What are the neural mechanisms underlying the generation of rhythmic motor patterns? As demonstrated by the papers in this volume, recent work in the fields of biophysics, behaviour, pharmacology and electrophysiology has greatly advanced our understanding of the neural basis of rhythmic behaviour in vertebrates and invertebrates. However, when we stand back and consider our comprehensive knowledge of the ‘grand scheme’ of motor output production — including the interactions between central pattern generation, inter-oscillator coordination, sensory feedback and central command — it seems as though we are still a long way from our goal of a ‘complete understanding’ of any one system. In this paper, we summarize our recent progress towards understanding one aspect of the rhythmic behaviours produced by the stomatogastric ganglion of the California spiny lobster. The aspect we have focused on is the production of patterned motor output by the two central pattern generators (CPGs) located within the stomatogastric ganglion. We do not discuss the role of sensory feedback or inter-system coordination, and touch only briefly on a few aspects of the control by the central nervous system of stomatogastric activity. We do not wish to minimize the importance of sensory feedback and central input in shaping the output of the CPG to a behaviourally appropriate motor pattern; however, we do exclude them as obligatory factors for the basic operation of the CPGs. Our rationale has been that a detailed description of the CPG must be a first step in the understanding of the system as a whole, and will serve as the basis for a subsequent determination of the functions and mechanisms of sensory feedback and central control.

In order to understand ‘how a CPG works’ one must be able to explain, in mechanistic terms, (1) the existence of a pattern (i.e. why do the neurons display cyclic rather than tonic activity?), (2) the phase relationships between the bursts of activity, and (3) the overall frequency. The appropriate ‘mechanistic terms’ with which to formulate such an explanation would involve causal relationships between physiologically important,
experimentally measurable parameters: for example, the degree to which cell A is inhibited by a burst of 10 action potentials in cell B. How much data must we obtain about a CPG in order to formulate such an explanation? Ultimately, the following pieces of data would be necessary for a complete, comprehensive model:

1. All of the neurons that participate in the production of the motor output pattern must be identified.
2. The synaptic connectivity or 'circuity' between the cells must be determined.
3. A physiological characterization of the intrinsic properties of each cell must be obtained.
4. The properties of the individual synapses, such as strength, time course and facilitatory properties, must be quantitatively measured.

It is unrealistic to expect that such a complete data set could ever be obtainable for any system – and indeed, such a total description might not even be necessary for a meaningful understanding of a complex motor system. Moreover, a different approach from the reductionistic one outlined above might conceivably circumvent the necessity for such an arduous strategy. However, our approach in this relatively simple invertebrate system has been this direct frontal assault, aided by the use of a new photoinactivation technique that allows a reduction in the complexity of the neural networks during our experimental analysis. Our goal has been the collection of as much of the 'ideal data set' as we could obtain, and the formulation of an explanation of pattern generation in this system. In the following pages we will summarize: (1) what we set out to learn about the stomatogastric system, (2) how we used the photoinactivation technique to simplify our experiments, (3) the data we obtained about the pyloric and gastric mill CPGs, and (4) our qualitative explanation of pattern generation in these systems.

What parameters should we measure?

The motor pattern generated by a neural network results from the interaction of complex elements (i.e. the neurons) in a complex arrangement (i.e. the circuit). It seems as though nature has offered an extensive 'palette' of intrinsic neuronal properties and connectivity schemes from which different CPGs have been assembled. Several comprehensive lists of important cellular, synaptic and network parameters have been published elsewhere. Two excellent reviews are those by Bullock (1976) and Friesen
Fig. 1. Cellular properties important for pattern generation. A neuron can be silent (A); tonically active (B); an endogenous burster requiring no synaptic or hormonal input (C); a conditional burster, i.e. requiring synaptic or hormonal input to maintain bursting (D); or may show plateauing, i.e. a bistable membrane potential requiring a short depolarizing pulse to trigger onset and a short hyperpolarizing pulse to trigger termination (E). Cells can also show varying degrees of postinhibitory rebound. The time to the onset of bursting (Δt) can be variable (F). Neurons also demonstrate accommodation to prolonged depolarization (G). V, voltage; I, injected current; Nerve stim., nerve stimulation.

& Stent (1978). The first provides a table of neuronal parameters that gives degrees of freedom in assembling networks, and the second summarizes several different types of nerve networks that can theoretically produce patterned output. The following list summarizes, in general terms, those cellular, synaptic and network properties that have been found to play important roles in pattern generation in the lobster stomatogastric ganglion.

Intrinsic cellular properties (see Fig. 1)

1. The relationship of firing threshold to resting potential. Is the neuron spontaneously active or inactive in its normal rest state?
2. The ability of a neuron to generate bursting pacemaker potentials (BPPs). Such neurons can be further subdivided into two categories: ones which produce BPPs when totally isolated from synaptic inputs (i.e. true 'endogenous bursters' as described by Alving, 1968), and those which
require some synaptic or neurohormonal activation of BPP generation (i.e. 'conditional bursters': Levitan, Harman & Adams, 1979; Anderson & Barker, 1981).

3. The ability of a neuron to generate 'plateau potentials', i.e. prolonged, regenerative depolarizations due to voltage-dependent conductances (Russell & Hartline, 1978). Such neurons have two stable points on their current-voltage relationships at which the net membrane current is zero: the normal resting potential and a depolarized 'plateau potential' above firing threshold. The distinction between a plateau-potential-generating neuron and a BPP-generating neuron is that the former presumably needs extrinsic neuronal inputs to act as 'triggers' for transitional jumps between the two polarization levels, whereas the BPP-generating cells can undergo the transitions spontaneously as a result of some intrinsic mechanism.

4. The amplitude and time course of postinhibitory rebound (PIR), which is the transient overshoot of the membrane potential above the initial resting potential following release from inhibition. Since this PIR can often reach threshold, it may trigger a burst of spikes or even a plateau potential transition.

5. The magnitude and time course of adaptation, which is the spontaneous decrease in spike frequency during constant excitation.

Properties associated with synaptic interactions (see Fig. 2)

1. The amplitude and time course of the synaptic conductance, which can be different at different synapses.

2. The function relating presynaptic voltage to transmitter release, i.e. does transmitter release require invasion of the terminal by an action potential, or is transmitter released as a continuous function of membrane polarization? (See Maynard & Walton, 1975; Raper, 1979; Graubard, Raper & Hartline, 1980.)

3. The coupling coefficients and rectification properties of electrotonic interactions.

Network properties (see Fig. 3)

1. The existence of reciprocally inhibitory cell pairs. The simplest oscillatory network that can be (theoretically) constructed from neurons that are individually incapable of generating BPPs is the 'half-centre' oscillator. This oscillator model, first proposed by McDougall (1903) and later reformulated by Brown (1914), required that (a) two neurons be coupled by reciprocally inhibitory synapses, and (b) some 'restorative' process intrinsic to each neuron would cause the inhibitory effect of each cell upon
Fig. 2. Synaptic properties important for pattern generation. A, longer time constants of the inhibitory postsynaptic potentials in cell B than in C lead to increased summation in response to a spike train in cell A. B, graded transmitter release can cause graded postsynaptic effects without spiking ($V_{BA}$) as well as discrete postsynaptic potentials, while a presynaptic neuron which does not release transmitter in a graded fashion causes unitary postsynaptic potentials only. $V_{BA}$ and $V_{FI}$ are voltage responses of the same cell to different types of presynaptic neurons ($V_{PS}$). C, electrical coupling allows spread of de- or hyperpolarizing current ($I_A$) into another cell ($V_B$) with different degrees of attenuation and rectification. The coupling coefficient can be altered by additional synaptic input (Nerve stim., nerve stimulation). D, excitation ($V_C$) may be transmitted by conventional mechanisms as in cell B ($V_B$) or be delayed as in cell C ($V_C$).

the other to decrease with time after the initiation of its activity. (Synaptic fatigue and adaptation are examples of such restorative processes; PIR has also been shown to serve the same function in this model by Perkel & Mulloney (1974).) With common tonic excitation from an extrinsic source, the two reciprocally inhibitory neurons would theoretically fire in alternating bursts of action potentials.

2. Phasic inhibition of active neurons. If a neuron that would otherwise fire tonically is rhythmically inhibited by another neuron, then it can be constrained to fire in bursts. Indeed, phasic inhibition of an otherwise inactive cell can result in bursts of impulses if the magnitude of PIR is great enough. The ‘driver’ neuron could be oscillatory due either to an intrinsic ability to generate BPPs or to reciprocal inhibitory interaction with another neuron.

3. Phasic excitation of inactive neurons. This mechanism is the complement of phasic inhibition. Note that the phase relationship between...
'driver' and 'follower' neurons will be different in the above two cases: rhythmic inhibition in synchronous bursts. However, within these general phasic constraints, the precise phase of the follower cell burst will depend upon the strengths and time courses of the driver synapses, the PIR kinetics, and upon the time course of any voltage-activated conductances. For example, if the excitatory postsynaptic potentials (EPSPs) of an excitatory driver cell turn on a potassium current in the follower cell, the time it takes for the follower cell to reach threshold can be extended, resulting in a significant phase lag of the follower burst (see Getting, this volume).

Finally, it is important to note that all of these intrinsic, synaptic and network properties could be labile. The intrinsic and synaptic parameters...
might be influenced by extrinsic synaptic or neurohormonal input, allowing
for changes in the pattern. By significantly altering the strength of certain
synapses, certain elements of the circuit could effectively be ‘re-wired’
during activity (see Eisen & Marder, 1982).

In order to understand pattern generation in the lobster stomatogastric
ganglion, all of the properties listed above will ultimately have to be
characterized quantitatively. Until recently this had seemed to be a hope-
less task. The functional roles and intrinsic properties of single neurons
could not be observed in isolation from the confounding effects of other
neurons, due to the complex activity of the network in which they were
embedded. The goals of our recent experiments have been to assess inde-
pendently the relative importance and functional roles of factors such as
BPP generation and reciprocal synaptic inhibition through the use of a new
cell inactivation technique.

**Dye-sensitized photoinactivation of specific identified neurons**

The technique for the inactivation of specific neurons is a two-step process
(Miller & Selverston, 1979). First, the cell to be inactivated is filled with the
fluorescent dye Lucifer Yellow via intracellular iontophoresis. The dye
itself has no adverse effects on the cell. Second, the tissue containing the
neuron is illuminated with intense blue light. Within 5 min the cell con-
taining the dye becomes totally and irreversibly inactivated – or ‘killed’ –
by the following criteria: (1) The membrane depolarizes to ground, (2)
action potentials are no longer recorded in the cell’s axon, and (3) post-
synaptic potentials (PSPs) can no longer be observed in cells postsynaptic
to the treated neuron (see Fig. 4). No other neurons are harmed if care is
taken to filter the ultra-violet and infra-red components out of the illu-
nimating beam. After an effective ‘recovery period’ of about 30 min
(necessary to allow a rinse-out of the transmitter released by the killed cell)
the experimenter is left with a reduced and simplified circuit from which
one neuron has been functionally ‘excised’. By filling and irradiating
several cells simultaneously, significant reductions in the network can be
obtained. Moreover, it is possible to inactivate small portions of single
neurons, by filling the whole cell with dye but irradiating only a small por-
tion of it with a focused microbeam (Miller & Selverston, 1979, 1982b).

We have followed four different strategies in our application of this
technique to the stomatogastric system (see Fig. 5):

1. In order to determine whether a particular identified cell was necessary
   for pattern generation, the cell was killed and the effects on the overall
   pattern observed.
Fig. 4. Dye-sensitized photoinactivation of an identified nerve cell. In this experiment, activity was recorded from the cell bodies and axons of two cells, the PD and LP cells, as shown in A. Panel B illustrates the activity of the cells before the LP cell was inactivated. The bottom trace shows a recording from the LP cell body which has been filled with the dye Lucifer Yellow. Note that the LP action potentials can be correlated with (i) discrete inhibitory postsynaptic potentials (IPSPs) recorded in the PD cell body (middle trace) and (ii) large extracellularly recorded spikes in a peripheral nerve (top trace). C shows the results of illuminating the dye-filled LP cell with intense blue light for 5 min: (1) the intracellularly recorded potential has depolarized to ground (actually it depolarized off-scale, and has been brought back into the figure here with a negative d.c. offset), (2) the IPSPs on the PD cell are no longer visible, and (3) LP spikes can no longer be recorded on the peripheral nerve.

Fig. 5. Strategy for the use of the photo-inactivation technique in studying the stomatogastric system. See text for details. A, deletion of one or more cells (cell A). B, isolation of a pair of cells (A and B). C, isolation of a single cell (cell A). D, killing part of a cell (axon of cell A).
2. In order to investigate the intrinsic properties of a single neuron, that cell could be isolated by photoinactivating all cells within the ganglion that were presynaptic to it.

3. In order to test the sufficiency of small subsets of the CPG networks for generating oscillatory activity, small groups of neurons could be isolated from the larger network by photoinactivating all other cells in the network that were presynaptic to the few cells of interest.

4. In order to 'uncouple' two neurons in different ganglia, a section of the interganglionic axon of one of the cells could be photoinactivated (by filling that whole neuron with dye, but irradiating only the small section of the nerve containing its axon).

In the following section we will describe the lobster stomatogastric system, and give specific examples illustrating how the application of this technique has increased our understanding of pattern generation.

The lobster stomatogastric system

The stomatogastric system of the California spiny lobster (Panulirus interruptus) contains 30 neurons that form the basis of two central pattern generators (CPGs: for a review see Selverston et al., 1975). One of the two rhythmic behaviours executed by the lobster stomach is a rapid (2 Hz) pumping and filtering of food particles in the pyloric region of the foregut. The sequence of striated muscle contractions which produces this behaviour is generated by the pyloric network – a group of 14 out of the 30 neurons. The other behaviour within the stomach is the mastication of large food particles by three internal teeth in the 'gastric mill' region. The gastric movements are generated by the gastric network – a group of 11 out of the 30 cells in the stomatogastric ganglion. In addition to these cells within the ganglion, several neurons have been identified in a serially adjacent pair of ganglia – the commissural ganglia – that contribute to the maintenance and control of the two rhythms. Although the movements associated with these behaviours occur within the thorax of the animal and are therefore not visible externally, the muscles involved are striated and require motor output patterns from the central nervous system for their operation (Fig. 6.4). Like the leg muscles, they have typical crustacean neuromuscular junctions and the overall pattern is modulated by peripheral sensory receptors in vivo. In fact, it may be helpful to think of the stomach as an internalized appendage rather than as an autonomic system.

Our standard physiological experiments are performed on 'combined preparations' in which the stomatogastric ganglion, the oesophageal
ganglion, the pair of commissural ganglia, and all of their interconnecting nerves are dissected off the lobster stomach. When this stomatogastric nervous system is pinned out in a chamber containing physiological saline, both rhythms persist for many hours. The pyloric system always operates when the commissural and oesophageal ganglia are removed, but generates a faster and more robust pattern when they are present. The gastric mill
Fig. 7. Burst patterns (A) and circuits (B) of the pyloric (A) and gastric (B) central pattern generators. The extracellular recordings are obtained from combined preparations as shown in Fig. 6B. There is one E and P neuron cell body located in each commissural ganglion. The inhibitory synapses are indicated by black dots, excitatory synapses by black triangles and electrotonic coupling by resistors. The diode indicates a rectifying electrotonic junction. Heavier lines represent the stronger synapses. Functional synapses are indicated by small dots. Staggered arrangements of cells indicate the total number of cells in the group.

system is more dependent upon these ganglia for stable operation, and produces a rhythmic pattern only infrequently when they are absent.

A diagram of the preparation is shown in Fig. 6. Extracellular recordings of activity in the motor nerves are shown in Fig. 7. The relatively large (40–80 μm) cell bodies in the stomatogastric ganglion are readily accessible to intracellular recording and manipulation by current injection. The
Table 1. Stomatogastric neurons

<table>
<thead>
<tr>
<th>Neuron</th>
<th>No.</th>
<th>Axon location</th>
<th>Approximate phase</th>
<th>Special features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pyloric</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anterior burster (AB)</td>
<td>1</td>
<td>stn</td>
<td>0</td>
<td>Interganglionic interneuron; endogenous burster; modulates P cells</td>
</tr>
<tr>
<td>Pyloric dilator (PD)</td>
<td>2</td>
<td>pdn</td>
<td>0</td>
<td>Strongly electrically coupled to AB</td>
</tr>
<tr>
<td>Lateral pyloric (LP)</td>
<td>1</td>
<td>lpn</td>
<td>0.5</td>
<td></td>
</tr>
<tr>
<td>Ventricular dilator (VD)</td>
<td>1</td>
<td>mvn</td>
<td>0.75</td>
<td>Phase shifted by input</td>
</tr>
<tr>
<td>Inferior cardiac (IC)</td>
<td>1</td>
<td>mvn</td>
<td>0.5</td>
<td>Weak activity, variable phase</td>
</tr>
<tr>
<td>Pyloric (PY)</td>
<td>8</td>
<td>pyn</td>
<td>0.65</td>
<td>Early and late types</td>
</tr>
<tr>
<td>P cell</td>
<td>2</td>
<td>stn</td>
<td>0.45</td>
<td>In commissural ganglia; general phasic excitor</td>
</tr>
<tr>
<td>Gastric</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Interneuron 1 (Int 1)</td>
<td>1</td>
<td>stn</td>
<td>0.6</td>
<td>Inhibits E cells</td>
</tr>
<tr>
<td>Lateral gastric (LG)</td>
<td>1</td>
<td>lgn</td>
<td>0.15</td>
<td>Synergists; close medial tooth</td>
</tr>
<tr>
<td>Medial gastric (MG)</td>
<td>1</td>
<td>mgn</td>
<td>0.0</td>
<td></td>
</tr>
<tr>
<td>Lateral posterior gastric (LPG)</td>
<td>2</td>
<td>lpgn</td>
<td>0.6</td>
<td>Opens lateral teeth; very tonic when not inhibited</td>
</tr>
<tr>
<td>Dorsal gastric (DG)</td>
<td>1</td>
<td>dgn</td>
<td>0.81</td>
<td>Synergists; conditional bursters; plateauing</td>
</tr>
<tr>
<td>Anterior median (AM)</td>
<td>1</td>
<td>amn</td>
<td>0.81</td>
<td></td>
</tr>
<tr>
<td>Gastric mill (GM)</td>
<td>4</td>
<td>aln</td>
<td>0.3</td>
<td>Tonic when E cells are active</td>
</tr>
<tr>
<td>E cell</td>
<td>2</td>
<td>stn</td>
<td>0.2</td>
<td>In commissural ganglia; general phasic excitor</td>
</tr>
</tbody>
</table>

neurons that participate in the production of the patterns have been identified, and their circuitry extensively studied (Maynard, 1972; Maynard & Selverston, 1975; Hartline & Gassie, 1979; Eisen & Marder, 1982). Circuit diagrams representing the synaptic connectivities are shown in Fig. 7. The neuron names corresponding to the abbreviations in these figures are given in Table 1. The circuit diagram of the pyloric network incorporates the data obtained recently by Eisen & Marder (1982). Except for the lumping of two classes of cells with slightly different properties (i.e. the ‘early’ and ‘late’ pyloric cells, or PE and PL cells) into one class (the ‘PY’ cells), this diagram is considered to be complete and precise. In the
diagram of the gastric circuit, however, several interactions are shown which are classified as 'functional', i.e. monosynapticity has not been established. This will be discussed in more detail below. Note that all but two stomatogastric ganglion neurons in the pyloric and gastric circuits are motoneurons. As well as making synaptic interconnections with each other, they send axons to the stomach musculature. Two neurons, the anterior burster (AB) cell in the pyloric network and interneuron 1 (Int 1) in the gastric network, are interganglionic interneurons, making connections with neurons in the stomatogastric and commissural ganglia.

The connectivity data incorporated into these circuit diagrams are the result of a very large amount of work. Don Maynard pioneered the stomatogastric preparation with his initial circuitry mapping of the pyloric network (Maynard, 1972). Several new connections were documented over the last few years (Maynard & Selverston, 1975; Hartline & Gassie, 1979). Eisen & Marder (1982) made a fundamental contribution towards understanding the pyloric network by taking a close, critical look at the synaptic interactions of an electrotonically coupled pool of cells. The AB cell and the two PD cells are strongly electrically coupled to one another, always fire in synchrony, and play important roles in pattern generation. Until Eisen & Marder's work was begun, it was thought that those three cells made exactly the same set of synaptic interactions with the other pyloric neurons. They realized, however, that the strong electrical coupling might be 'masking' differences in their connectivities. By photoinactivating different combinations of pyloric neurons they demonstrated two important points. First, they showed that the AB and PD cells do not make the same sets of connections with the LP and VD cells (see Fig. 7). Second, they showed that in cases where these three cells do make synapses onto the same postsynaptic cell, the PSPs produced have very different time courses. The basis for this effect was shown to be the use of different transmitters by the two cell types. The corresponding receptors on the follower cells yielded PSPs with different biophysical characteristics. The overall summed character of the PSP in this case is dependent upon the relative activity levels of the two cell types, which in turn is dependent upon the strength of the electrotonic coupling between these cells. This in turn is variable, allowing a postsynaptic response with a continuously 'adjustable' time course and amplitude within a particular range of parameters.

Equivalent surprises may await us in the gastric mill circuitry, and may await others of our colleagues in the systems they study. We feel, however, that these circuits are certainly close enough to being complete for a meaningful discussion of pattern generation mechanisms; this is presented in the following sections.
Fig. 8. Bursting continues in the pyloric system after PD and AB cells are removed but inputs from the commissural ganglia are left intact. A, control pyloric rhythm; B, when PD-AB cells are removed by photoinactivation, bursting continues; C, sucrose block of stomatogastric nerves causes the remaining cells to fire tonically or become silent; D, stimulation of stomatogastric nerve proximal to the sucrose block (arrow) reactivates the bursting mechanism.

The pyloric network

Until our recent work using the photoinactivation technique, we considered the existence, phases and frequency of the pyloric pattern to be derived from the intrinsic properties of three neurons: the AB and the two PD cells. All three were thought to be 'endogenous bursters', which drove the pyloric rhythm via their strong inhibitory synapses with the rest of the pyloric neurons. To test this hypothesis the AB and PD cells were simultaneously photoinactivated, as shown in Fig. 8 (Selverston & Miller, 1980). The remaining portion of the pyloric network still maintained a stable, rhythmic pattern, demonstrating that the AB and PD cells could not be considered as 'drivers'. However, if the inputs from the commissural ganglia were blocked with a sucrose pool on the stomatogastric nerve, all patterned activity ceased. A short stimulus volley to the stomatogastric nerve, between the sucrose block and the stomatogastric ganglion, would re-initiate a few cycles of patterned activity.

Subsequent experiments helped to explain these observations. In one set of experiments individual pyloric neurons were tested for their ability to generate bursting pacemaker potentials (BPPs) (Miller & Selverston, 1982a). This was done by inactivating all cells within the pyloric network known to be presynaptic to the test neuron, and blocking all extraganglionic input into the stomatogastric ganglion. In this condition, only one of the three cells previously presumed to be bursters continued to generate BPPs.
Stomatogastric ganglion

Fig. 9. PDs are not endogenous bursters, while AB is. A and C show the control pyloric rhythms with the AB and PD cells recorded intracellularly. In B, the inputs to AB were removed and the neuron continued to burst. In D, when the inputs to the PD were removed, the cells fired tonically.

This was the AB cell, which is also the only interganglionic interneuron in the pyloric network (see Fig. 9). The two PD cells, in contrast, fired tonically when isolated from synaptic inputs and from their tight electrical coupling with the AB cell. However, a short high-frequency stimulus volley to the stomatogastric nerve would elicit an episode of BPP generation in the PD cells. Stimulation of this nerve also elicited BPP generation in two other pyloric cell types: the VD and LP cells. Thus, four cells in the pyloric system can be considered as 'conditional bursters'.

Fig. 10 shows an episode of bursting initiated by stomatogastric nerve stimulation in all four of these conditional bursters (the VD, LP and two PD cells). The first part of panel A shows the spontaneous bursting pattern of these cells that was generated when: (1) the cells presynaptic to them within the stomatogastric ganglion were inactivated, and (2) the inputs from the commissural ganglia were left intact. The second half of panel A shows the results of blocking the stomatogastric nerve with sucrose: the cells go tonic or silent. Panel B begins with the cells in this non-bursting state, with the input nerve sucrose-blocked. During the interval marked
with a bar, the stomatogastric nerve was stimulated at 50 Hz. This short stimulus initiated an episode of bursting from these cells lasting 26 s, the first few seconds of which are shown here. After 30 s, the cells reverted to their tonic or silent modes. These results demonstrate: (1) that other inputs from the central nervous system to the stomatogastric ganglia are important to the control and maintenance of the pyloric rhythm, (2) that one of the mechanisms underlying these functions is the modulation of BPP generation properties in some of the neurons, and (3) that those inputs need not be continuously or phasically active to exert their effects.

Although it would seem from these experiments that BPPs are essential for pattern generation in this system, other experiments demonstrated that resonant network properties were equally important. When the AB interneuron in a stomatogastric ganglion is killed and the stomatogastric nerve is sucrose-blocked, none of the remaining neurons spontaneously produce BPPs (i.e. only nerve stimulation will elicit that activity in this case). However, this ‘burster-less’ network will still generate patterned output (Selverston & Miller, 1980). This indicates that the pattern in this case is an ‘emergent property’ of the network, and suggests that the pattern in a fully intact preparation results from a cooperative interaction of intrinsic BPP
and resonant network properties. In order to investigate the network properties contributing to pattern generation, we determined the minimal subset of pyloric neurons that could generate rhythmic activity due to network interactions alone. We observed the activity of isolated pairs of neurons which were functionally 'dissected' away from the rest of the pyloric network. This was accomplished by photoinactivating and hyperpolarizing all neurons presynaptic to the two cells of interest. In order to eliminate the involvement of BPP generation, these experiments were performed with the endogenously oscillatory AB cell killed and with the input nerve blocked. We found that a network of two cell types - the LP and PD cells - would go into stable reciprocal oscillations when their overall activity levels were appropriately adjusted by a steady depolarization of their cell bodies. This is shown in panel C of Fig. 10. To our knowledge, this is the first demonstration of a real 'half-centre' oscillator in a biological system, and clearly shows that patterned activity can be generated through the interconnection of neurons that would otherwise fire tonically.

The different factors contributing to pattern generation can be understood through a progressive 'thought exercise'. In this exercise, functional components of the pyloric CPG are successively added on to an elemental half-centre 'skeleton network'. The transformation of the pattern at each step of the exercise is explained mechanistically in terms of cellular and network properties. This thought exercise is presented in Fig. 11. For each step, we present diagrams of the synaptic connectivity and of observed activity patterns for the neurons being considered. These activity phase diagrams were prepared from experimental data, examples of which are also presented in the figure.

For purposes of simplification, the synaptic interactions were divided into two categories according to synaptic strengths. Strong interactions are drawn as continuous lines (e.g. LP to VD) and weak interaction by broken lines (e.g. LP to FY). Synapses were defined as 'strong' if the postsynaptic response to a presynaptic burst had an absolute amplitude of greater than 5 mV, as measured from data presented elsewhere (Miller & Selverston, 1982b).

We begin our thought exercise with the VD–LP cell pair, and with the stomatogastric nerve in the normal, unblocked condition. As shown at the left of the first panel of the figure the VD and LP cells establish a basic two-phase pattern due to (1) their reciprocally inhibitory synaptic interactions, and (2) their input-induced oscillatory behaviour. We could also have begun our exercise with the PD and LP cells. These cells are also capable of generating reciprocal bursts of action potentials, even in the absence of commissural inputs, due to their 'half-centre-like' activity.

In the second panel, we examine the activity generated by the circuit
Fig. 11. A thought experiment on how the pyloric system works. In each part of this figure, an example of the activity recorded from an isolated subset of the pyloric network is shown. Also shown in each panel is a block diagram representing one typical cycle of that activity, and a diagram of the synaptic connections of the cells within that subset. For the circuit diagrams, all symbols and abbreviations are as previously used. Relative synaptic strengths are indicated by continuous lines (strong synapses) and broken lines (weak synapses). For all of this figure the stomatogastric nerve is assumed to be in its normally active, unblocked state. See text for more details.

made up of all three of these cell types: the PD, VD and LP cells. The pattern is still biphasic, with the PD and VD cells firing simultaneously, in antiphase to the LP cells. This activity would be expected from an examination of the circuit. The VD and PD cell types both make strong reciprocal inhibitory connections with the LP cell. Since either a VD or PD cell alone will fire in antiphase to the LP cell, and since the VD and PD cells do not inhibit each other's activities, then they would be expected to fire simultaneously, in antiphase to the LP cell. The simultaneity of PD and VD activity is reinforced by their weak electrical coupling. Thus, we can understand this stage of our exercise as an elaborated 'half-centre' oscillator: a biphasic activity pattern, based upon reciprocally inhibitory pairs, and reinforced by the generation of BPPs in all three cell types.

In the next step of our exercise, the IC and PY cells are added to the circuit. The IC cell is inhibited by the VD and PD cells, but is not affected by LP cell activity. Therefore, it would be expected to fire some time during
the LP activity. This was observed to be the case. The addition of the PY cells establishes a third phase element to the pattern. The PY cells intercalate their bursts after the LP/IC group, and before the PD/VD group, by 'prematurely' terminating the LP bursts with respect to their activity in the preceding step of the thought exercise.

The PY cells are strongly inhibited by the PD cells, and one subset of the PY cells (the 'late' or 'PL' cells) is inhibited by the VD cells. Thus, the PYs would not be expected to fire during the PD/VD activity period. The PY cells are weakly inhibited by the LP cell, and receive no inhibition from the IC cell. Thus, though the PY activity would be weakly restrained by the LP cell activity, the PY cells might be expected to escape from that inhibition. (The accommodation of LP firing would contribute to PY escape.) Upon escape from LP inhibition, the PY cells in turn inhibit the LP and IC cells. The first of the PY cells to fire are the 'early PYs', or 'PE' cells. These PE cells weakly inhibit the LP cell, causing a decrease in its firing rate, indicated by a dashed outline in the bar diagram of this step. When the three 'late' PYs ('PLs') fire during the latter portion of the indicated PY period, their stronger inhibitory synapses onto the LP cell shut off LP activity completely. Therefore, the observed activity is totally consistent with the synaptic connectivity and synaptic strength data.

Although these results are consistent with the known synaptic connectivity and strength parameters, an equally consistent alternative activity pattern might have been observed. Since the PD/VD group inhibits both the LP/IC group and the PY group, and since the LP/IC group and PY group are mutually reciprocally inhibitory, no data have been presented that would explain why the LP/IC group fires before the PY group. In other words, we would not predict, from the circuitry and synaptic strengths alone, why the pattern did not progress from PD/VD to PY to LP/IC, rather than from PD/VD to LP/IC to PY groups. The resolution of this apparent indeterminacy lies in a property of the PD to PY synapses discovered and characterized by Hartline (1979). It was demonstrated in this study that the PY cells experience a 'rebound delay' to PD cell activity, caused by a hyperpolarizing conductance change activated in the PY cells by the PD cell inhibitory postsynaptic potentials. This delay in the recovery of the PY cells accounts for the pre-emptive escape and firing of the LP and IC cells.

In the final step of our exercise, the circuit is completed by addition of the AB interneuron. All of the resulting effects are understandable in terms of the strong synapses made by the AB cell onto all other neurons in the network. It is strongly electrically coupled to the VD cells, and does not inhibit the PD cells. Thus, the AB and PD cells always fire synchronously.
The AB cell strongly inhibits all other cells in the network. All other cells are shut off during and for a short time after the AB cell burst, as would be expected. Except for the change in the VD cell activity phase, these other neurons maintain the same basic phase relationships as they displayed in the previous step of the exercise. The shift in the VD phase is due to the strong inhibition it receives from the AB cell, which overwhelms the weak electrical coupling between the VD and PD cells. Since the VD cell is also inhibited by the LP and IC cells, but not by PY cells, it is constrained to fire during the PY activity period.

Note that in this last step of the experiment, the VD and PY cell types are simultaneously active; even though the VD cell makes an inhibitory synapse onto one class of PY cells only the three PL cells are inhibited by it. The other five PE cells would not be inhibited by VD cell activity. This is the first and only such contradiction within the structure of this argument. One possible explanation for this PE activity would be the influence of postinhibitory rebound (PIR) from AB, PD and LP cell inhibition. Conceivably, this PIR could overwhelm the VD inhibition, allowing the PE cells to recover and fire during what would normally be a ‘restricted’ period. A full resolution of this problem will require further experimentation on the relative strengths of these two opposing parameters.

One additional effect of adding the AB cell to this circuit is an increase in the overall pattern frequency (note the time base of the physiological recordings). The AB cell acts as an overall frequency controller for the whole network. As well as shutting off all other activity during AB/PD burst, rebound from the AB cell’s strong inhibition serves to accelerate the progression of the bursts within the pattern.

To summarize the results of our thought exercise:

1. The existence of the pyloric pattern results from (a) oscillatory membrane properties of the individual neurons in combination with (b) the multiple reciprocally inhibitory interactions within the network.

2. The precise phase relationship derives from the synaptic connectivity pattern, and depends upon relative synaptic strengths, PIR, rebound delay, and the kinetics of the plateau and BPP generation mechanisms.

3. The overall cycle frequency is determined by the AB interneuron, via its inherent oscillatory behaviour and very strong synapses with the rest of the pyloric neurons.
The gastric mill network

The movements of the teeth in the gastric mill grind up food previously stored in the cardiac sac. To produce 'chewing', the two lateral teeth close and the medial tooth is pulled over them after a short delay. Towards the end of this movement the lateral teeth are opened, followed shortly by resetting of the medial tooth. This pattern is seen in the extracellular recordings of the motor nerves (Fig. 7), where two pairs of powerstroke- and resetting-motoneurons can be observed for the lateral-teeth-neurons (LG, MG versus LPG) slightly ahead of the medial-tooth-neurons (GM versus DG, AM).

Although the gastric network is composed of a smaller number of neurons than the pyloric network, fewer of the cells have identical connectivity which can be lumped together and therefore viewed as a unit in the circuit diagram. This results in considerably more complex circuitry (Fig. 7) (Mulloney & Selverston, 1974; Selverston & Mulloney, 1974). For that reason we took a different approach in the investigation of the pyloric system and attempted to conceptually simplify the circuit. This simplification, together with known intrinsic properties of the cells, we hoped would enable us to make predictions as to how the remaining system would behave after the elimination of a functional group of cells. We have begun to test these predictions using the photoinactivation method and preliminary results appear encouraging.

In the first simplification step, we lumped the synergistic motoneurons together, reducing the number of elements in the circuit. In a second step, we decided to consider only the strongest synaptic connections.

Ideally the measurements of synaptic strength should be accomplished by removing those neurons which might provide serial pathways between the pre- and postsynaptic neurons in question. For the present purpose, however, we found it useful to estimate synaptic strength by recording from three or more neurons intracellularly, looking for direct and indirect effects, reasoning that such effects actually contribute to pattern generation. An example of this kind of data is shown in Fig. 12. Here it can be seen that LPG inhibition by LG is typical of a strong monosynaptic connection. We can also see that although LG and MG are synergists, they are connected with weak inhibitory synapses so that the MG cell is also weakly inhibited. The inhibition of LG onto GM is so weak that it cannot even be seen here.

The various categories of synapses do not lend themselves to a sharp definition (Wiens, 1982) and we are still trying to arrive at a more quantitative picture. Furthermore, we have evidence that, as in the pyloric system,
Fig. 12. Approximation of synaptic strength. Simultaneous recording of more than two cells allows the estimation of possible serial interactions. Two depolarizing current pulses to LG which can be monitored on the extracellular LG trace can be seen to have strong inhibitory effects onto LPG and weaker effects onto the MG cell. Note that the strength of inhibition from LG to MG or GM could be masked by disinhibition via Int I or the E cells.

Synapses release transmitter as a continuous function of the membrane potential, which complicates measurements of synaptic strength even more. With this in mind, the strongest synapses in the gastric circuit appear to be:

Int I to AM, DG, GM and LG
LG to Int I and LPG
MG to LPG
LPG to MG

To ensure complete antagonistic activity between AM, DG and GM, inhibition of DG and AM onto GM has also to be included; this is a borderline case, however, depending on where one draws the line between weak and strong synapses.
Fig. 13. DG is a conditional burster which is activated in the presence of octopamine. A, extracellular recordings from LG, LPG, DG and GM in the control condition show lack of activity in the lateral-teeth system (except for pyloric modulation of LPG). B, block of the stomatogastric nerve stops irregular bursting in the DG. C, bath application of 10^{-4} M octopamine induces slow pacemaker potentials in DG. No signs of postsynaptic potentials can be detected. The effect is fully reversible (not shown).

Almost all of the cellular properties which we have discussed in our consideration of the pyloric rhythm are also present in neurons of the gastric system. The only major exception is the absence of cells which, by our definition, can be considered true endogenous bursters, i.e. cells having negative resistance components which allow them to generate BPPs when completely free of synaptic influences or other extrinsic factors. We have found up to now, however, that the DG and AM neurons are capable of generating BPPs in the presence of octopamine (Fig. 13). Since this amine
has been found in the stomatogastric system (Barker, Kushner & Hooper, 1979) it very probably plays a role in the activity of these two neurons.

_Hypothesis for gastric mill pattern generation_

Fig. 14 presents the gastric circuit that has been simplified by (1) eliminating the weak synapses, and (2) ‘lumping’ together the neurons which normally fire approximately synchronously. Throughout this discussion we assume that the excitatory commissural inputs are intact. The activity levels of the cells in a non-cycling preparation are indicated in the figure: Int 1, the LPG cells and the GM cells will fire tonically, the LG/MG group will be inactive; the DG and AM cells will show weak, irregular bursting. Given the circuitry, the intrinsic properties and these activity levels, we propose that (see Fig. 15):

1. Reciprocal inhibition between Int 1 and the LG/MG group produces an alternating two-phase rhythm in the system probably via a half-centre oscillatory mechanism. These cells show no intrinsic ‘burstiness’, but the oscillations could be aided by PIR and possibly by generation of plateau potentials.

2. The periodic bursts set up in the LG/MG group act to phasically interrupt the tonic firing of the LPG neurons, thus causing the LPGs to burst in antiphase.

3. Inhibitory feedback from LPGs onto the LG/MG group reinforces
Fig. 15. Hypothesis for the mechanism underlying gastric bursting. Complete circuit diagram (upper left) is reduced to a simplified version (upper right). The building blocks of our hypothesis (1 to 6) are reassembled in a ‘thought’ experiment (see numbered points 1 to 6 in the text for explanation).
Table 2. Testing of the hypothesis for the mechanism underlying gastric bursting by photoactivation experiments

<table>
<thead>
<tr>
<th>Cell(s) killed</th>
<th>Role of cell(s) in model</th>
<th>Remaining cells</th>
<th>Predicted results</th>
<th>Experimental results</th>
<th>No. of experiments</th>
</tr>
</thead>
<tbody>
<tr>
<td>GMs</td>
<td>Follower cells</td>
<td>Int 1</td>
<td>Bursting</td>
<td>As predicted</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>LG/MG</td>
<td>Bursting</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>LPGs</td>
<td>Bursting</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>DG/AM</td>
<td>Bursting</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DG/AM</td>
<td>Driven by Int 1 and intrinsic burst mechanism</td>
<td>Int 1</td>
<td>Bursting</td>
<td>As predicted</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>LG/MG</td>
<td>Bursting</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>LPGs</td>
<td>Bursting</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>GMs</td>
<td>Bursting</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LPGs</td>
<td>One-half of reciprocally inhibitory network with LG/MG, Inhibition onto MG is a functional synapse</td>
<td>Int 1</td>
<td>Bursting</td>
<td>Tonic</td>
<td>B in 1 experiment, A in 2 experiments if LGs are slightly depolarized</td>
</tr>
<tr>
<td></td>
<td></td>
<td>LG/MG</td>
<td>Bursting</td>
<td>Tonic</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>DG/AM</td>
<td>Bursting</td>
<td>Silent</td>
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</tr>
<tr>
<td></td>
<td></td>
<td>GMs</td>
<td>Bursting</td>
<td>Bursting</td>
<td></td>
</tr>
<tr>
<td></td>
<td>A. Most likely</td>
<td></td>
<td>Weakly tonic</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>B. Less likely</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Int 1</td>
<td>One-half of monosynaptic reciprocal inhibitory network with LG/MG</td>
<td>MG/LG</td>
<td>Silent</td>
<td>Bursting</td>
<td>A, as expected (LG reduced activity). Note, however, DG and Int 1 kill resulted in B</td>
</tr>
<tr>
<td></td>
<td></td>
<td>LPGs</td>
<td>Tonic</td>
<td>Bursting</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>DG/AM</td>
<td>Irreg. burst</td>
<td>Bursting</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>GMs</td>
<td>Strong tonic</td>
<td>Bursting</td>
<td></td>
</tr>
<tr>
<td>MG/LG</td>
<td>One-half of both reciprocal inhibitory networks formed with Int 1 (monosynaptically) and LPGs (monosynaptically and functionally)</td>
<td>Int 1</td>
<td>Tonic</td>
<td>As predicted</td>
<td>5</td>
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<tr>
<td></td>
<td></td>
<td>LPGs</td>
<td>Tonic</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>DG/AM</td>
<td>Irreg. burst</td>
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<tr>
<td></td>
<td></td>
<td>GMs</td>
<td>Weak tonic</td>
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Table 2. (cont.)

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<thead>
<tr>
<th>Cell(s) killed</th>
<th>Role of cell(s) in model</th>
<th>Remaining cells</th>
<th>Predicted results</th>
<th>Experimental results</th>
<th>No. of experiments</th>
</tr>
</thead>
<tbody>
<tr>
<td>DG</td>
<td>See above</td>
<td>Int 1</td>
<td>Bursting</td>
<td>As predicted</td>
<td>1</td>
</tr>
<tr>
<td>4 GMs</td>
<td></td>
<td>LG/MG</td>
<td>Bursting</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>LPG</td>
<td>Bursting</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>GM</td>
<td>Bursting</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DG, AM</td>
<td>See above</td>
<td>Int 1</td>
<td>Bursting</td>
<td>Tonic</td>
<td>A, as expected</td>
</tr>
<tr>
<td>2 GMs</td>
<td></td>
<td>LG/MG</td>
<td>Bursting</td>
<td>Silent</td>
<td>1</td>
</tr>
<tr>
<td>LPGs</td>
<td></td>
<td>2 GMs</td>
<td>Bursting</td>
<td>Weak tonic</td>
<td></td>
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</table>

this alternation. This reciprocal inhibitory loop might be strong enough to produce alternate bursting even when Int 1 is inactive.

4. The bursts which have been set up in Int 1 trigger delayed bursting in DG and AM. This may be aided by the irregular generation of BPPs or plateau potentials in DG and AM. Under normal conditions, strong synapses from Int 1 keep both the lateral and medial tooth subsystems coordinated.

5. Tonic firing in the GMs would be phasically interrupted by inhibitory inputs from the DG/AM pair and Int 1. They would then fire in antiphase to the DG and AM cells.

6. The irregular ‘burstiness’ of the DG/AM group might make it possible for the medial tooth subset to produce an independent oscillatory pattern even when the lateral teeth subset is not operating.

Testing the hypothesis

We have begun to test the validity of the model by using the photoinactivation technique. Removal of particular elements from the circuit should have predictable results in terms of altering the normal gastric pattern. These predictions are listed in Table 2, along with the actual experimental results. Examples of the effects of the killing experiments on the gastric pattern are also shown in Fig. 16.

The GM neurons, in our model, are viewed as follower cells. Their removal therefore should have no effect on other cells. Although we have only performed this experiment once, and in a preparation that was bursting somewhat irregularly, killing all four GM cells did not prevent the bursting in the LG/MG group. Overall, the rhythm continued in the same manner as before.
Fig. 16. Killing of cells which are thought to be most important for the maintenance of a regular pattern (LG/MG in A and Int 1 in B) results in a breakdown of the pattern leaving the remaining cells in a state predicted by our hypothesis. Note in A a brief hyperpolarization to Int 1 (see text for details).

Removal of the DG/AM group from the medial tooth subset should have similar effects, since these neurons are driven by Int 1 in our model. The lateral teeth subset should show no qualitative effects of the killing and the GMs should continue to burst because of the phasic inhibition they continue to receive. One experiment in which both DG and AM were killed gives support to this notion, as do kills of DG and AM singly: none of these experiments led to marked changes in the pattern.

Inactivation of the LPG cells should be slightly disruptive to the pattern, since the feedback from LPGs onto the MG cell is probably just polysynaptic in comparison with monosynaptic Int 1 to LG/MG connections. These predictions match the observed results since we have to depolarize LG tonically to restore the pattern fully.

Since the LG/MG group, according to our hypothesis, makes up one-
half of the oscillator network, the simultaneous removal of these two neurons should have severe effects on the pattern. We would predict that Int 1 as well as the LPGs, both of which make reciprocal inhibitory connections with the LG/MG cells, would fire tonically (Fig. 16). We would also predict that the GMs would be silent or fire very slowly as a result of the tonic inhibition they would receive from Int 1. Only the DG/AM group might be expected to continue to fire in irregular bursts, because of their intrinsic ‘burstiness’ and the excitation they would be receiving from Int 1. The results again show the predictions to be validated.

Killing the other half of this oscillator, Int 1, should completely terminate the rhythm of the lateral teeth, unless the reciprocal inhibition between the LG/MG cells and the LPGs is strong enough to continue alternate bursting. It is somewhat difficult to know what to expect in the medial tooth system. The intrinsic bursting mechanism in the DG/AM group should be unaffected except for the loss of excitation from Int 1. They should continue to burst, therefore, but at a slightly slower rate. Similarly, the GMs could lose their strong inhibition from Int 1 but continue to be inhibited by DG and AM: they would fire either tonically or with modulation from the DG/AM group. In all cases, GM activity was elevated with modulation out of phase to a mostly reduced DG and AM activity. One experiment in which Int 1 was killed clearly fulfills all the other predictions and presents, up to now, the only single-cell kill which resulted in a complete cessation of the gastric rhythm (Fig. 16). However, in one combined kill of DG and Int 1 the pattern persisted. Further experiments are needed to clarify this apparent contradictory result.

We have tried to isolate oscillatory subsets of the gastric network in the same manner as in the pyloric circuit. Because of the large number of neurons which must be filled and irradiated, this has proved to be extremely difficult. However, in one experiment we have succeeded in killing the DG/AM group, two GMs and the LPGs. Under these conditions only two GMs are left and according to our hypothesis should have little effect on the remaining reciprocally inhibitory connections. This was found to be true, with the LG/MG group (and the two remaining GMs) continuing to burst (although somewhat irregularly). Unfortunately we have no recordings from the interneuron in this experiment. In a similar experiment we killed all of the GMs and the DG neuron. As we would have predicted, the lateral teeth subset continued to produce bursts.

As indicated in Table 2, the experimental results are for the most part in reasonable agreement with the predictions. Thus, the simple model provides a ‘first approximation’ of the gastric CPG mechanism and supports our present view that LG and MG play a crucial role in generating the
gastric pattern. Their reciprocal inhibitory networks with LPG, however, seem to be less important than those with Int 1. The discrepancies between experiment and prediction suggest many more specific experiments, and offer hope that the precise phase relationships and burst lengths may become more predictable as we include consideration of more and more of the 'weaker' synapses as well as extraganglionic connections.

Summary and conclusions

The pyloric and gastric mill patterns result from a complex synthesis of cellular, synaptic and network properties. Considering the range and complexity of the membrane properties in the different neurons, and the dependence of these properties upon synaptic and hormonal inputs from other parts of the central nervous system, the classification of each neuron as either an 'endogenous burster' or as a 'follower' cell would lead to confusion and oversimplification. In the same sense, the categorization of either of these systems as a 'burster-driven' or a 'network-based' CPG would be equally naive. All of these different factors contribute to and reinforce each other in the generation of the stomatogastric patterns.

It is interesting to compare the pattern generation mechanisms in this system with those in another lobster, *Homarus gammarus*. Studies over the last few years by Moulines, Robertson, Nagy and their colleagues have demonstrated significant similarities and differences between stomatogastric 'strategies' in the two genera (Robertson & Moulines, 1981; Nagy, Dickinson & Moulines, 1981). In *Homarus*, the CPGs for pattern generation are also located largely within the stomatogastric ganglion, and many aspects of the circuitry seem to be directly homologous to those in *Pandalus*. However, the commissural ganglia of *Homarus* contain neurons which are (1) endogenously oscillatory, (2) phase-locked to the stomatogastric patterns, and (3) necessary for the maintenance of the stomatogastric rhythms. In other words, these cells (one pair of cells associated with each of the two rhythms) are actually 'members' of the CPG, and appear to play essential roles in driving the rhythms. Moreover, the oscillator neurons associated with the gastric rhythm (the commissural gastric drivers) receive input from fibres in the posterior stomach nerve, which is known to contain mainly sensory fibres (M. Moulines & R. M. Robertson, personal communication). It will be important to compare and contrast the homologies and analogies in 'equipment' and 'strategies' that have evolved in these two genera of lobsters since their divergence. If we hope to uncover general principles of the organization and operation of CPGs, such comparative studies of homologous systems in related genera will serve as a first step.
It is not possible here to compare the stomatogastric system with other CPGs controlling diverse behaviours in other animals. But the reader will observe from the other papers in this volume that there appears to be a large variety of organizational schemes that have evolved for the production of rhythmic motor patterns. This diversity in the structure of CPGs might be interpreted as suggesting a lack of common ‘building block’ mechanisms or general organizational principles. However, just as the biophysical character of a neuron is determined by the particular subset of ionic conductance mechanisms that were selected from all of those upon nature’s grand ‘palette’ (as suggested by Dr Noble, this volume), a rich ‘palette’ of cellular, synaptic and network properties allowing the expression of diverse CPG schemes can be imagined. Although no two ‘creations’ may ever be found to have used identical elements in the same arrangements, several elements on the palette seem to have been very popular throughout evolution. Reciprocally inhibitory cell pairs have been found in circuits generating the leech heartbeat, tadpole and leech swimming as well as cat locomotion (all discussed in this volume). Cells with bursting capabilities have been found in the leech heartbeat system, the snail feeding system, and possibly in the lamprey swimming system. Finally, the involvement of generalized excitation has been demonstrated in Lymnaea feeding, leech swimming, frog embryo swimming and cat locomotion and respiration. Continuing progress in this important area of neurobiology may result in the emergence of other general principles which might help to unify our understanding of CPGs, and perhaps of even more complex parts of central nervous systems.

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