muscles, which control rapid abdominal tailflips, and a superficial set of tonic muscles which control posture (Fig. 1B,C). Each anterior segment also contains a pair of appendages called swimmerets that are homologous with the walking legs (Fig. 1C,D). These are the exclusive motor structures in those ganglia.

In the 6th abdominal segment, the endopodite and exopodite of the swimmerets are expanded into the uropods; a 7th segment has become the telson. Together, these structures form the tailfan (Fig. 1E). The alterations in the exoskeleton of the terminal segment have had important consequences for both the muscles and sensory structures. The greatly enlarged surface area of the tailfan is covered with sensory hairs (and presumably functions as a sensory organ), although it is not as specialized in this regard as are the cerci of some insects. The muscles have also been considerably modified. Comparative work on the telson muscles and their innervation is being carried out within (e.g. Dumont & Wine, 1983) and among species (Paul, 1981).

The abdominal nervous system

The central nervous system

Each anterior segment is controlled by a single ganglion (Fig. 2A) which communicates with neighbouring ganglia *via* paired connectives and with the periphery *via* three paired nerves or roots. The most anterior (or 1st) is a mixed sensory/motor nerve that mainly innervates the swimmerets; the 2nd nerve is also mixed and innervates the extensors and the remaining surface of the segment. The most caudal, or 3rd nerve, is the purely motor nerve to the flexor muscles (not shown), which is cleanly split into tonic and phasic branches.

The supracellular organization of a middle abdominal ganglion was studied by Skinner (1984*a,b*) based on serial sections of plastic embedded material. She provides the following general picture (Fig. 2). All cell bodies are located in the ventral rind, dorsal to which are the neuropile, connective axon tracts and commissures. Twelve axon tracts and four commissures are discernible; all but three tracts and one commissure are named for similar and possibly homologous features seen in orthopteran insects (Gregory, 1974; Pipa, Cook & Richards, 1959). Five neuropile regions are also named. The *horseshoe neuropile* is largest and most ventral, and is named for its appearance in the horizontal plane; the open part of the horseshoe is anterior. The *lateral neuropiles* are next largest and form large bulges near the 1st and 2nd roots. The *anterior* and *posterior midline neuropiles* are very small, ventral regions; the remaining interstices between tracts and commissures are filled with *tract neuropile*. In the light microscope, each neuropile region (except the two midline neuropiles

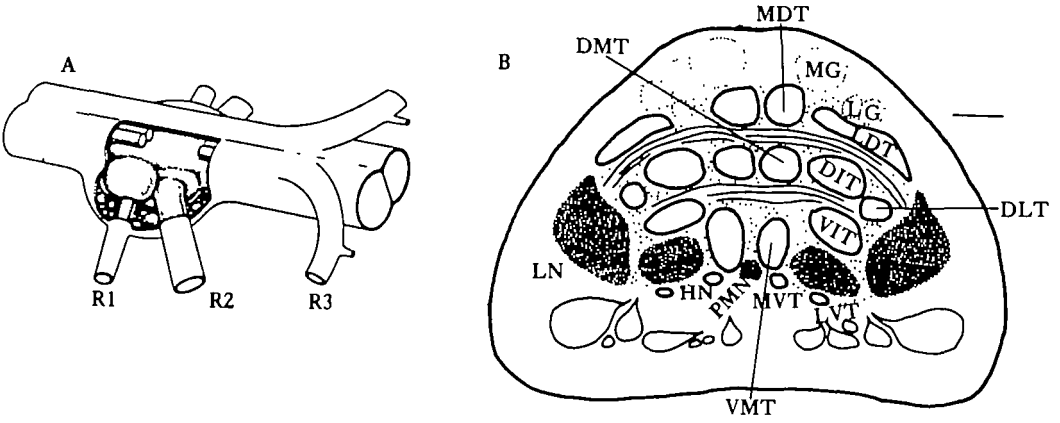


Fig. 2. (A) Major nerves of an abdominal ganglion. (B) Supracellular organization of the 4th abdominal ganglion. This is a schematic diagram of a mid-ganglion transverse section showing the two commissures, nine major through-tracts and three neuropile areas present at this level (from Skinner, 1984*a*,*b*). DIT, dorsal intermediate tract; DMT, dorsal median tract; DLT, dorsal lateral tract; HN, horseshoe neuropile; LDT, lateral dorsal tract; LG, lateral giant axon; LN, lateral neuropile; LVT, lateral ventral tract; MDT, medial dorsal tract; MG, medial giant axon; MVT, medial ventral tract; PMN, posterior midline neuropile; VIT, ventral intermediate tract; VMT, ventral medial tract.

which are not differentiated from one another) displays a characteristic texture which can be resolved by electron microscopy to differences in the size and distribution of profiles, differences in concentration of dense core vesicles and certain inclusion bodies, and whether or not a glomerular organization is present.

Each anterior abdominal ganglion contains the cell bodies of approximately 300 pairs of neurones plus a small number of unpaired cells. Cobalt backfill studies (unpublished) show that approximately 100 of the neurones are efferents, 100 others project axons into the connectives, and the remaining 100 are local interneurones. In future work, it will be important to place individually identified neurones in the context of the newly defined, supracellular organization. That will be informative in two ways: it will indicate the extent to which tracts and neuropile regions have functional significance, and should provide insight into the types of synaptic interactions we can expect from a given neurone.

The last abdominal ganglion is a fusion product of two ganglia (Fig. 3C,D), with a cluster of neurones near the posterior intestinal nerve that innervate the gut. (The gut neurones have no obvious homologues in anterior ganglia, J. P. C. Dumont, personal communication.) Despite fusion, the terminal ganglion contains about the same number of neurones as a middle ganglion, perhaps because some descending interneurones and the efferents to one set of appendages have been lost. In addition, any remaining neurones that would have projected to the next anterior or posterior segment are classed here as intraganglionic interneurones.

Differences sometimes occur in the numbers of homologous neurones found in different ganglia (e.g. Mittenthal & Wine, 1978). It has been shown in grasshoppers that such differences can arise either because a homologue develops a sufficiently different structure to be no longer recognizable or because the cell is deleted (Bate, Goodman & Spitzer, 1981).

More than 600 neurones in the abdominal nervous system are identified, although

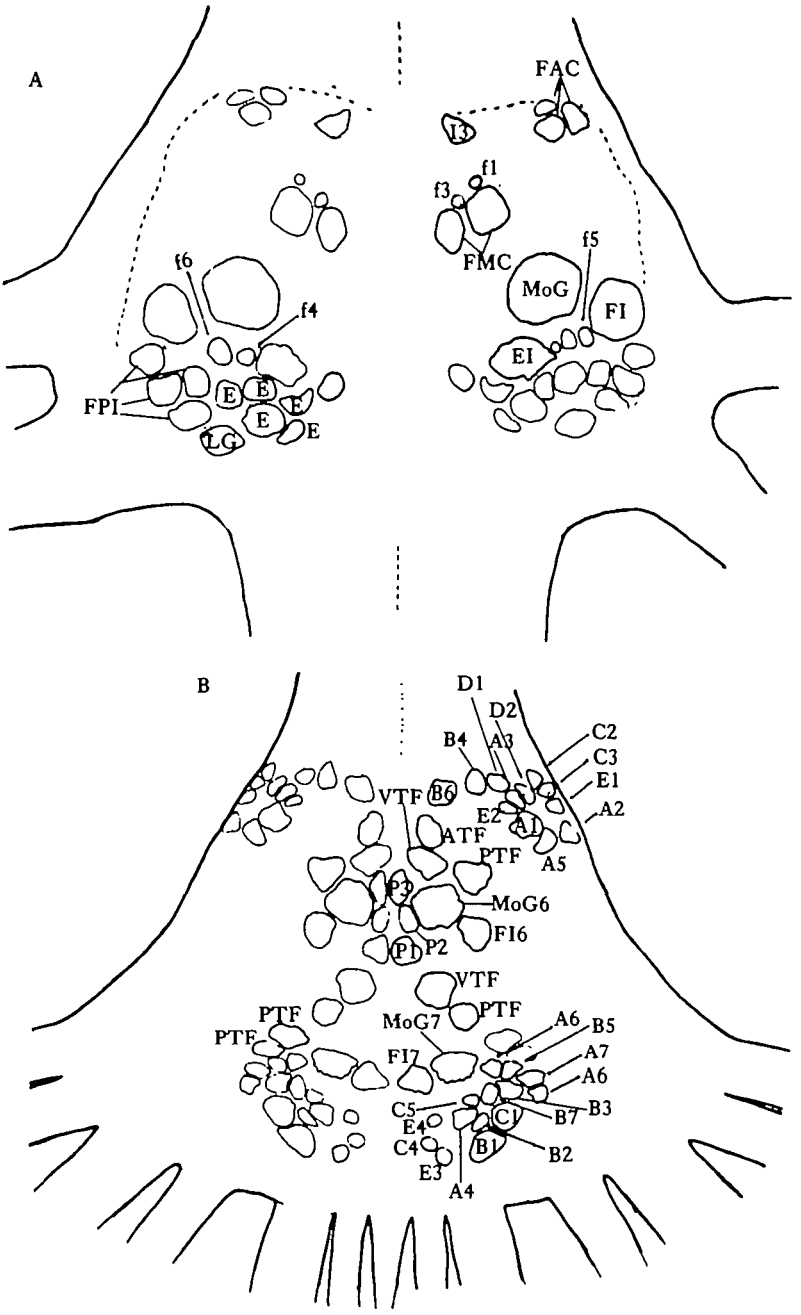
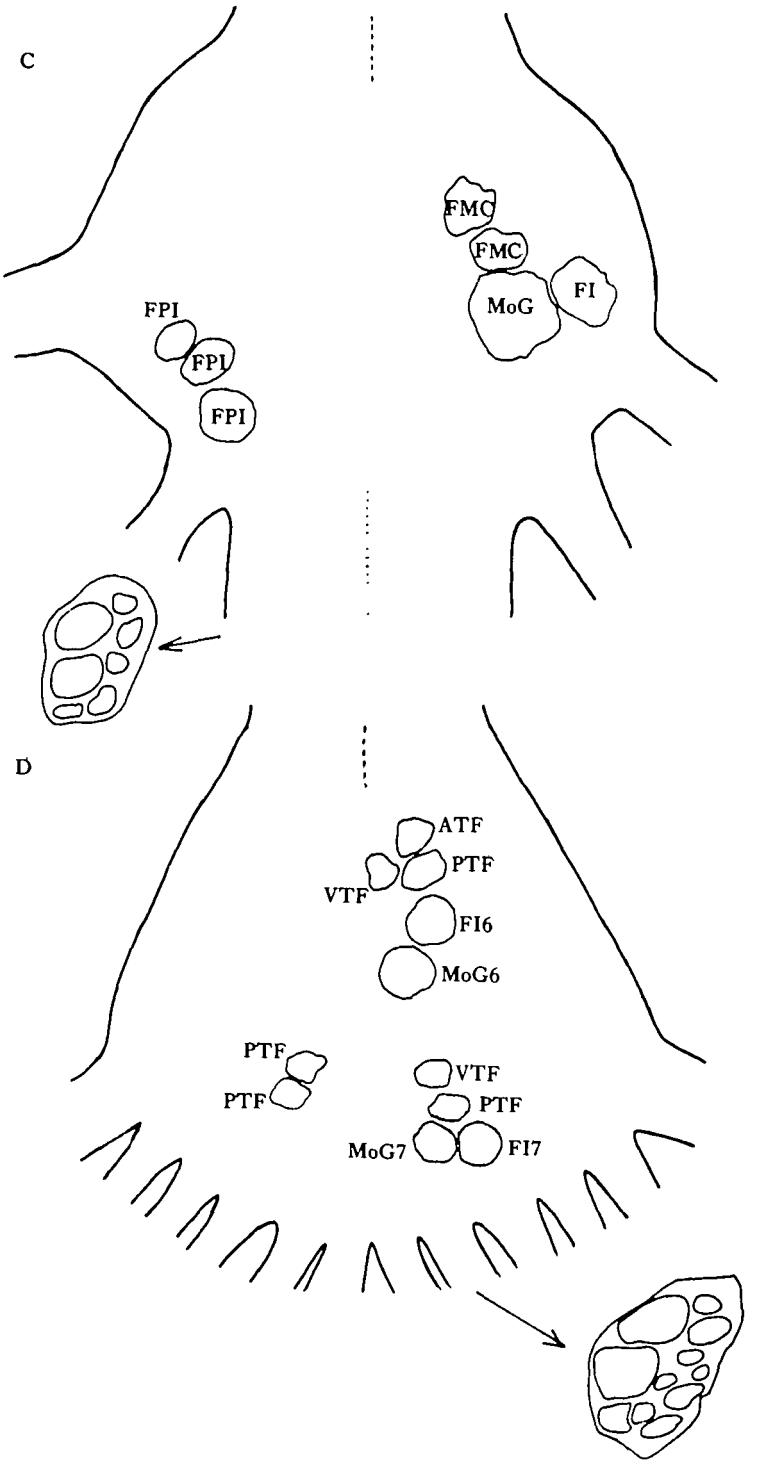


Fig. 3. (A),(B) Soma maps (somewhat idealized) of the 3rd and 6th ganglia. Positions of identified cell bodies were determined by toluidine blue staining, cobalt backfills and Lucifer Yellow injections. For abbreviations, see Table 1. (C),(D) Hypothesized homologies between fast flexor and telson flexor efferents. The 5th ganglion contains seven fast flexor efferents; the 6th ganglion contains eleven. Since the distinctive motor giant and flexor inhibitor homologues are doubled in the terminal ganglion and are found in separate clusters, we propose that the adjacent FMC homologues are also doubled, whereas the FPI cluster has been reduced to just two neurones. The 'anterior telson flexor' is thought not to be homologous to any fast flexor (from Dumont & Wine, 1983 and in preparation).


C
FMC
FMC
MoG
FI
FPI
FPI
FPI
D
ATF
PTF
VTF
FI6
MoG6
PTF
PTF
VTF
PTF
MoG7
FI7

in some cases the identifications are not unique but instead consign the cell to a group of several cells which cannot be distinguished except by their peripheral terminations, which are rarely mapped. Identified neurones are listed in Table 1. Only a few motor pools are completely mapped: about 90% of the cells in the abdominal CNS are unidentified; for interneurones of the anterior abdominal ganglia the figure is more than 95% unidentified.

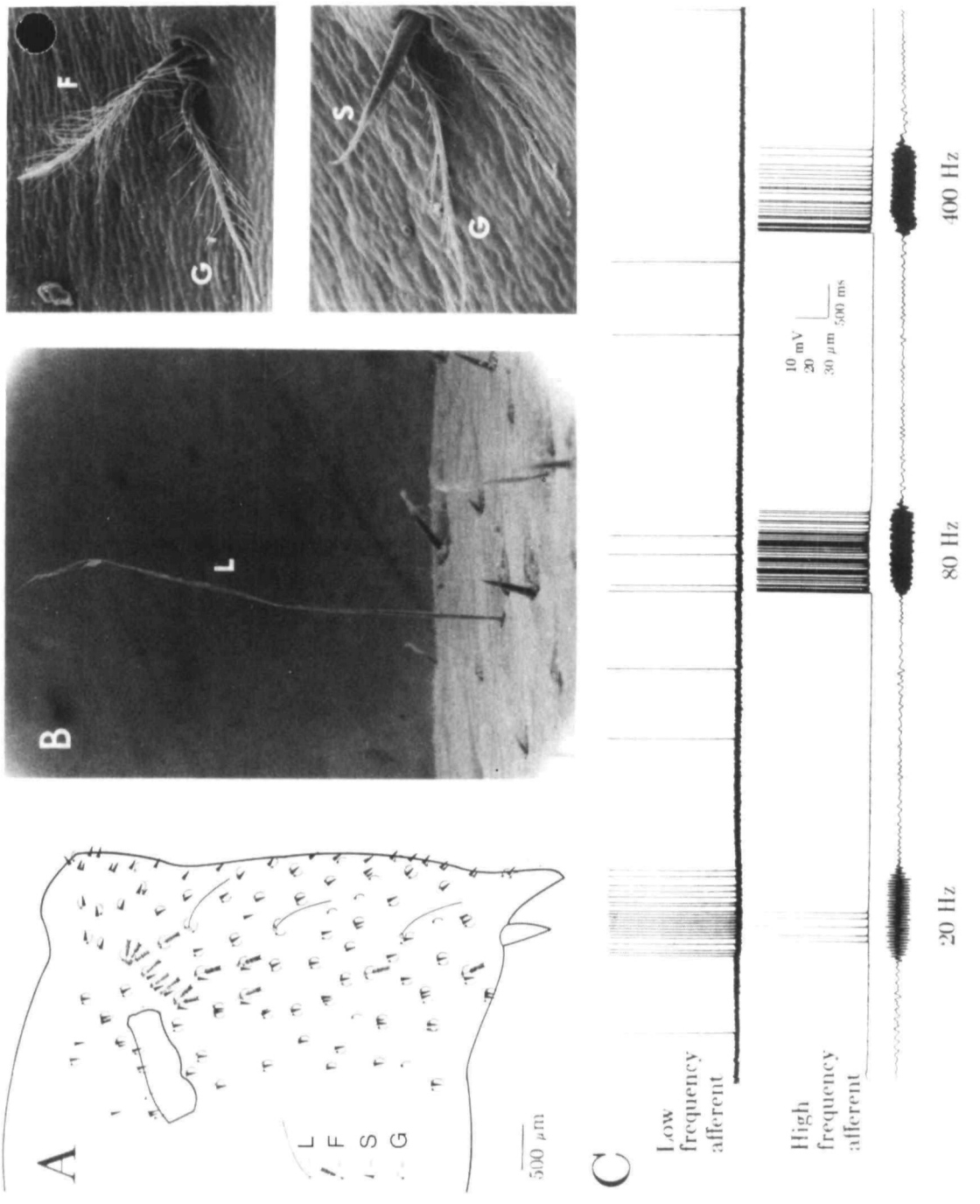
Composite soma maps of identified neurones in the 3rd and 6th ganglia are shown in Fig. 3. Because the identified cells tend to be the largest cells in the ganglion, the maps give an exaggerated impression of the proportion of neurones that have been identified. The problem of studying integrative mechanisms at the unit level in a ganglion of this size is greatly understated by counting only cells that originate in the ganglion. Integration occurs in the neuropile, which contains terminals from interneurones and afferents that outnumber the neurones originating in the ganglion by at least ten to one. Some projecting interneurones cause special complications since they have input and output sites in several ganglia (Hughes & Wiersma, 1960) and can participate in integration for weeks even though they lack their cell body (Krasne & Lee, 1977). Another potential complication is the claim that significant post-hatching cell addition occurs in crayfish central ganglia (Roth & Suppes, 1973). Since preliminary evidence suggests that all large efferents and interneurones are present shortly after hatching (e.g. Kennedy, 1974), cell addition may occur preferentially in local interneurones.

Neurotransmission within the abdominal CNS is beyond the scope of this review. However, some interesting new findings are that (1) proctolin elicits swimmeret beating when perfused through the ventral artery (B. Mulloney, personal communication); and (2) octopamine and serotonin are probably present (Kravitz *et al.* 1976; Eloffson, 1983; Beltz & Kravitz, 1983), and have opposite effects (serotonin inhibits and octopamine excites) on the lateral giant escape response (Glanzman & Krasne, 1983).

The peripheral nervous system

Mechanosensory. The cell bodies of all exteroceptors and most proprioceptors are located in the periphery. The only known mechanoreceptors with cell bodies in the abdominal *central* nervous system are the non-spiking stretch receptors described by Heitler (1982), although the cord stretch receptors (Wiersma & Hughes, 1961; Grobstein, 1973) also presumably have central cell bodies. The most common exteroceptive mechanoreceptors are the hair cells. These cells are typically dually innervated and some of them are directionally sensitive to waterborne stimuli (Wiese, 1976). Four types of hairs have been found on the telson, some types in reproducibly

Fig. 4. Properties and distribution of mechanosensory hairs on the crayfish telson. (A) Idealized map, based on surveys of 20 individual animals, showing numbers and locations of four hair types on the telson. The three long hairs are individually identifiable. Scale bar, 500 μm. (B) Scanning electron micrographs showing each type of hair (L, long hair, 85×; F, feathered hair; S, smooth hair; G, guard hair, 400×). (C) Responses of two types of afferents to various frequencies. Low frequency afferents continue to fire at low threshold to a frequency of 0·2 Hz. Tests were performed with the tailfan in a sealed chamber having rigidly coupled diaphragms at each end. This causes the entire column of water to move at the imposed frequency and minimizes harmonics and echoes (from Plummer, Dumont & Wine, 1983).


A
L
F
S
G
500 μm
B
L
F
G
S
G
C
Low frequency afferent
High frequency afferent
10 mV
20
30 μm
500 ms
20 Hz
80 Hz
400 Hz

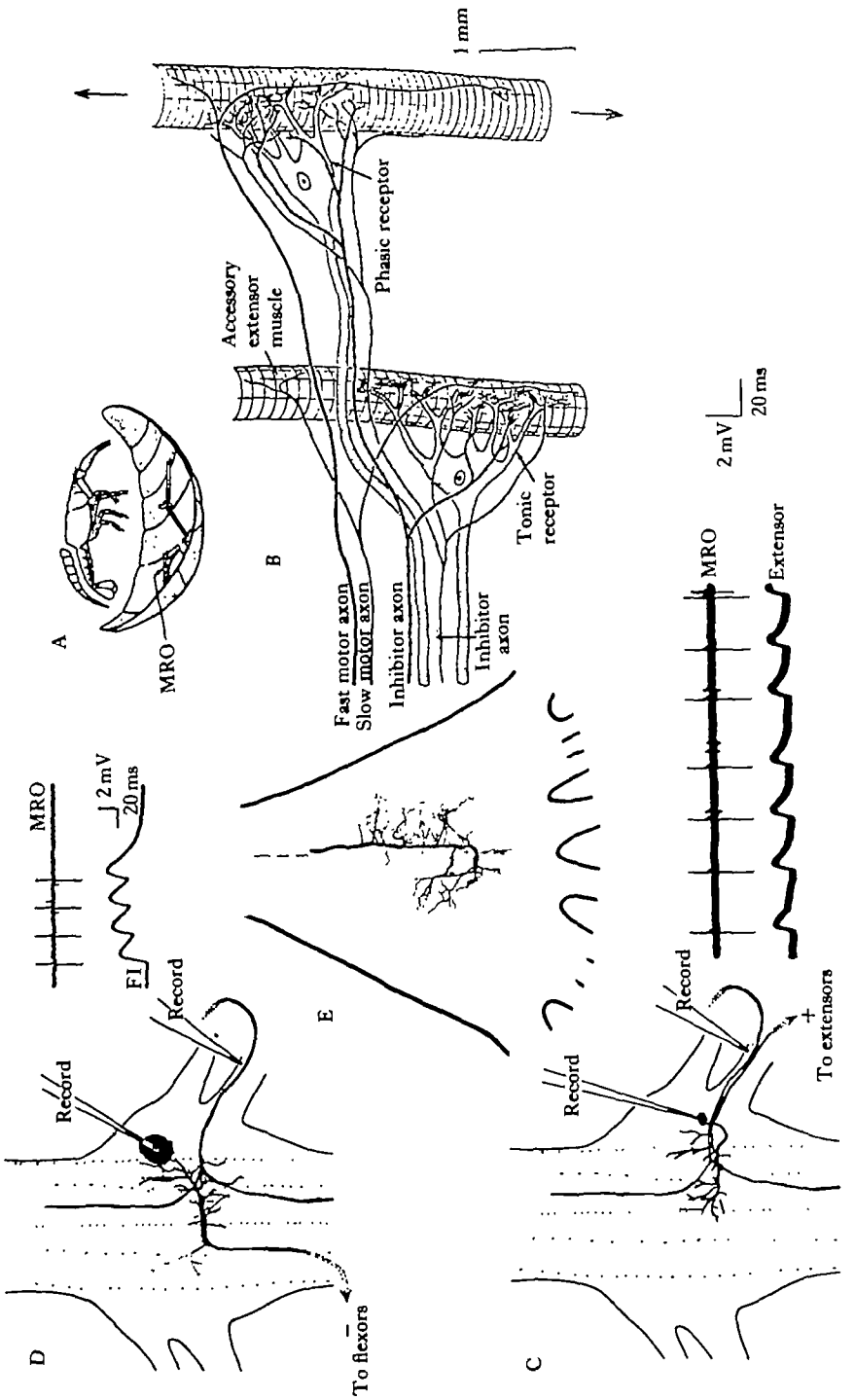


Fig. 5. The muscle receptor organs and their central connections. (A) The muscle receptor organs (MRO) are located under the dorsal carapace of the abdomen. Each MRO spans the joint between two adjacent segments. (B) Each side of a segment contains a tonic and a phasic MRO. Reflex pathways for both kinds of MROs have been described (Eckert, 1961; Jansen, Nja & Walløe, 1970; Barker, Herbert, Hildebrand & Kravitz, 1972). (C) The tonic MRO excites fast extensor motor neurones. Tonic activity due to stretch of the tonic MRO (top trace) causes 1:1 EPSPs in the extensor motor neurone (Extensor; soma recording). (D) The tonic MRO also excites the peripheral inhibitor to the fast flexor muscles (FI; *soma trace*). (E) Termination of MRO axon in the 6th abdominal ganglion. (A,B from Bullock & Horridge, 1965; C,D from Wine, 1977*b*; E from Bastiani, 1981.)

characteristic positions (Fig. 4A,B). Hair receptors on the telson are also frequency selective: receptors have been found which respond well to water movements as slow as 0·2 Hz and have a fairly flat frequency threshold curve, while others appear to be acceleration sensitive and have an optimal response at about 80 Hz (Plummer, Dumont & Wine, 1983, and Fig. 4C). The receptors which respond to very low frequencies are especially interesting because their spontaneous firing is suppressed by high frequency stimulation. This suppression could be achieved by efferent inhibition, mechanical uncoupling, or summation of hyperpolarizations (either endogenous or mediated by peripheral, interafferent inhibition) that might be produced by antiphase movements. Preliminary correlations of physiologically characterized receptors with sensory hair types reveal that long hairs and feathered hairs (Fig. 4B) are innervated by low-pass receptors.

The muscle receptor organs (MROs) are the most thoroughly studied proprioceptors and four central effects have been documented (Fig. 5), all of which act to silence the MROs (negative feedback). So far, it has been documented that the MROs excite their own peripheral inhibitors (Eckert, 1961), the peripheral inhibitors of phasic flexors (Wine, 1977*b*), and both phasic and tonic extensor motor neurones (Fields, 1966; Wine, 1977*b*). The entire central course of the muscle receptor axons has been worked out by Bastiani (1981). Tonic and phasic axons have an overlapping distribution and are indistinguishable on structural grounds. The MRO terminals in the 6th abdominal ganglion make characteristic 'J' structures and innervate numerous cells bilaterally (Fig. 5E; Bastiani, 1981).

All known receptors in the abdomen are listed in Table 1. Many types of receptors have almost certainly been missed; receptor hunting is somewhat out of fashion, and for the few active experts the specialized appendages offer richer grounds, since it seems unlikely that new types of receptors will be found in the abdomen. However, it will be useful if receptor types found elsewhere are also found in the abdomen, since it will be easier in the abdomen than elsewhere to trace their central connections. So far as is known, all receptors are cholinergic (Barker, Herbert, Hildebrand & Kravitz, 1972). Orthograde degeneration is rapid for all receptor axons tested (Bittner & Johnson, 1974) with the sole exception of the muscle receptors, whose isolated axons persist for many weeks (Bittner, 1977).

Axial muscles and motor neurones. Almost all axial abdominal motor neurones have been identified (Table 1). Motor neurones have multiterminal endings on many muscle fibres, and the motor fields overlap so that each fibre is polyinnervated. All axial muscle fibres also receive peripheral inhibition. Neuromuscular synapses are typically stable or facilitating. An exception is the depression-prone neuromuscular synapse of the flexor motor giant (Bruner & Kennedy, 1970). The excitatory transmitter of the motor neurones is probably glutamate (Takeuchi & Takeuchi, 1974); the inhibitory transmitter (in lobsters) is gamma-aminobutyric acid (GABA) (Otsuka, Kravitz & Potter, 1967). Evidence is accumulating that the pentapeptide proctolin is co-released by three of the five tonic flexor motor neurones (Bishop, Wine & O'Shea, 1984). As in insects (Adams & O'Shea, 1983) proctolin appears not to affect the amplitude of synaptic potentials; unlike in insects it appears to enhance tension amplitude without affecting tension duration (C. A. Bishop & F. Nagy, personal communication).

Table 1. *Identified neurones in the crayfish abdominal nervous system*

When name is followed by [P] it indicates that a population of cells is being designated. Individual cells within the population are not discriminable unless noted. References are to earliest known citation or to most comprehensive citation. The figure number indicates that the cell is shown in this paper. The column 'CELLS' gives total number of such cells in the abdominal nervous system. It includes pairing and serial homologues. This list does not include interneurones identified by Wiersma on the basis of receptive field and axon position. For those, see Wiersma & Hughes (1961) and Wiersma & Bush (1963).

NAME	DESCRIPTION	CELLS	REFERENCES
AFFERENTS			
MRO1 (SR1)	Abdominal stretch receptors, slow or tonic, 1 per hemisegment, root 1	10	Fig. 5 Fields, 1966
MRO2 (SR2)	Abdominal stretch receptors, fast or phasic, 1 per hemisegment, root 2	10	Fig. 5 Fields, 1966
LF1	Long feathered hair, telson, most anterior in root 4 field	4	Fig. 4 Plummer, Dumont & Wine, 1983
LF2	Long feathered hair, telson, middle of root 4 field	4	Fig. 4 Plummer *et al.* 1983
LF3	As above, most posterior in root 4 field	4	Fig. 4 Plummer *et al.* 1983
Pabst [P]	Population of multipolar receptors with cell bodies in the 2nd roots. Probably are proprioceptors for the tonic flexors, they respond to gentle distortion of the soft cuticle near the insertion of the tonic flexors		Pabst & Kennedy, 1967
CSR1	Cord stretch receptors, tonic. One per hemisegment, respond to elongation of cord sheath. Known only by effect and by axon position	10	Grobstein, 1973
CSR2	As above, phasic	10	Grobstein, 1973
FH [P]	Feathered hair. Dorsal tergum and tailfan. Dually innervated with directionally selective afferents. In 4th root field there are about 15 of these; respond preferentially to low frequency water movements	Many	Fig. 4 Weise, 1976
SH [P]	Smooth hairs. Most numerous of hair type. Many receptors dually innervated, responsive to touch and high frequency water movement, outnumber feathered hairs more than three to one		Fig. 4 Plummer *et al.* 1983
(CPR)	Caudal photoreceptor. Classified as an interneurone. See 6A1 below		Fig. 8 (see below)
NSSR–A	Non-spiking stretch receptors, axon in ipsilateral root 1, anterior soma, G1–G5	10	Heitler, 1982
NSSR–P	As above, posterior soma, G1–G5	10	Heitler, 1982
INTERNEURONES (Command and premotor)			
LG	Lateral giant. Command neurone for pitch forward escape response. Actually a network of 14 interconnected segments within the abdomen	14	Fig. 11 Wine & Krasne, 1982
MG	Medial giant. Command neurone for dart backward escape. Exists only as paired axons in abdomen, all inputs are to dendrites in brain	2	Fig. 11 Wine & Krasne, 1982
I2	Premotor cell in flexion circuit with soma in G2 and dorsal axon with terminals in G6. Fired by giant axons and sometimes active during non-giant escape	2	Fig. 13 Kramer, Krasne & Wine, 1981*b*

Table 1. *Continued*

NAME	DESCRIPTION	CELLS	REFERENCES
I3	As above except soma in G3, different outputs in G6	2	Fig. 13. Kramer *et al.* 1981*b*
SG	Segmental giant. One per hemisegment, classed as an interneurone even though axon in 1st root (destination not known). Fired by giant axons and in turn fires all fast flexor motor neurones on same side as well as other cells. Can be thought of as output branches of giant axons	12	Fig. 13 Roberts *et al.* 1982
Type 1	Premotor slow flexor, descending axon. Found in G2 and G4	4	Miall & Larimer, 1982*b*
Type 2	Premotor slow flexor, descending axon, G4	2	,,
Type 3	Premotor slow flexor, ascending axon, G4	2	,,
Type 4	Premotor slow flexor, bifurcating axon, G4	2	,,
Type 5	Premotor slow flexor, descending axon, G4	2	,,
Type 6	Premotor slow flexor, bifurcating axon, found in G3–G5	6	,,
Ext(4)	Premotor slow extensor, G5	2	,,
fi(1)	Inhibitor of slow flexors, G4	2	,,
A/B	Premotor slow flexor, bifurcating axon, G3	2	Larimer & Jellies, 1983; their Fig. 5
C/D	Premotor slow flexor, bifurcating axon, found in G2 and G3	4	,,
E/F	Premotor slow flexor, ascending axon, G5	2	,,
G/H	Premotor slow flexor, bifurcating axon, G5	2	,,
L&J, Fig. 4	Premotor slow flexor, ascending axon, G3	2	,,
INTERNEURONES (Sensory, projecting)			
6A1	Slosh interneurone, same as CPR, bidirectional, soma in G6	2	Fig. 8 Wilkens & Larimer, 1972
6A2	Slosh interneurone, tailward, G6	2	Fig. 8
6A3	Slosh, tailward, G6	2	Fig. 8 Sigvardt, Hagiwara & Wine, 1982
6A4	Slosh, headward, G6	2	,,
6A5	Slosh, tailward, G6	2	,,
6A6	Slosh, headward, G6	2	Fig. 8
6A7	Slosh, tailward, G6	2	,,
6B1	Touch, G6, ('interneurone A')	2	Fig. 8 Zucker, 1972
6B2	Touch, G6 (K/L, premotor slow flexor)	2	Fig. 8 & Larimer & Jellies, 1983
6B3	Touch, G6 (I/J, premotor slow flexor)	2	Fig. 8 Sigvardt *et al.* 1982 & Larimer & Jellies, 1983
6B4	Touch, G6	2	Fig. 8
6B5	Touch, G6	2	Fig. 8 Sigvardt *et al.* 1982
6B6	Touch, G6	2	Fig. 8
6B7	Touch, G6	2	Fig. 8
6C1	Touch, G6, ('interneurone C')	2	Fig. 8 Zucker, 1972

Table 1. *Continued*

NAME	DESCRIPTION	CELLS	REFERENCES
6C2	Touch, G6	2	Fig. 8 Sigvardt *et al.* 1982
6C3	Touch, G6	2	Sigvardt *et al.* 1982
6C4	Touch, G6	2	,,
6C5	Touch, G6	2	,,
A13	Touch, G5	2	Wiersma & Hughes, 1961
6D1	Proprioception, G6	2	Fig. 8 Sigvardt *et al.* 1982
6D2	Proprioception, G6	2	,,
6E1	Proprioception, G6	2	,,
6E2	Proprioception, G6	2	,,
6E3	Proprioception, G6	2	,,
6E4	Proprioception, G6	2	,,
6P1	Pinch, G6	2	,,
6P2	Pinch, G6	2	,,
6P3	Pinch, G6	2	Fig. 8
5P1	Pinch, G5	2	Unpublished
INTERNEURONES (Local, in G6 unless otherwise noted)			
LDS	Non-spiking commissural interneurone that inhibits 6A1 and 6A4 on one side when it is stimulated with low frequency water movements on other side	2	Fig. 9 Reichert, Plummer & Wine, 1983*a*
6PM–1	Non-spiking, excites units innervating endopodite	2	Reichert *et al.* 1982
6PM–2	Non-spiking, inhibits units innervating endopodite	2	,,
6PM–3	Non-spiking, inhibits some and excites other units innervating exopodite, G2	2	,,
6PM–4	Non-spiking, inhibits units innervating exopodite	2	,,
6PM–5	As above	2	,,
6PM–6	Touch, spiking	2	,,
6PM–7	Touch, spiking	2	,,
6S–1	Touch, spiking	2	,,
6S–2	Touch, spiking	2	,,
6S–3	Touch, spiking	2	,,
6S–4	Proprioception, spiking	2	,,
6S–5	Touch, spiking	2	,,
6X	Touch, spiking	2	,,
PADI–1	Inhibitor of primary afferents	2	Kirk & Wine, 1983
PADI–2	Inhibitor of primary afferents	2	,,
SC	Semi-to-b cell, inhibits MoGs	2	Fig. 13
SPI(2)	Non-spiking, reciprocal effects on slow flexors and extensors, G2 and G3	8	M. R. Plummer & J. J. Wine, in preparation
INTERNEURONES (Other)			
MoGI–1	Inhibitor of all MoGs, fire by FFs	2	Wine, 1977*a*
PADI–3 [P]	Inhibitor of primary afferents, G6	2	Kirk & Wine, 1983
C/D	Bilateral, ascending axons, G3 and G5	4	Larimer & Jellies, 1983

Table 1. *Continued*

NAME	DESCRIPTION	CELLS	REFERENCES
MOTOR NEURONES			
MoG	Motor giant, one per hemisegment, excites fast flexor muscles, fired by lateral and medial giants, G1–G5	10	Fig. 3 Furshpan & Potter, 1959
FMC1	Fast flexor, one per hemisegment, axon in contralateral root 3, fired by SG, G1–G5	10	Fig. 3 Wine & Mistick, 1977; Mittenthal & Wine, 1978
FMC2	Fast flexor, as above	10	,,
FAC1	Fast flexor, one each in G1–G4, axon exits in anterior contralateral root 3	8	,,
FAC2	As above, but only in G2–G4	6	,,
FAC3	As above, but only in G2 and G3	4	,,
FPI(4)	Fast flexors, four in each hemiganglion except G5 which has three and G6 (see below), axon exits ipsilateral root 3	38	,,
FI	Peripheral inhibitor of fast flexor muscles, one per hemisegment, G1–G5	10	,,
ATF	Anterior telson flexor. G6, axon in R6, fired by LGs	2	Fig. 3 Dumont & Wine, 1983
MoG6	Homologue of MoG that excites posterior telson flexors, G6	2	,,
MoG7	Homologue of MoG that excites ventral telson flexors, G6	2	,,
PTF(2)	Posterior telson flexor motor neurone, ipsilateral axon in root, G6	4	,,
PTF(2)	As above, but axon in contralateral root 6, G6	4	,,
VTF(2)	Ventral telson flexor motor neurone, contralateral axon in root 6, G6	4	,,
FI6	Peripheral inhibitor to VTF and dorsal as well as ventral telson flexor muscles, G6	2	,,
FI7	Peripheral inhibitor to ventral part of PTF as well as VTF muscles, G6	2	,,
Red MN1	Reductor exopodite excitor, axon in root 2, G6	2	Nagayama, Takahata & Hisada, 1983
Red MN2	As above	2	,,
Red MN3	As above	2	,,
Add MN(2)	Adductor exopodite excitor, axon in root 2, G6	4	,,
f1	Slow flexor motor neurone, axon in contralateral anterior superficial root 3, G1–G5	10	Wine, Mittenthal & Kennedy, 1974
f2	Slow flexor motor neurones, axon in ipsilateral superficial root 3, G1–G5	10	,,
f3	As above, contralateral axon, G1–G5	10	,,
f4	As above, ipsilateral axon, G1–G5	10	,,
f5	Peripheral inhibitor to slow flexor muscles, G1–G5	10	,,
f6	Slow flexor motor neurone, ipsilateral axon, G1–G5	10	,,
E(5)	Fast extensor motor neurones, axon exits ipsilateral root 2, G1–G5	50	Wine & Hagiwara, 1977

Table 1. *Continued*

NAME	DESCRIPTION	CELLS	REFERENCES
EI	Peripheral inhibitor of fast extensor muscles, G1–G5	10	Wine & Hagiwara, 1977
e(5)	Slow extensor motor neurones, G1–G5	50	Kennedy, Evoy & Fields, 1966
eI	Peripheral inhibitor to slow extensor muscles, G1–G5	10	,,
Acc	Accessory motor neurones innervating muscle receptor organs. Four per ganglion, G1–G5	40	Fig. 5 Eckert, 1961
OTHER CELLS			
UR7	Unpaired cells, one per ganglion, with axons in root 7	7	Unpublished
G6R	Small cells, spiking cell bodies and axons in root 7	50	Unpublished
G1S	Serotonin-containing cell	2	Beltz & Kravitz, 1983
R1, R2	Neutral-red staining, vesicle-rich neurosecretory cells with axons in anterior connective, two each G1–G5, >4 in G6	28	R. L. Roth, unpublished

Muscles and motor neurones of appendages. Less is known about the innervation of the swimmerets and their homologues, the uropods. A cell count based on extracellular nerve recordings and intramuscular recordings has been made for the uropods (Larimer & Kennedy, 1969), which indicates that at least 27 motor neurones and up to 5 peripheral inhibitors are involved. Motor pools were visualized with cobalt backfills (Reichert, Plummer & Wine, 1983*b*); these revealed about 42 central cells with axons in the 2nd and 3rd roots of the 6th ganglion, but some of those might be afferents. Cell by cell identification of efferents to the appendages are now being carried out by W. J. Heitler, M. Hisada and B. Mulloney (personal comments). For an extensive review of crustacean neuromuscular systems see Govind & Atwood (1982).

OUTLINE OF A SENSORY SYSTEM

Our understanding of the processing of sensory information by the crayfish abdominal nervous system is still at a preliminary stage. Although much is known about mechanosensory receptors and primary sensory interneurones, most (and perhaps all) behavioural systems require converging input from many sensory interneurones before they are activated, and this has made it difficult to trace information through the nervous system. In the isolated abdomen, long sensory-motor pathways that pass through the rostral nervous system are eliminated, and the focus is on shorter pathways that engage the abdominal motor systems directly. Even so, only one behavioural system, the LG-mediated escape system, has been traced from receptors to muscles, and even that system is understood only in outline (Fig. 6). In an attempt to quantify the convergence required to activate motor systems, we have been using intracellular techniques to do a cell by cell study of interganglionic sensory interneurones, many of which Wiersma and his colleagues had previously catalogued with extracellular methods (Hughes & Wiersma, 1960; Wiersma & Hughes, 1961; Wiersma & Bush, 1963).

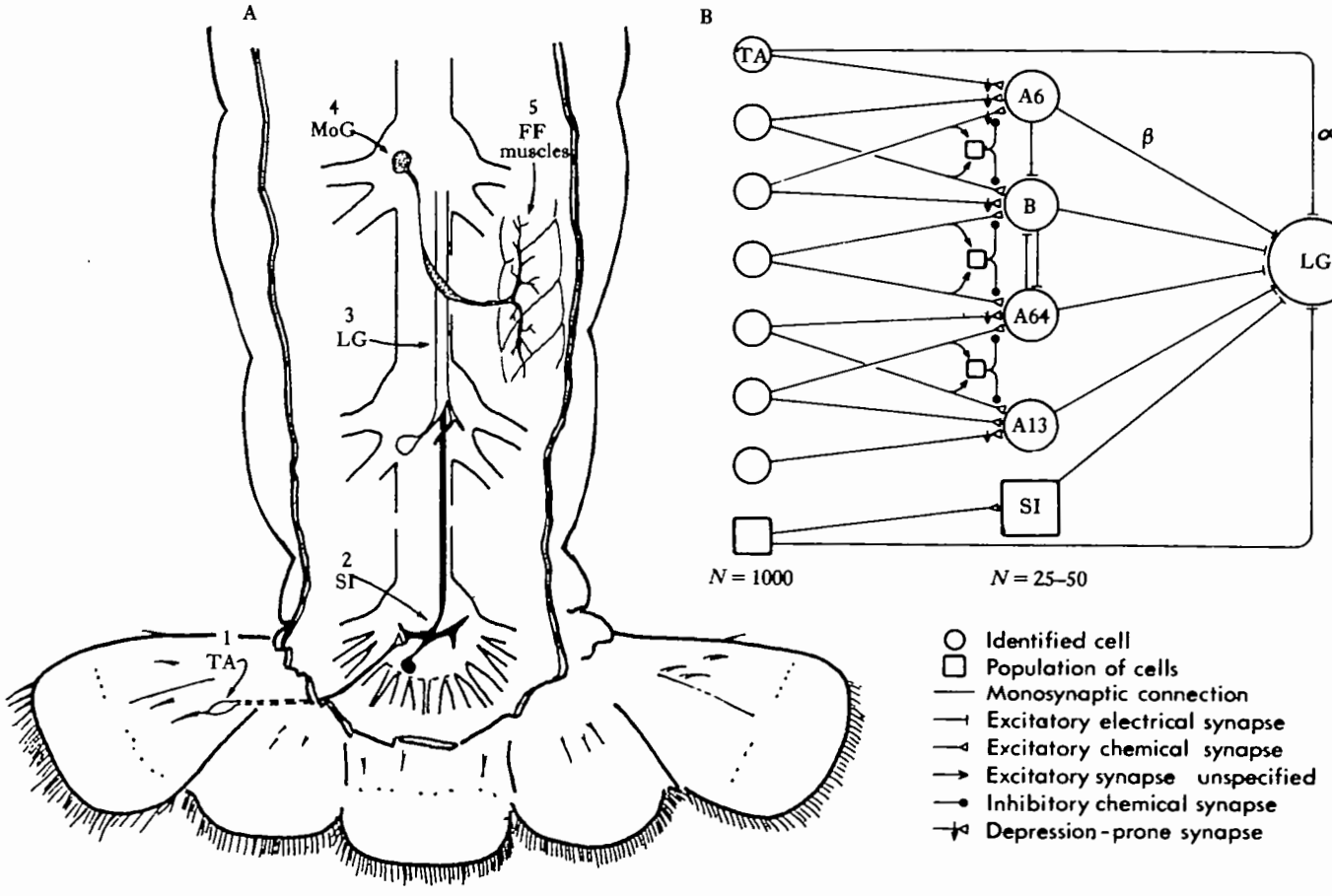


Fig. 6. Wiring diagram for afferent pathways to LG interneurone. (A) Semi-schematic representation of the shortest major pathway from receptors to muscles; this pathway involves five stages (numbered 1–5). The nervous system and elements are not drawn to scale, and only one lateral giant (LG) segment is shown. (B) Schematic of afferent pathways to LG interneurone, emphasizing considerable convergence. It is not known if the pathway is as direct as shown (from Wine & Krasne, 1982, based on Krasne, 1969; Zucker, 1972; Kennedy, Calabrese & Wine, 1974).

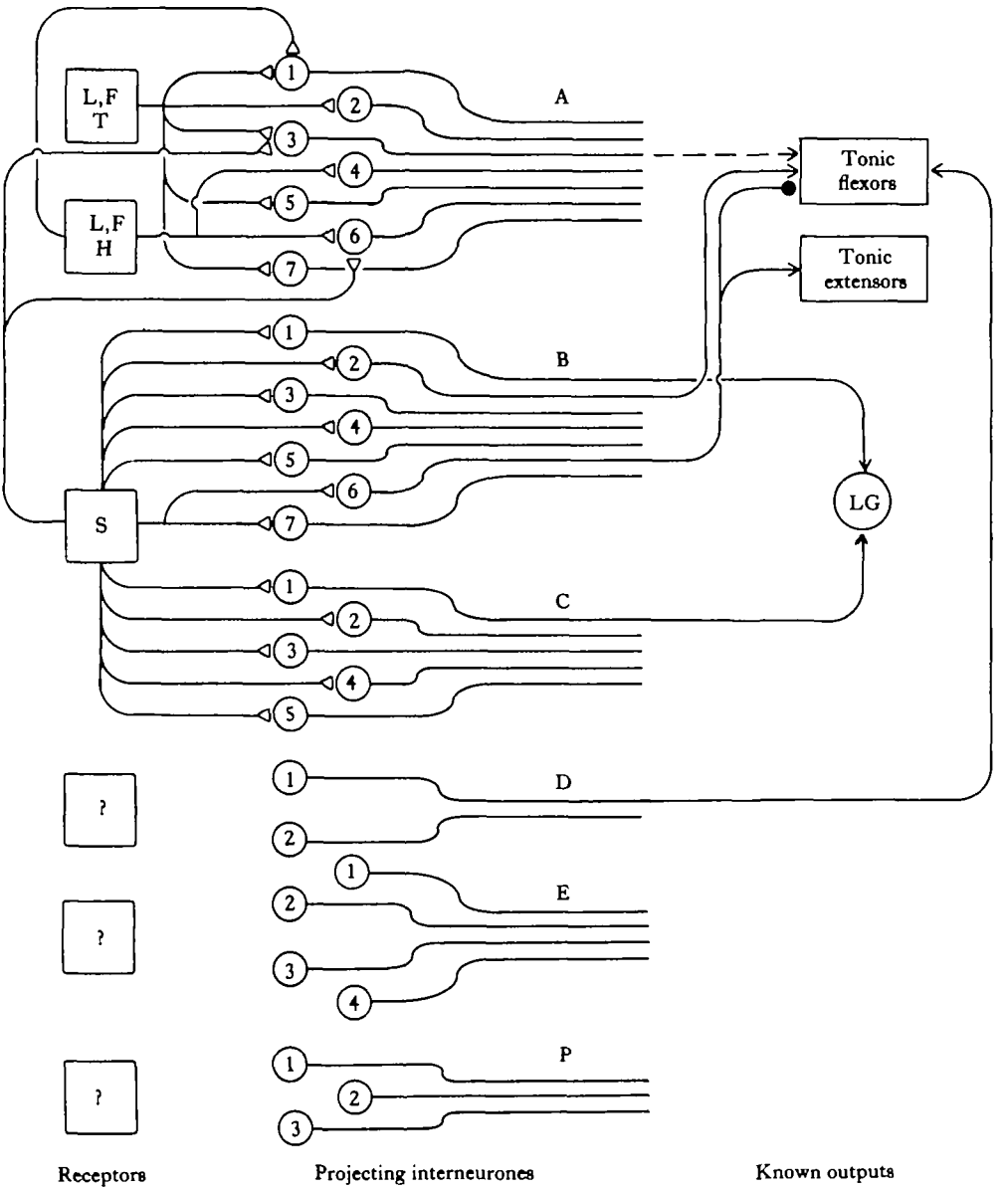


Fig. 7. Overview of the 6th ganglion sensory processor as presently understood. Schematic diagram of 28 identified projecting interneurones. Interneurones have been grouped in six categories as follows. Type A: 'slosh' or low-pass interneurones, receive input from long (L) and feathered (F) hairs, are directional [tailward (T) or headward (H)] and do not adapt. Types B and C: 'touch' cells, broad-band or high-pass, respond to breaking water surface or touching tailfan. Type B have non-spiking somata and mainly unisegmental receptive fields; type C have spiking somata and multisegmental receptive fields. Type D: appendage movement. When spontaneously active these interneurones are inhibited by touch or slosh. Type E: proprioceptive; they differ from type D in having spiking cell bodies. Type P: respond best to compression of exoskeleton. Only one quarter of the interneurones have identified targets (based on Sigvardt, Hagiwara & Wine, 1982; M. R. Plummer, G. Hagiwara, H. Reichert, K. A. Sigvardt, K. Wiese & J. J. Wine. unpublished; Zucker, 1972).

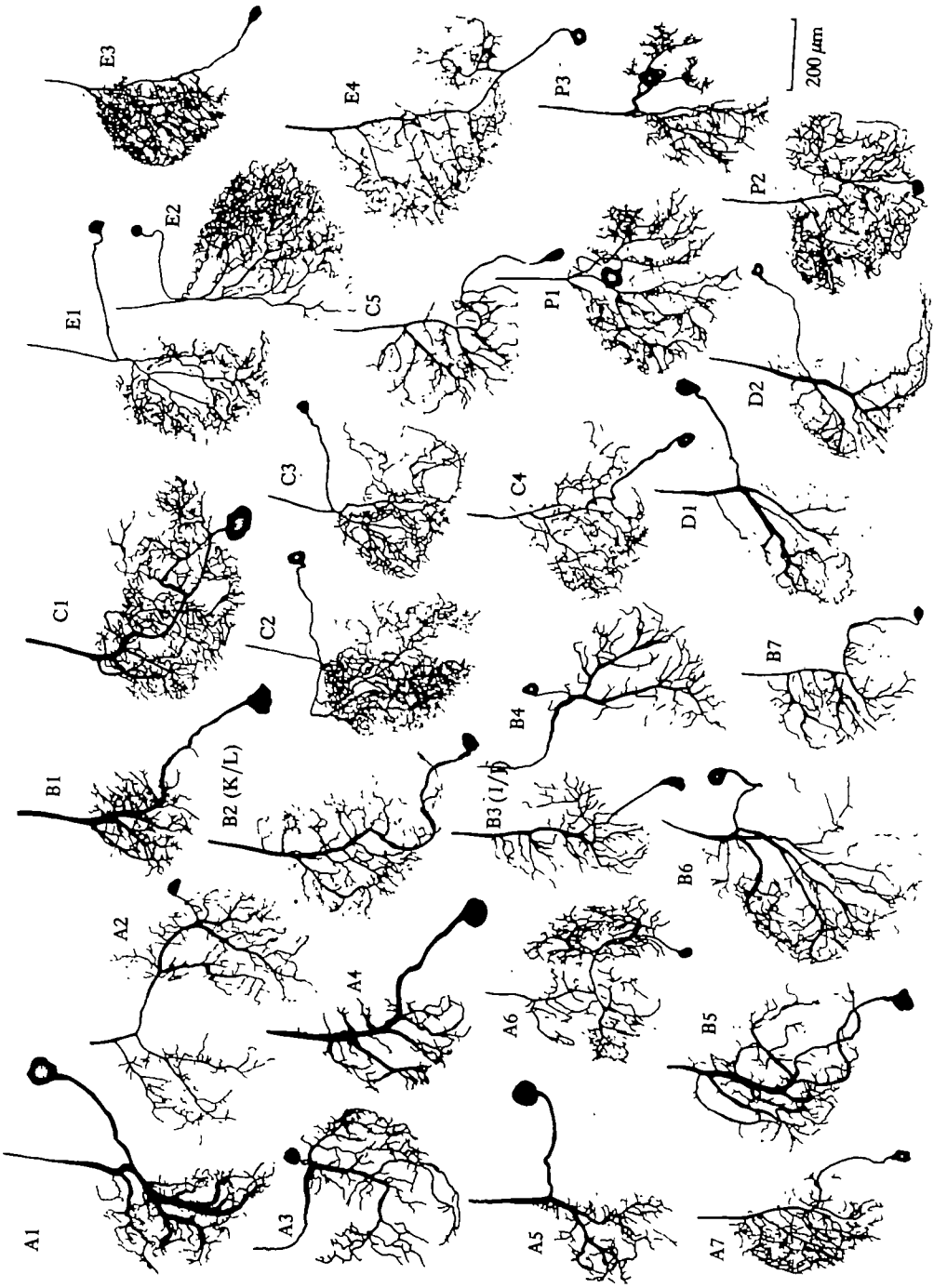


Fig. 8. Structures of 28 identified projecting sensory interneurones in the last abdominal ganglion of the crayfish.

General features of the sensory interneurones

Projecting sensory interneurones have been studied most intensively in the 6th abdominal ganglion. This ganglion innervates the tailfan, which appears to be specialized for sensory reception, and most processed sensory information leaves the ganglion *via* a small set of paired interneurones with cell bodies in the ganglion. We used cobalt backfills to obtain an estimate of about 50 pairs of interneurones, and then began studying these cells individually, using Lucifer Yellow injections to identify each cell and intracellular recordings during natural stimulation to establish its physiological profile (Sigvardt, Hagiwara & Wine, 1982).

Twenty-eight sensory interneurones have been identified so far (Figs 7, 8). In addition, several other projecting interneurones have been found which do not respond reliably to sensory stimulation. These include the lateral giant, 'corollary discharge' interneurones that are fired by the giant axons (including some neurones that produce primary afferent depolarization, PAD) and unidentified, 'higher order' interneurones. Some of the latter, as well as some of the interneurones that we have classified as sensory, may function as 'command' neurones for the postural or swimmeret system (Wiersma & Ikeda, 1964; Kennedy, Evoy, Dane & Hanawalt, 1967; Miall & Larimer, 1982*a*; Larimer & Jellies, 1983). We have not searched diligently for pre-motor axons which might have axons in the dorsal cord, and our techniques lead us to under-sample neurones which have small axons and poor responses to sensory input. Therefore, many of the projecting interneurones in the terminal ganglion which remain unidentified are likely to be non-sensory.

In general terms, what we have learned so far is as follows (Fig. 7).

(1) Each identified sensory interneurone has a unique set of anatomical and physiological properties.

(2) Primary sensory interneurones (cells close to and highly responsive to primary afferent input) can be either conventional (i.e. having non-spiking dendrites and a single spike-initiating zone), or 'pan-spiking' (i.e. having highly electrogenic dendritic membrane and multiple spike-initiating zones). Pan-spiking neurones always have multi-segmental receptive fields (Sigvardt *et al.* 1982).

(3) We have scant evidence for hierarchical synaptic organization among the projecting interneurones in the last ganglion. Some projecting interneurones weakly excite others (Zucker, 1972), but this interaction is usually reciprocal. We have no evidence for strong coupling such as is found among descending interneurones from the brain (Glantz, 1978). At present we consider the projecting cells as mainly independent, parallel channels.

(4) Some projecting interneurones are influenced by local sensory interneurones. Only one such pathway has been identified (Reichert *et al.* 1983*a,b*), but it remains possible that the majority of polysynaptic inputs are *via* local interneurones.

(5) Post-synaptic inhibition evoked by escape commands has been detected in only two interneurones, and in one of these it has been shown, in part, to be caused by some of the same inhibitory neurones that cause presynaptic inhibition of the afferent-to-interneurone synapse (Kirk & Wine, 1983; M. D. Kirk, unpublished data).

(6) Interneurones sensitive to water movements can be differentiated by their responsiveness to frequencies between 0·5 Hz and 400 Hz into 'low-pass', 'high-pass'

and 'broad-band' cells. Interneurones responsive to low frequencies are 'inhibited' at high frequencies: inhibition involves the peripheral suppression of firing in low-frequency afferents mentioned earlier; whether this is abetted by postsynaptic inhibition of the interneurones has not yet been established (Plummer *et al.* 1983).

(7) So far, we have only modest evidence that the sensory interneurones at this level are abstracting information, rather than simply summing it. As a first approximation, we can identify three classes of exteroceptors: (a) low-pass headward, (b) low-pass tailward and (c) high-pass. We thus have examples of interneurones which receive information from each class selectively, from (a) and (b), and from all three. The only spatial interaction documented so far is lateral inhibition among low-pass, headward-sensitive units (see next section).

Focus on a sensory subsystem: the low-frequency, non-adapting, directional system ('slosh' system)

If the fluid surrounding the tailfan is moved gently at frequencies as low as 0·2 Hz, seven pairs of sensory interneurones in the last ganglion fire vigorously for as long as the oscillations continue. Three pairs of these non-adapting interneurones do not respond to touching of the tailfan and are actually inhibited by high frequency water movements (i.e. >200 Hz). If the oscillations are directed along the rostro-caudal axis of the horizontal plane, one interneurone fires vigorously only in response to headward movements, one exclusively to tailward movements, and the third is bidirectional. The excitatory receptive fields of these three interneurones are restricted to the ipsilateral uropods and telson. When sensory roots within their excitatory sensory field are stimulated electrically the cells receive mixed excitation and inhibition and usually fire only one or two impulses even to intense shocks. We call these three interneurones 'slosh' cells. Four other directional interneurones have been found which differ from pure 'slosh' cells in that they also respond to touch, adapt slightly, have either bilateral, contralateral or multisegmental receptive fields, and fire short bursts of impulses to sensory nerve shocks. We call these 'slosh-touch' interneurones. Prolonged searches of the cell body layer, the neuropile and the connectives have not revealed evidence for more than seven interneurones in the 'slosh' and 'slosh-touch' category (M. R. Plummer, G. Hagiwara, H. Reichert, K. A. Sigvardt, K. Wiese & J. J. Wine, in preparation). Each of these interneurones has an axon which runs the entire length of the abdomen and presumably terminates in the brain.

A local, non-spiking 'slosh neurone' has also been found (Reichert *et al.* 1983*a*,*b*). This neurone is excited by headward water movement across the half of the tailfan ipsilateral to its cell body. It projects across the midline and inhibits both the headward and the bidirectional slosh cells on the other side. Since a bilateral pair of such cells exist, the system displays feedforward, lateral inhibition, and as such the subsystem should display common-mode attenuation. The sensory subsystem as we now understand it is shown in Fig. 9.

It is now technically possible to record from all 14 axons while exposing the tailfan to various stimuli. Individual neurones display moderate directional selectivity to an oscillating probe (Wiese & Wollnick, 1983). However, without knowing either the destination of the 14 axons in this system, or the animal's response to activity in the system, we cannot hope to discover what information is being extracted. We could,

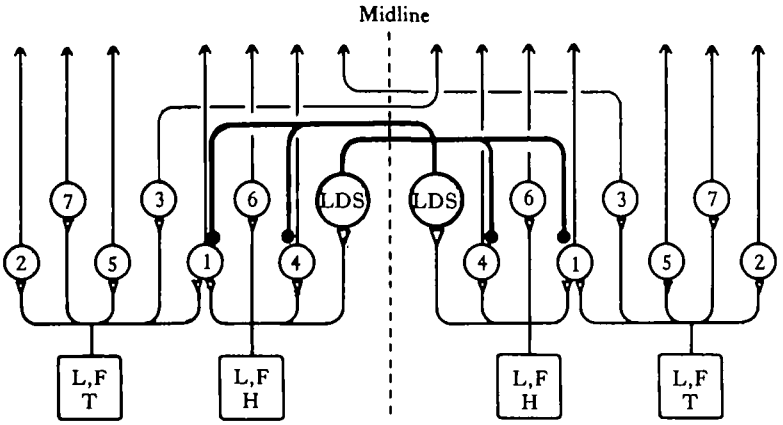


Fig. 9. Schematic diagram of the low-frequency directional system. Numbered cells are projecting interneurones, LDS is a local, non-spiking, headward selective, inhibitory interneurone. On each side, four projecting interneurones are excited by tailward (T) afferents, two are excited by headward (H) afferents, and one is excited by both. Two of the cells that are excited by ipsilateral, headward water movement are inhibited, *via* LDS, by the same movement made contralaterally. Some cells, (2, 5, 7) appear to be redundant in this simplified diagram, but cell 2 has bilateral branches and cell 7 is less selective (it responds to touch as well as water movements) and has a more extensive receptive field. L, long; F, feathered hair.

of course, record from all 14 axons simultaneously and identify patterns that are reliably evoked by various stimuli. But we would have no assurance that the animal looks at the output of these interneurones in the same way: it may compare only a subset of the cells, or it may integrate the output of this system with other information.

GENERAL FEATURES OF THE ABDOMINAL CNS

In a system as highly differentiated as the abdominal CNS, few generalizations about anatomical organization are likely to be absolute. Nevertheless, the system is highly ordered, and such order is of enormous practical significance to investigators, quite apart from any issues it might pose for developmental biologists. A brief summary of generalizations is as follows.

Afferents rarely cross the midline, rarely synapse on motor neurones, and do not directly inhibit their postsynaptic targets. All exteroceptive afferent cell bodies are located in the periphery, and their axons usually terminate in the ganglion where they enter, but some run anteriorly for several segments or posteriorly for one segment. Afferents continue to be added during postembryonic development and eventually outnumber efferents by about 100 to 1. Afferents are cholinergic, and show rapid, orthograde degeneration. The *receptors of the muscle receptor organs* are atypical in that their axons bifurcate and are distributed along the entire length of the CNS, their terminals cross the midline in the last abdominal ganglion, they synapse directly with efferents, they display very slow orthograde degeneration, they are fixed in number at hatching and they are postsynaptically inhibited in the periphery.

Efferents are fixed in number at approximately 100 per hemisegment, the majority of which innervate the appendage. Motor neurones to axial muscles may decussate, but appendage motor neurones rarely do. Efferents typically have single axons which

exit *via* a root in the ganglion of origin or, rarely, *via* a root in the next anterior ganglion. Most have single sites for spike initiation. Orthograde degeneration is very slow (weeks to months); putative transmitters are glutamate, GABA, proctolin and acetylcholine.

Interneurones are so diverse that only limited generalizations can be made. They range from small, local, non-spiking neurones to 'pan-spiking' projecting interneurones which run the entire length of the nerve cord and can initiate spikes at many sites within each of many ganglia. Most interneurones which have axons have only one, which either ascends or descends in the connectives; about 18 % have bifurcating axons (Jones & Page, 1983). Commissural interneurones are intraganglionic interneurones which have a single axon that projects *via* a commissure to the other side of the ganglion (e.g. Reichert *et al.* 1983*a,b*). Projecting interneurones show slow orthograde degeneration (Wine, 1973). Beyond the putative use of GABA by some inhibitory interneurones (Ochi, 1969) the transmitters used by crayfish interneurones are entirely unknown: the only well-characterized excitatory interneuronal synapses are electrical (e.g. Furshpan & Potter, 1959; Zucker, 1972; Roberts *et al.* 1982).

Integration begins with receptors which are rather narrowly tuned to specific stimulus features such as the direction and frequency of water movement or the bending of a single joint. These features are maintained and in some cases sharpened at the level of primary projecting interneurones. Receptor input is heavily modulated at the very earliest stages: both efferent control and presynaptic inhibition have been documented – the latter may be ubiquitous. Not all sensory interneurones are discriminitive; some respond to a very broad frequency range and have receptive fields that encompass half the animal's body surface. It is believed that specific sets of sensory interneurones converge on higher order interneurones which in turn make divergent connections *via* premotor 'driver' neurones to motor neurones. In at least one system (the swimmerets) endogenous oscillations can be generated by the premotor and motor neurones; these are coordinated by interganglionic connections (some made by motor neurone branches) and are controlled by interganglionic 'command' neurones (Wiersma & Ikeda, 1964). Integration at the level of interacting behaviour patterns, details of how a sensory stimulus recruits a specific pattern, of how spatial and temporal patterns of movement are generated by cellular interactions, and of how an activated motor system acts back on the animal's sensory systems have so far been worked out only for escape behaviour. The escape system is described briefly in the next section.

THE STRUCTURAL BASIS FOR A SIMPLE MOTOR PROGRAMME

Overview

Crayfish escape the rapid strikes of predators like racoons and fish by rapid backward swimming. The shock wave produced by a forceful movement toward the crayfish is detected by hair receptors that converge *via* sensory interneurones onto either the lateral giant (LG) or medial giant (MG) interneurones whose giant axons (the largest by far in the CNS) run the entire length of the nerve cord and drive abdominal flexor motor neurones (see Fig. 6) and, at the same time, inhibit the receptors, motor neurones and muscles of the abdominal extensor system. Thus, the

swimming powerstroke is produced at short latency by contractions of the massive abdominal flexor muscles. The flexion powerstroke is completed within about 30 ms. Even before flexion is completed, virtually all of the elements in the flexion circuit, from receptors to muscles, are also inhibited by long-lasting inhibitory postsynaptic potentials (IPSPs) that are triggered by the command signal and persist for the duration of flexion and re-extension. Re-extension is a chain-reflex caused by sensory feedback from flexion (Reichert, Wine & Hagiwara, 1981). It is triggered by the coincidence of sensory influx and the termination of the short-duration IPSPs produced by the flexion command neurones in extensor motor neurones, extensor muscles and the extensor muscle stretch receptors. In summary, the first tailflip of a short-latency escape response involves five steps:

(1) sensory triggering of the flexion command axons,
(2) excitation of the flexor muscles,
(3) short-lasting inhibition of the extensor system,
(4) delayed, long-lasting inhibition of the flexor system and
(5) delayed, feedback excitation of the extensors.

Usually, the initial tailflip is immediately followed by a series of tailflips occurring at a frequency of 10–20 cycles s^{-1}. This is escape swimming. Swimming is not a reiteration of the initial tailflip. It does not involve the giant axons, which are in fact inhibited during swimming, and the phase relations of extension and flexion are reversed so that extension leads flexion, which follows at short and relatively constant latency. Thus, when a crayfish swims slowly it keeps its abdomen tucked while coasting and then rapidly extends and flexes it again at the next cycle. This means that, during swimming, extension cannot be a flexion-induced reflex. In fact, swimming, like all cyclic behaviour patterns studied so far, is under the control of a central pattern generator (CPG), and the extension reflex is inhibited during swimming.

The swimming CPG is turned on by the same stimulus that causes the giant axons to fire. The CPG's onset is always considerably delayed, so that the first tailflip occurs before the CPG becomes active (Reichert & Wine, 1982, 1983).

In summary, the sequence of events in a crayfish rapid escape response is shown in Fig. 10. It may be useful to clarify some of the predictions of this model: It should be possible to obtain either a single command-mediated tailflip in isolation or the CPG in isolation; direct stimulation of the command neurone should produce only a single flexion and extension; and interference with flexion should interfere with re-extension. All of these predictions have been verified.

Orientation of the escape response is achieved in two ways. The initial response has only two variations: a rapid backward dart or a forward pitching movement. Each movement is commanded by a separate pair of giant axons with independent decision processes. The initial decisions ignore much detailed information about the stimulus, such as its laterality, and produce laterally symmetrical responses. The delayed activation of the CPG is accompanied by greater finesse in movement; the CPG codes at least the laterality as well as the rostro-caudal direction of the stimulus, giving a minimum of four distinct response modes.

Escape responses are only one aspect of a crayfish's behaviour and must be integrated with everything else it does. This means escape responses influence and are influenced by other behaviour patterns, including past behaviour. Once triggered,

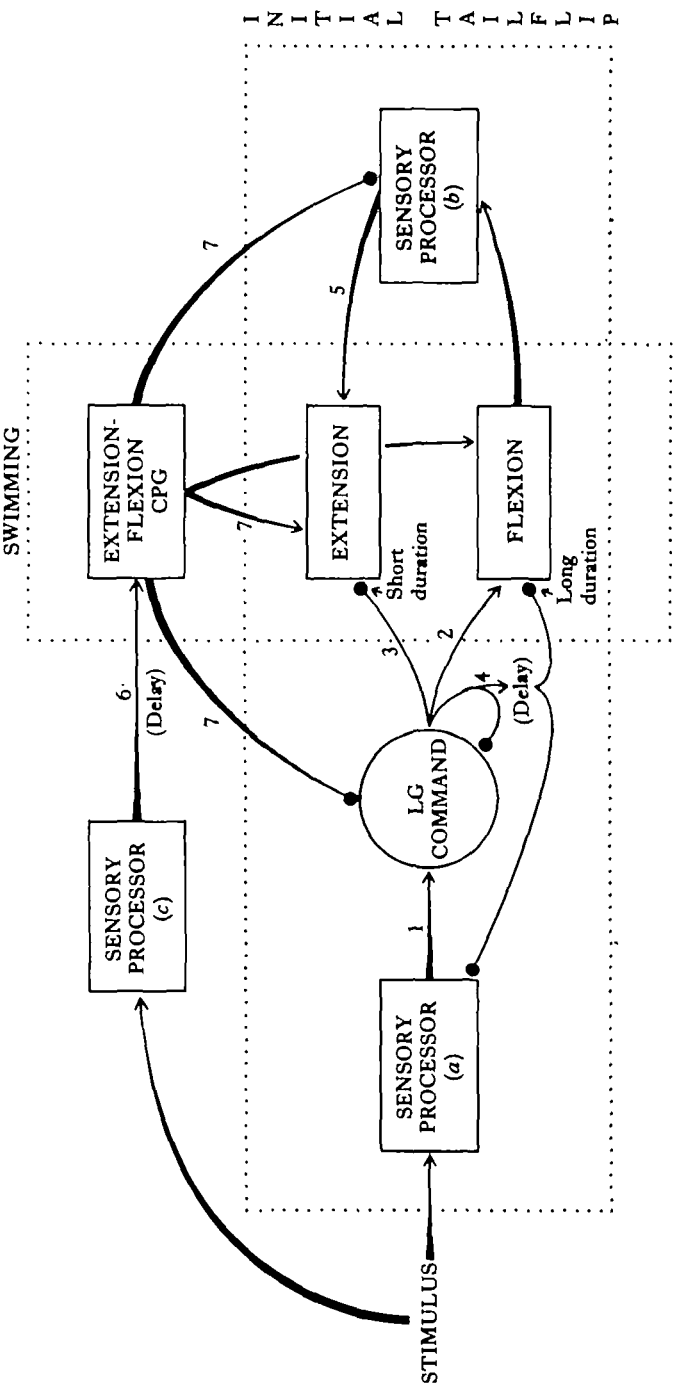


Fig. 10. Block diagram of major relations among components of the escape response. The horizontal, dotted rectangle encloses the components of the first, giant-mediated tailflip, the vertical, dotted rectangle encloses the components of the swimming central pattern generator. (Not all flexion elements are common to both systems.) The three 'sensory processors' (*a,b,c*) are designated by their functions and may have elements in common. Numbers on lines refer to sequence of events. Arrows indicate excitation and dots indicate inhibition. Elements have been identified within all boxes except the one labelled 'extension-flexion CPG'. Delay for feedback inhibition is only about 10 ms; delay for onset of swimming is 50–100 ms. Location of delay in the pathway to the CPG is unknown.

Escape has the highest priority of any behaviour pattern in the animal's repertoire: it will inhibit or override any competing pattern. However, the excitability of the triggering process is modulated by a wide range of influences so that in some circumstances it is virtually impossible to elicit escape (Krasne & Wine, 1975; Wine, Krasne & Chen, 1975).

In the preceding overview, the details of transduction, excitation-contraction coupling, synaptic action and connectivity patterns among the receptors, interneurones, motor neurones and muscles were deliberately ignored to give a broad perspective of the behaviour pattern. To an ever increasing extent, the pattern can be explained in terms of such details and a comprehensive review was recently published (Wine & Krasne, 1982).

Selected details of the neural circuitry involved in giant-mediated flexion patterns

In this section, I review selected recent findings about just one aspect of the escape response: the neural connections that mediate the initial patterns of flexion when the giant axons fire. After summarizing the 'basic' giant axon–giant motor neurone flexion circuit, I review recent evidence for sets of parallel connections with non-giant neurones which appear useless or maladaptive. To explain these connections it has been hypothesized that the giant axon circuitry evolved from the more complex non-giant circuitry that still controls most tailflips, and it has been proposed that all connections which the giants once had with the non-giant circuits have been maintained unless selected against. Connections detrimental to the motor patterns produced by the giant axons have either been lost, weakened or inhibited (Wine & Krasne, 1982; Krasne & Wine, 1984). Because the effects of weakened or inhibited synapses can be overridden, at least in principle, it has gradually emerged that the giant axon motor circuits contain numerous mechanisms that might enable them to produce altered motor patterns to the same command (e.g. Kramer, Krasne & Bellman, 1981*a*; Miller *et al.* 1984). Whether and under what circumstances they might do so is a question that is now being investigated.

Giant axon circuitry: basic flexion patterns produced by the giant interneurone–giant motor neurone networks

In each of the seven abdominal segments, a pair of giant flexor motor neurones innervate almost all of the fast flexor (FF) muscles in its segment *via* powerful but depression-prone synapses. The motor giants are in turn fired monosynaptically by rectifying electrical synapses from the giant interneurones. A striking difference exists in the pattern of connections between the MG and LG axons and the motor giants: the MG axons synapse with the motor giants in every segment, whereas the LG axons synapse with the motor giants in the anterior three segments but not in the posterior four segments. The behavioural consequences of that arrangement are shown in Figs 11 and 12A.

The role of two corollary discharge interneurones

The first level of complexity added to that simple arrangement is as follows. In the 2nd and 3rd abdominal ganglia, the LGs and MGs fire a pair of large interneurones, termed I2 and I3, which descend to the terminal ganglion where they synapse on

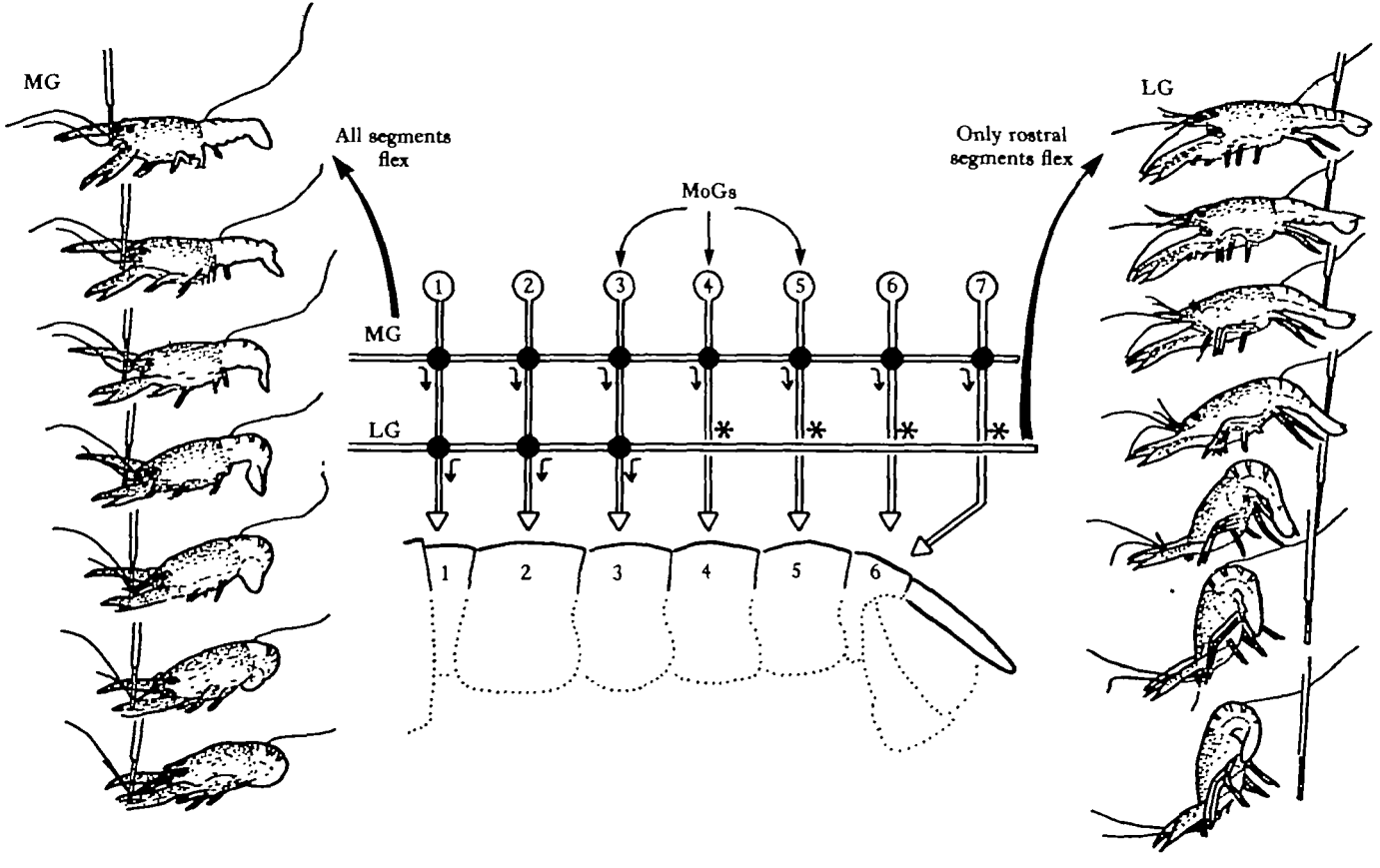


Fig. 11. Connectivity patterns of the giant command axons with the motor giants explain the orientation of the initial escape movement. The side panels are tracings from high speed films that show the responses to tapping the head, which fires the medial giant (MG) command axons, and to tapping the abdomen, which fires the lateral giant (LG) command axons. When MGs fire, all motor giants (MoGs) are excited, all segments flex, and the abdomen curls and propels the animal backward. When the LGs fire, there is no output to the caudal segments, which remain straight and so cause the thrust to be directed mainly downward, thus pitching the animal forward. Since the MGs respond to rostral inputs and the LGs to caudal ones, each tailflip pattern removes the animal from the stimulus source. Approximately 50 ms of escape behaviour are shown. The centre panel is a schematic diagram of the synaptic connections between the command axons (horizontal lines) and the motor giants (vertical lines). Direct, rectifying, electrical, one-to-one synapses are represented as filled circles; omitted synapses are indicated by asterisks (based on Furshpan & Potter, 1959; Wine & Krasne, 1972; Mittenthal & Wine, 1973). The motor giant homologues in the terminal ganglion were recently identified

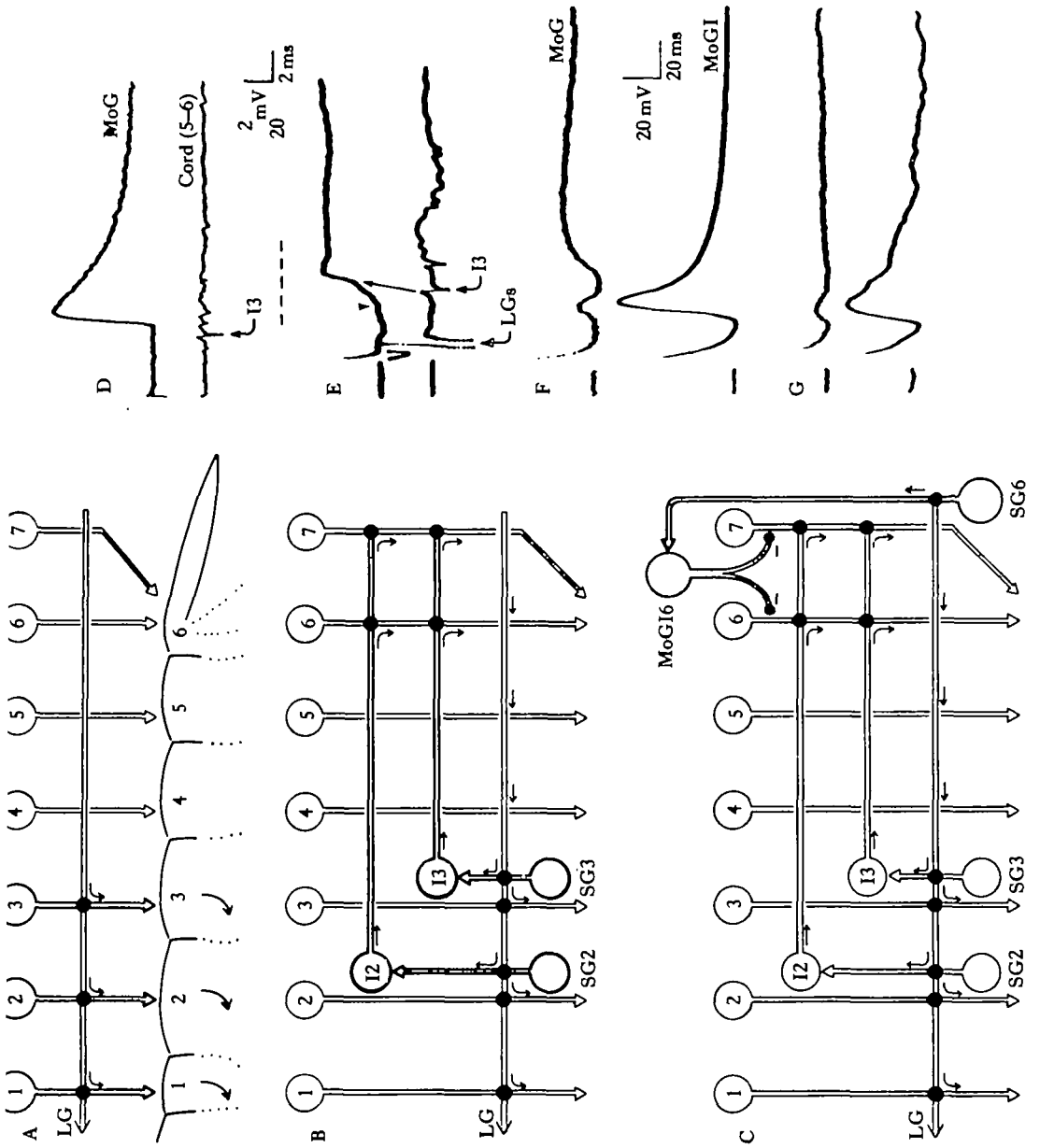


Fig. 12. The role of feedforward inhibition in motor patterning. (A) Basic flexion circuit between the lateral giant axons and the motor giants. Same conventions as Fig. 11. (B) The corollary discharge pathway. The LGs fire segmental giant neurones in ganglia 2 and 3; the SGs in turn fire I2 and I3, which bypass motor giants (MoGs) in intervening ganglia but strongly excite the MoGs to the telson flexors. (SGs are fired by the LGs in every ganglion, but are shown only in ganglia 2 and 3 for clarity.) (C) The disruptive effects of the telson motor giant output are cancelled by feedforward inhibition of the motor giants *via* the SG in the last ganglion and a local neurone inhibitor of the telson flexor MoGs. (D) Intracellular recording from a telson flexor motor giant (MoG). The cell is fired by stimulation of a single I3. (E) Same neurone, but now the LG is stimulated. Although I2 and I3 both fire, the cell is inhibited by the early IPSP (from Kramer, Krasne & Wine, 1981*b*). (F) Simultaneous recording in the telson flexor motor giant inhibitor (MoGI) and motor giant (MoG) while stimulating a terminal ganglion SG. (G) When the MoGI is hyperpolarized, the IPSP in the MoG is lost (based on Dumont & Wine, 1983; M. D. Kirk, J. P. C. Dumont & J. J. Wine, in preparation).

many neurones, including the motor giants to the telson flexor muscles (Kramer *et al.* 1981*a*; Kramer, Krasne & Wine, 1981*b*; Fig. 12B). The summated EPSPs of I2 and I3 are sometimes sufficient to fire the motor giants (MoG) (Fig. 12D). This pathway is redundant with the basic connectivity pattern of the MGs, but for the LGs the effect would be disruptive and would cause its motor output pattern to resemble the MG one. However, the telson motor giants are strongly inhibited within a few ms of MG and LG firing (Kramer *et al.* 1981*a,b*; Dumont & Wine, 1983; Fig. 12E,F). This inhibition comes too late to interfere with the direct input from the first MG impulses, but is early enough to cancel the effect of the input from I2 and I3. As far as we can tell, the net effect of all of this additional wiring is to leave the basic motor pattern unchanged (Fig. 12C). All the elements shown in Fig. 12C have been identified (Fig. 13).

The role of non-giant motor neurones

A second level of complexity is that, in each ganglion, the motor giants are in parallel with up to nine non-giant, fast flexor (FF) motor neurones which, in aggregate, co-innervate the same muscles as the motor giants (Fig. 14). In other words, each fast flexor muscle fibre receives input from both a motor giant and one or more non-giant motor neurones. In the 2nd and 3rd ganglia, the LGs and MGs make equivalent, suprathreshold connections to all the FF motor neurones *via* the intervening 'driver' interneurone called the segmental giant (Roberts *et al.* 1982; Fig. 13A). These connections reinforce the effects of the LG and MG commands. However, in the posterior four segments, firing of the FFs would disrupt the basic motor pattern of the LG-MoG network. The obvious solution to this problem would be to eliminate the synapses between the LGs and SGs in the posterior four segments, just as the synapses to the MoGs were eliminated. However, the crayfish has not adopted the obvious solution. Instead, perhaps because the SGs are important for exciting other neurones, the LGs and MGs maintain equivalent, suprathreshold connections to the SGs the whole length of the abdomen, but the LG pattern is preserved because in the posterior segments the strength of the synapses between the SGs and FFs is below threshold for firing the motor neurones (Miller *et al.* 1984; J. P. C. Dumont & J. J. Wine, in preparation; Fig. 14).

Even though the SG-to-FF synapse is usually subthreshold in posterior segments, single LG impulses sometimes fire FF motor neurones (Miller *et al.* 1984) and multiple impulses, which often occur with natural stimuli (Wine & Krasne, 1972), summate to fire the cells with much higher probability (Miller *et al.* 1984). This suggests that some activity in the posterior fast flexor muscles should occur during tailflips mediated by multiple LG impulses. Such activity would disrupt the forward pitch of the animal and cause the trajectory to resemble an MG-mediated, backward tailflip. One way to quantify the tendency of the animal to pitch forward is to measure the angle of the body, relative to the substrate, at the termination of initial flexion (Fig. 15). For 37 naturally evoked, LG-mediated tailflips this angle was 107 ± 12°, whereas for 10 naturally evoked, MG-mediated tailflips the angle was 26 ± 9°. When the LGs were fired directly *via* implanted electrodes, single shocks gave a pitch angle of 76 ± 15° ($N = 36$), whereas triplets (at 200 Hz) reduced the angle to 64 ± 12° ($N = 11$) (Fig. 15; G. Hagiwara, L. Miller & J. J. Wine, unpublished). The decreased angles seen with

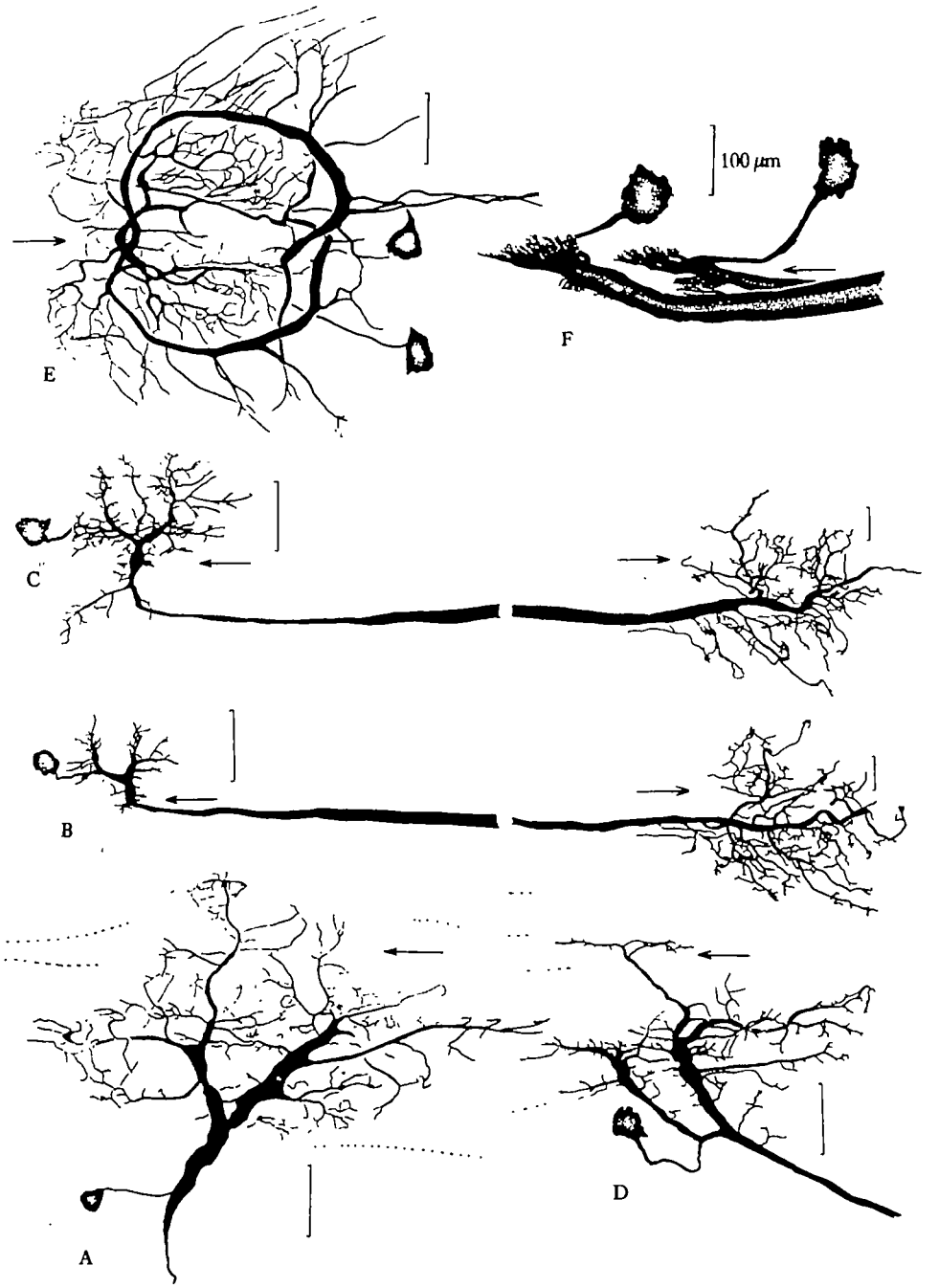


Fig. 13. Identification of elements in the corollary discharge pathway. (A) Segmental giant in ganglion 2. (B) Input and output regions of I2. (C) Input and output regions for I3. Dendrites and terminals were filled in animals of different sizes. (D) Segmental giant in terminal ganglion. (E) Inhibitors of the telson flexor motor giants. (F) The motor giants of the telson flexors. (A from Roberts *et al.* 1982; B & C from M. D. Kirk, unpublished; D & E from M. D. Kirk, J. P. C. Dumont & J. J. Wine, in preparation; F from J. P. C. Dumont & J. J. Wine, in preparation). Scale bars, 100 μm.

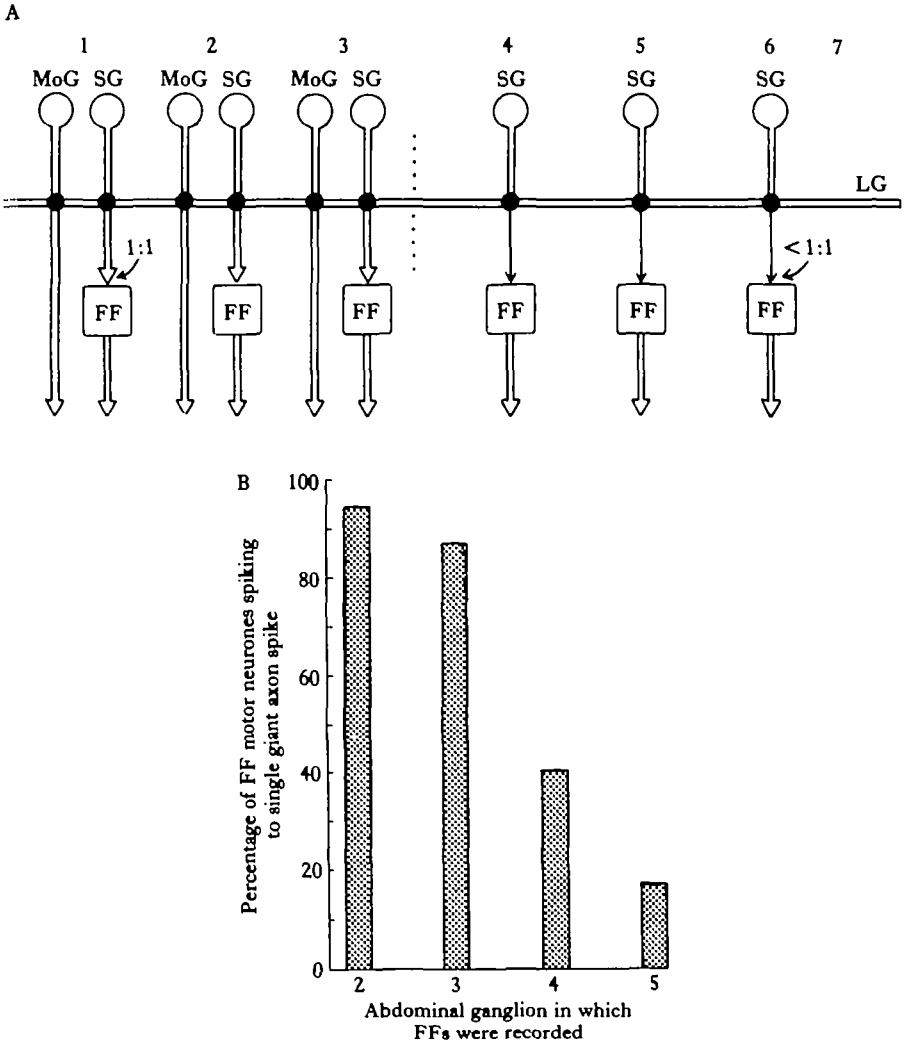


Fig. 14. Parallel pathways of giant and non-giant (FF) motor neurones to the flexor muscles. Every ganglion has, in addition to the MoGs, a parallel pathway consisting of the SG and from five to nine non-giant, fast flexor motor neurones. However, posterior to the 3rd ganglion (dotted line) the MoGs are not excited by the LGs (hence they are omitted from the diagram) and the SG-to-FF pathway is much weaker, so that the probability of FF motor neurones firing in the posterior ganglia is low. The decreased probability of FFs firing in ganglia 4 and 5 is shown in (B). Nevertheless, the FFs in posterior ganglia do sometimes fire, and the probability of firing can be increased to near 1·0 by multiple impulses in the LG (based on Miller, Hagiwara & Wine, 1984).

central stimulation may mean that in these instances, as in physiological preparations, some FF motor neurones in posterior ganglia are being recruited by the LG spikes. How then are we to explain the greater angle and relatively invariant form of naturally-evoked, LG-mediated tailflips?

A novel role for peripheral inhibition: a hypothesis

It appears that another level of complexity is required to explain the spatial pattern

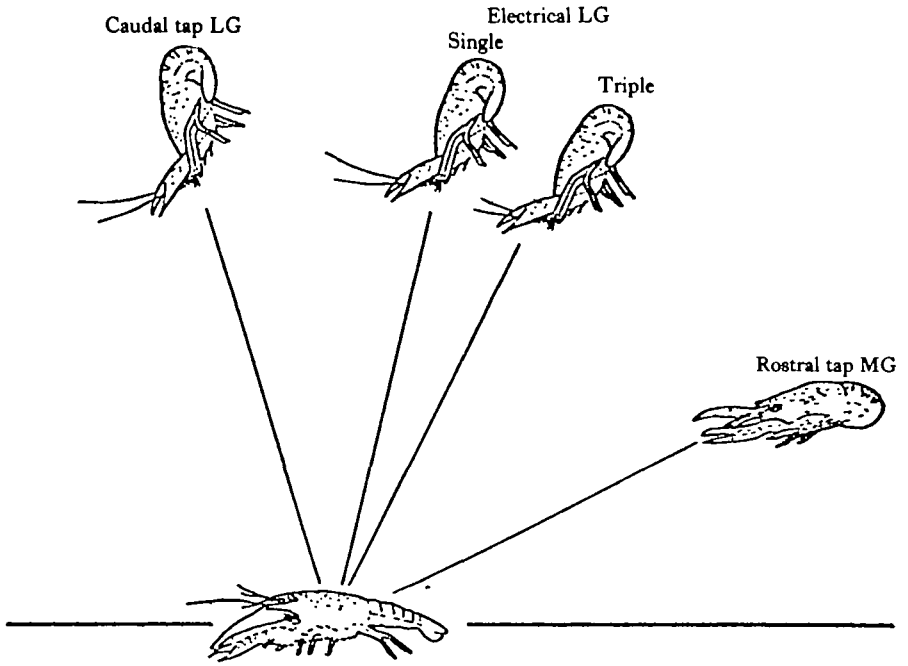


Fig. 15. Variations in escape trajectories during LG-mediated tailflips. Note more pronounced forward pitch following natural stimulation. Tailflips evoked by direct, electrical stimulation of the LGs produced a less pronounced forward pitch; further degradation of LG-mediated responses occurred when the LGs were directly stimulated to fire bursts. The LGs were not monitored during naturally evoked tailflips, but on the basis of other work multiple impulses probably occurred on some trials.

of LG tailflips. We have circumstantial evidence that, during LG-mediated tailflips, the flexor muscles in the posterior segments, and especially in the telson, are inhibited by early and strong firing of the flexor peripheral inhibitors* (FIs), while in anterior segments FI firing is delayed and serves mainly to limit the duration of the response. The evidence for this is as follows (Fig. 16). When a crayfish is tapped on the abdomen (an adequate stimulus for an LG tailflip) a powerful sensory barrage is produced in the peripheral inhibitory neurones of the fast flexor muscles (FI neurones). In fact, shocks to sensory nerves in the last ganglion typically fire at least one of the FIs to the telson flexor muscles well below the threshold for LG activation (J. P. C. Dumont & J. J. Wine, in preparation and Fig. 16C). The FIs are also excited, with a delay, by central pathways activated by the giant axons (Wine & Mistick, 1977). This delay is shortest in posterior ganglia (Uyama & Matsuyama, 1980; Fig. 16D) and becomes very short in the telson (Fig. 16E). Furthermore, although the MGs and LGs have equivalent effects on the FIs in anterior ganglia (Wine & Mistick, 1977), only the LGs

*'The anterior telson flexor muscle' is excited by the LGs, and does not receive any peripheral inhibition (Larimer & Kennedy, 1969). However, this apparent exception to the rule of peripheral inhibition of fast flexors is simply due to the misnaming of the muscle. The ATF muscle has its insertion on the anterior telson which gives it little leverage on that structure. It in fact acts mainly on the uropods (J. P. C. Dumont & J. J. Wine, in preparation).

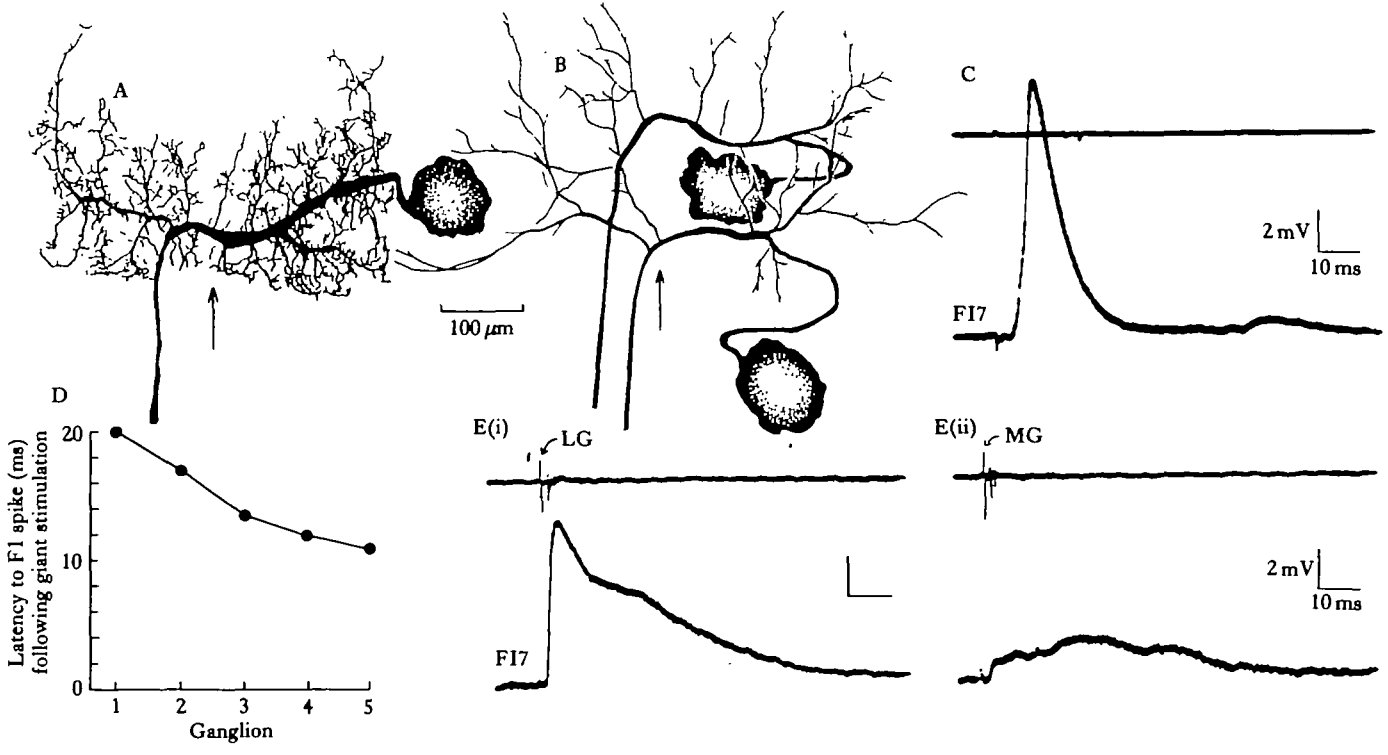


Fig. 16. Does peripheral inhibition help maintain the spatial pattern of flexor activity during LG-mediated tailflips? (A) Structure of flexor inhibitor in ganglion 2. (B) Structure of telson flexor inhibitors in terminal ganglion. (C) Firing of a telson flexor inhibitor by sensory input at an intensity considerably below LG threshold. (D) Earlier firing of FIs in ganglia 4 and 5. (E) Differential firing of a telson flexor inhibitor by the LGs but not the MGs (from Dumont & Wine, 1983; Uyama & Matsuyama, 1980).

Latency to F1 spike (ms)
following giant stimulation

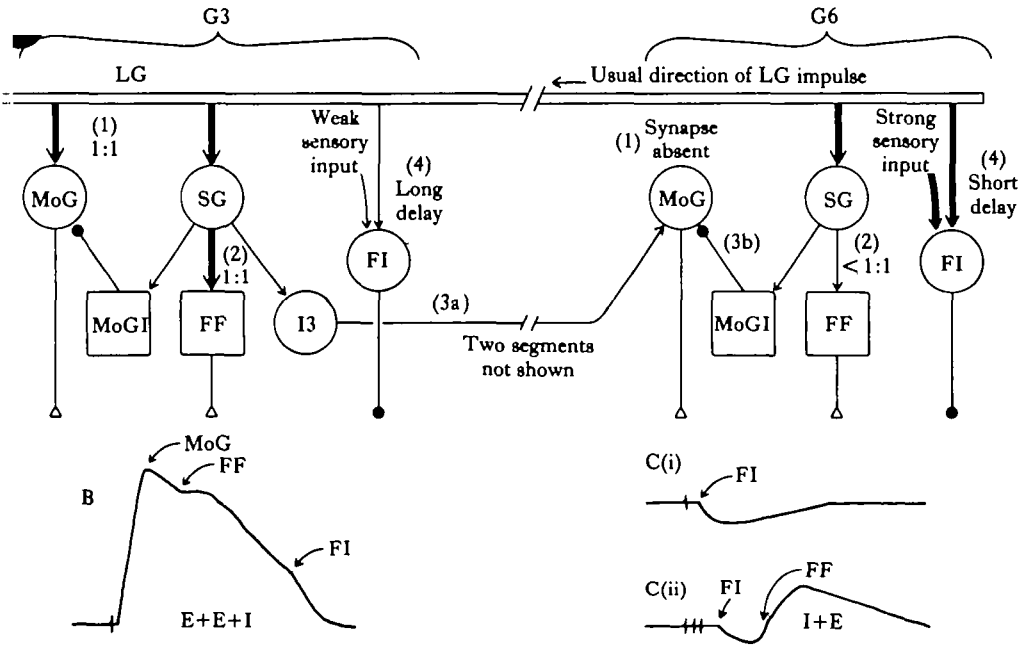


Fig. 17. Four mechanisms for producing the spatial pattern of LG tailflips. (A) Summary of differences in premotor organization of homologous elements in two abdominal ganglia. Under usual conditions, an impulse in the LG axons causes a strong flexor discharge only in the anterior segments of the abdomen. The reasons for this are as follows. (1) The most direct pathway to the muscles, the monosynaptic pathway from LGs to MoG, is missing in the posterior ganglia. (2) The disynaptic pathway from LGs to FFs *via* the SGs is much weaker in the posterior ganglia. (3) Though the MoGs in the telson receive delayed excitation *via* I3 (pathway 3a), its effects are inhibited (pathway 3b).(4) Finally, whereas the flexor inhibitor in anterior ganglia receives weak sensory input and delayed central input, the FI in G6 receives strong sensory input and short latency central input, so that it fires early enough to inhibit FF output. (B) Idealized pattern of activity in a fast flexor muscle fibre of an anterior segment following an LG impulse. The MoG fires with a very brief central delay, FFs follow within about 2 ms, and the FI fires after a delay. (C) Idealized activity in a telson flexor muscle fibre to an LG impulse. (i) Under some (most?) circumstances, neither the MoGs nor non-giant telson flexor motor neurones fire and the only response is inhibitory. (ii) On occasion, particularly with multiple LG impulses, summation acts to fire non-giant telson flexors, but the greater delay required by summation, the lack of MoG, and the shorter delay of FI allow peripheral inhibition to cancel the motor output. E, excitation; I, inhibition. (Based on J. P. C. Dumont & J. J. Wine, in preparation; Kramer, Krasne & Wine, 1981*b*; Miller, Hagiwara & Wine, 1984.)

strongly excite the FIs to the telson flexor muscles (J. P. C. Dumont & J. J. Wine, in preparation; Fig. 16E).

All these findings are consistent with early and strong inhibition of flexor muscles in the posterior segments and telson during LG-mediated tailflips. Peripheral inhibition in these segments might be very effective. Since the motor giants never fire in segments 4 and 5 during LG tailflips (because they receive no input from LGs in those segments) and rarely fire in the telson segments (because the polysynaptic pathway to them from the LGs is inhibited, Fig. 12), peripheral inhibition only needs to counteract the effects of the FFs. Because the FFs typically require summation to fire in these segments, they are probably firing at least several ms later than they fire in anterior segments, and hence are more vulnerable to peripheral inhibition. Thus, although it has not yet been demonstrated directly, we have considerable

circumstantial evidence that peripheral inhibition of the flexor muscles helps maintain the spatial motor pattern of LG tailflips.

To summarize: an LG tailflip has its characteristic spatial pattern because the segments innervated by ganglia 4, 5 and 6 do not flex, whereas the segments innervated by the first three ganglia do (Fig. 11). We believe four different features jointly contribute to the production of the spatial pattern. At the most basic level, the differential activation of the flexor muscles in the anterior and posterior segments of the abdomen occurs because the motor giants are monosynaptically excited by the LGs only in the anterior segments. However, the LGs indirectly excite telson flexor MoGs and non-giant FFs in ganglia 4 to 6. The disruptive consequences of these potential flexor outputs in the posterior segments are prevented by central inhibition of the telson MoGs, by a weakened excitatory pathway to the FF motor neurones in the posterior segments, and by earlier and stronger peripheral inhibition of the fast flexor muscles in posterior segments. These features are summarized in Fig. 17.

CONCLUSION

We are attempting to uncover principles of neural integration by concentrating on a single invertebrate species. For the crayfish, we have found it necessary to focus still more narrowly: our anatomical and neurophysiological studies are confined to the abdominal nervous system, and our neurobehavioural analysis deals almost exclusively with escape behaviour. Within these narrow confines, our studies have provided neural underpinnings for several basic ethological concepts, such as innate releasing mechanisms and fixed action patterns. The neural networks we are constructing are more complicated than many people expected, and our progress is therefore slower. The complexity might be an artifact of our inadequate understanding, and we may eventually discover a much simpler way of expressing the circuitry. We doubt that. It seems more likely that our present picture is, in fact, oversimplified.

Why do simple behaviour patterns require complex neural networks? We do not yet know, but one possibility for the apparent mismatch is that conventional descriptions of behaviour may overlook many nuances that have evolved to ensure optimal performance in a wide range of conditions. This leads to simplified descriptions of behaviour, and causes us to underestimate the complexity of the neural circuits that control behaviour.

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