Effects of spike-frequency adaptation on neural models, with applications to biologically inspired robotics

by

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A thesis submitted in conformity with the requirements
for the degree of Doctor of Philosophy
Graduate Department of Aerospace Engineering
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“I hate quotations.” —Ralph Waldo Emerson
Thesis title: Effects of spike-frequency adaptation on neural models, with applications to biologically inspired robotics

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Abstract

Animals are impressive biological machines, and their ability to handle unstructured environments is something roboticists wish to emulate. The behavioural competence of animals derives largely from the functioning of their nervous systems. Mathematical modelling of the functioning of neurons may enable us to extract useful principles from biology to be applied in robotics. Here, several systems with relevance to biologically inspired robotics are analyzed. The qualitative dynamics of a biological property called spike-frequency adaptation are added to existing analog neural models, and analysis shows the conditions under which the augmented model can generate oscillatory solutions. A network of these augmented analog neurons is then used to generate a walking gait for a six-legged robot in such a way that the system recovers rapidly from perturbations to the legs. The dynamics of oscillations arising in two coupled populations of integrate-and-fire neurons are studied; an analysis of the system provides good predictions of the oscillatory period and the range of coupling strengths for which oscillations will occur. A signal-processing phenomenon known as noise-shaping, wherein noise in a system is shifted out of the low frequencies up into higher frequency ranges, is demonstrated in networks of integrate-and-fire and conductance-based neurons: it is shown that spike-frequency adaptation provides certain signal-processing advantages in such networks. The effect of spike-frequency adaptation on the variability in integrate-and-fire neurons' firing records is analyzed.
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Chapter 1

Introduction and Background

1.1 Motivation

When watching an animal moving around in its environment, it is always impressive to consider
the fluidity of its movement, the complexity of the decision-making it exhibits, and the speed
with which it reacts to new situations. Considered simply as a device, biological organisms are
remarkable machines. They have been tuned by aeons of natural selection to be able to handle a
complex world, moving over difficult surfaces and making rapid and effective decisions in response
to a barrage of sensory stimuli.

Robots are not currently able to match this level of performance, and any roboticist observing
an animal feels a sense of awe, and of envy. We would like to be able to build devices as robust
and flexible as animals—imagine a robot capable of scampering around on the surface of Mars
and gathering rock samples as competently as a squirrel collects and caches nuts. This sense of
wonder has led, in recent years, to an increased interest in understanding the operation of these
biological "machines," with the goal of extracting principles that may be applied in robotics.
The idea is essentially to perform a sort of reverse engineering: the mechanisms that generate
these behaviours exist, and are available for study, so why not use this information in future
designs? This approach has led to formation of a new field, called variously "biological robotics."
"biologically inspired robotics," or "biorobotics."

Of course, the concept of emulating animal forms when building artificial devices is nothing
new. For centuries, engineers have looked to nature for inspiration for the design of various au-
tomata. Biologically inspired robotics may be seen as simply a revival of this tradition. However,
the tremendous advances in modern neuroscience offer the possibility that we may be able to
move beyond emulating the external forms of animals, and start to gain some understanding of
the mechanisms which underlie their behaviour.

Strictly speaking, everything about an animal's physiology contributes to its behaviour. It is
clear, however, that the key player in generating behaviour is the nervous system, with its huge networks of coupled, signal-processing cells, the neurons. Most workers have focused on neurons as the keys to understanding animal behaviour, and I will do so in this thesis. It seems clear that if we are to transfer insights from biological "wetware" to artificial hardware, we will require an understanding of the operation of nervous systems at a mathematical level. My thesis work has thus concentrated on mathematical models of neurons. Clearly we are nowhere near truly understanding how nervous systems operate: the work presented here consists of a few problems to which mathematical and computational techniques may be successfully applied. In particular, I consider the effects of a common property of biological neurons, spike-frequency adaptation, on various neural models, showing how it alters the dynamics of neurons in ways which alter their oscillatory behaviour, their signal-processing capabilities, and their response to noise. My hope is that work such as this may serve as a starting point for future attempts to transfer information between the realms of biology and robotics.

The following sections will provide an introduction to the structure and function of neurons, then proceed to discuss a few of the common mathematical models used to represent them. The chapter will conclude with an overview of the contents of the remaining chapters.

### 1.2 Nervous systems

An organism's nervous system is simply the complete set of neurons (and associated support cells) that it uses to process signals and produce behavioural responses. In vertebrates, this is divided into two main sections: the central nervous system (CNS), consisting of the brain and spinal cord; and the peripheral nervous system (PNS), consisting of the nerve cells extending out from the spinal cord into the body, carrying signals to and from the brain.

The most striking feature of nervous systems is the massive number of individual neurons involved. Although the simplest nervous systems have a relatively small number of individual cells, the number grows rapidly as the size and "complexity" (a word I will not attempt to define here) of the organism increases; see Table 1.1.

In addition to neurons, nervous systems contain large numbers of glial (or neuroglial) cells, outnumbering neurons by about nine to one [5]. The glia serve a number of support functions for the neurons: they act as a structural scaffold; certain glial cells produce the myelin sheath which coats the neural axons (see section 1.3.3); and they provide active buffering to maintain the required ionic concentrations in the vicinity of neurons. It is possible that glial cells may have important signal-processing properties [6], but the work presented here will focus exclusively on neural modelling, leaving aside the influence of glial cells.

A great deal is known about the neuroanatomy of various organisms (the structure, interconnections, and functional roles of different areas of the central nervous system). Rather than
Table 1.1: Numbers of neurons in various species. Exact numbers of neurons are difficult to establish, so these numbers are approximate; sources for the figures are given in the third column. The figure for the number of neurons in the human brain is particularly variable: the most commonly cited figures range from 100 billion to 1 trillion.

discussing anatomy here, the remainder of the chapter will concentrate on the basic structure and operation of individual neurons (sections 1.3 and 1.4), and on some of the mathematical models that have been proposed (section 1.5).

There is a vast literature on neuroscience. Good introductions can be found in [7, 8, 9, 10, 11, 12]; any of these will provide a starting point for the interested reader. And these introductory remarks have drawn extensively on these sources. Zigmond et al. [7] and Kandel and Schwartz [9] are particularly comprehensive and clearly written. Arbib [8] provides an encyclopaedia-style reference, with short review pieces on a wide range of topics.

The sheer variety of cells and organisms in the biological world means that it is difficult to make definitive statements of the form: "Neurons always display property X." Inevitably, there will be examples of cells that do not display X, or indeed display the opposite of X. The material presented here will address the most common properties rather than dwelling on the exceptions.

### 1.3 Neuron morphology

There are many different varieties of neurons, perhaps as many as a thousand distinct types [5]. However, they may all be placed into one of three broad categories: sensory neurons; motor neurons; and interneurons. Sensory neurons transduce external energies (light, mechanical vibrations, heat, and so on) into neural electrical signals. Motor neurons stimulate muscle spindles and thus cause movements in the organism’s body. Interneurons do not have direct connections to either sensory or motor systems: rather, they receive their inputs from, and send their outputs to, other neurons. (For those with a background in artificial neural network models: sensory neurons, interneurons, and motor neurons are roughly equivalent to input, hidden layer, and output nodes in artificial neural networks.)
Table 1.2: Functional roles of the four main neural regions. These are, of course, not the only functions these portions of the neuron carry out, but they are the most significant in terms of signalling in the nervous system.

Figure 1.1 shows stylized representations of the main types of neuron. Neurons are divided into four main morphologically distinct regions (to be discussed in the next four sections): the dendrites; the soma (or cell body); the axon; and the axon terminals. Table 1.2 summarizes the functional roles of each of these regions. The flow of signals through a neuron is typically as follows:

- incoming signals (from sensory receptors or from the axons of other cells) are received at the dendrites
- the signals from the dendrites are integrated at the soma
- when the cellular membrane voltage at the beginning of the axon (the axon hillock) rises far enough, an action potential (see section 1.4.2) is generated
- the action potential, a brief pulse of increased membrane voltage, travels down the axon
- when an action potential (or "spike") arrives at the end of the axon, it triggers the release of chemicals known as neurotransmitters, which diffuse across a gap (the synapse) between the axon terminals and the dendrites of another cell, influencing the generation of action potentials in this second cell

The electrical properties of the neural membrane will be discussed in section 1.4. In the following sections, I briefly describe each of the four main divisions found in the typical neuron.

### 1.3.1 Dendrites

The dendrites are a set of fine, highly branched structures which convey inputs from other neurons to the cell body; typical neurons will receive dendritic inputs from hundreds or thousands of other cells [5, 7]. These inputs arrive in the form of neurotransmitters diffusing across a gap (the synapse) between the dendrite and the axon terminal of an incoming axon. These neurotransmitters are chemicals which affect the electrical potential of the cell (increasing or decreasing it; see section 1.4.3), and this voltage change is conveyed along the dendrite to the
Until recently, dendrites were viewed as passive "cables" conveying voltage changes towards the cell body, but it is now known that some dendrites are active elements that generate pulses similar to the action potential produced in the axon [14, 15].

### 1.3.2 Soma

The soma, or cell body (also called the perikaryon), contains the cell's nucleus and much of the biochemical machinery of the neuron. It is where gene expression takes place, and where inputs from the dendritic tree are combined.

Like all cells, the neuron is surrounded by a cellular membrane. This membrane has an electrical potential difference across it, arising from differences in ionic concentrations on the interior and exterior surfaces of the membrane. This potential is the key to the neuron's ability to process signals. See section 1.4 for more detail; briefly, the dendritic inputs are integrated by combining their effects on the soma's membrane voltage. When the membrane voltage rises far enough, an action potential is generated in the axon.

### 1.3.3 Axon

The axon is a tubular structure that emerges from the cell body and extends for some distance away from it. Axons generally reach much farther away from the soma than the dendrites do, and some axons may be over one meter long. (For example, in humans there are single axons that extend from the hands and feet to the spinal cord.) At the end of the axon, many fine branches called axon terminals (or presynaptic terminals, or synaptic boutons) emerge from the axon and connect it to other cells.

The axon's functional role is to propagate the neuron's output signals from the cell body to the axon terminals, where contact is made with other cells (either neurons or muscle cells), which may be influenced by the output. The neuron's output takes the form of action potentials. brief pulses of high membrane voltage that propagate in a self-regenerating manner down the axon's length. Many axons have an insulating sheath made of a fatty substance called myelin; this insulation is broken at intervals, and the exposed sections of bare axon are called the nodes of Ranvier (see Figure 1.1). The myelin sheath greatly increases propagation speed in the axon, by allowing the action potential to "jump" from one node of Ranvier to the next, a process called saltatory conduction: see [13, 16] for more detail.

### 1.3.4 Axon terminals

The axon terminals are specialized bulbs at the ends of fine branches emerging from the end of the axon. They rest near the cellular membranes of other cells (muscles or other neurons), and
affect the state of these other cells when action potentials arrive after being propagated down the axon.

Motor neurons have axon terminals that attach to muscle fibres (see Figure 1.1), and the effect of incoming action potentials is to stimulate these fibres to contract. The axon terminals of interneurons typically make contact with the dendrites of other neurons (axo-dendritic connections), but it is also possible for them to contact the cell bodies (axo-somatic connections) or the axons (axo-axonic connections) of other cells [17].

The small distance between the axon terminal and the cell it influences is called the synapse (or synaptic cleft); see Figure 1.5 on page 13. The cell whose axon terminal is doing the signalling is therefore known as the presynaptic cell, while the cell which is being influenced is called the postsynaptic cell. There are two main types of synapse: chemical and electrical. In chemical synapses, the presynaptic cell exudes neurotransmitters which affect the postsynaptic cell. Electrical synapses have direct electrical coupling through membrane-bound proteins called gap junctions, through which the two cells can directly exchange ions.

1.4 Neuron electrical properties

1.4.1 Membrane potential

Neurons, like most other cells, have an electrical potential difference—called the membrane potential or membrane voltage—between the inside and outside of their cellular membrane, maintained by differences in the distribution of ions on the interior and exterior membrane surfaces. The main ions which vary across the cellular membrane are: potassium (K⁺); sodium (Na⁺); chloride (Cl⁻); and calcium (Ca²⁺).

The cellular membrane is selectively permeable. Most of the membrane consists of a lipid bilayer which prevents almost all substances from crossing, but bound into this are proteins called ion channels that span the membrane and allow ions to travel from the extracellular fluid to the cytoplasm. Ion channels are typically ion-specific, only allowing ions of a particular species to pass through. Some ion channels are voltage-dependent, varying their permeability as the membrane voltage changes. Figure 1.2 shows a section of the cellular membrane, with ion channels indicated.

There are two gradients which act to drive ions across the membrane: electrical potential, and concentration. See Figure 1.3. Positive ions flow to regions of negative electrical potential, while negative ions seek positive potentials. At the same time, ions tend to flow from high concentration to low concentration regions. A steady state is reached when the fluxes induced by the electrical potential and concentration gradients are equal, and ions flow out of the cell as quickly as they flow in. The voltage at which this occurs is called the equilibrium potential (also known as the Nernst potential), and is given by the Nernst equation,
Figure 1.2: A section of a neuron's cellular membrane. The structures shown spanning the membrane are ion channels, each of which is associated with a particular species of ion. Next to each channel, typical concentrations of its affiliated ion are given (in mM, except for intracellular Ca\(^{2+}\)). The values \(E_{Na}\), \(E_K\), and so on are the equilibrium potentials for each ion (in mV). (In the text, the more common notation \(V_{Na}\), \(V_K\), and so on has been used, as in equation (1.1)). The Na\(^{+}\)-K\(^{+}\) pump is shown at the bottom of the figure; this is an ion pump that acts to keep the intracellular and extracellular concentrations of sodium and potassium from approaching their equilibrium values; the pump expends energy to bring K\(^{+}\) into the cell, while removing Na\(^{+}\).

Figure 1.3: Gradients affecting ion flows across the neural membrane. The ion—here, potassium (K⁺)—is at a higher concentration inside the cell than outside, and thus tends to flow down the concentration gradient and out of the cell. Opposing this is the voltage difference across the membrane: the inside is more negative than the outside, so that the positive K⁺ ions tend to flow down the voltage gradient and into the cell. At the equilibrium (Nernst) potential, the flows induced by the two gradients are balanced, and there is no net change in concentration. From *Fundamental Neuroscience*, edited by Zigmond et al. [7] © 1999 by Academic Press. Used with permission by Academic Press.

\[ V_{\text{ion}} = \frac{RT}{zF} \ln \frac{[\text{ion}]_{\text{out}}}{[\text{ion}]_{\text{in}}} \]  

where: \( R \) is the gas constant (8.315 J K⁻¹ mol⁻¹); \( T \) is the absolute temperature; \( F \) is Faraday's constant (96,485 C mol⁻¹); \( z \) is the charge of the ion; and \([\text{ion}]_{\text{out}}\) and \([\text{ion}]_{\text{in}}\) are the concentrations of the ion outside and inside the cell, respectively.

If the membrane were permeable to only a single ion species, the membrane potential would be exactly the equilibrium potential for that ion; for example, a membrane permeable only to
potassium would have an equilibrium potential of $V_K = \frac{RT}{zF} \ln(3/135) = -102$ mV. In fact, the membrane is permeable to multiple species of ions, and the membrane voltage is the result of the combined effects of all of them, weighted by the relative permeability of the membrane to each ion. The resulting membrane voltage varies among different types of neurons, from about -75 mV to -40 mV [7].

Since the membrane potential sits at a value which is not equal to the equilibrium potential for any particular ion species, the flows of ions into and out of the cell do not balance. For example, a cell with a membrane voltage of -60 mV is above the equilibrium potential for potassium, and below the equilibrium potential for sodium. This means that $K^+$ ions flow out of the cell while $Na^+$ ions flow into it, each following their concentration gradient. To maintain the high potassium and low sodium concentrations inside the cell, proteins called ion pumps act to transport ions against their concentration gradients, expending energy to do so. The $Na^+-K^+$ pump brings potassium back into the cell while removing sodium, obtaining the energy for this from the hydrolysis of ATP (as shown in Figure 1.2). Other ion pumps perform similar operations for the other ionic species.

Early neurophysiologists discovered this potential difference across the cellular membranes of neurons, and described the neuron as being polarized. This has led to the following use of terminology, which can be rather confusing initially: increasing the membrane voltage is called depolarization (making the cell less polarized, i.e. moving it away from its negative resting value and up towards 0 mV), while decreasing the voltage is called hyperpolarization (polarizing the cell further, i.e. moving it to a more negative value, further away from 0 mV). Synaptic or other influences on a neuron are often described in this language, as either depolarizing or hyperpolarizing. Equivalent terms for synaptic inputs are excitatory (depolarizing) and inhibitory (hyperpolarizing).

1.4.2 Action potential generation

The action potential is a brief, large-magnitude increase in the membrane potential: typical action potentials last 1 to 10 ms, and increase the membrane voltage by 70 to 110 mV [5]. Action potentials are initiated at the axon hillock, and propagate down the axon, being actively regenerated as they travel. Figure 1.4 shows a typical sequence of action potentials from a real neuron.

The main players in action potential generation are voltage-dependent sodium and potassium ion channels. As the membrane voltage (described in section 1.4.1) rises to a certain level (which varies from neuron to neuron), voltage-dependent $Na^+$ channels open, allowing more sodium ions to flow into the cell. Past a certain threshold voltage, this becomes a positive feedback process: the voltage increase induced by the influx of $Na^+$ ions causes more sodium channels to open, causing even more depolarization, and thus the membrane voltage "explodes" upwards.
Figure 1.4: Voltage trace from a neuron in the rat somatosensory cortex, artificially stimulated with a current of 0.7 nA. Unpublished data, recorded by David Pinto (Brown University and Boston University) from a neuron in cortical layer II/III of the somatosensory cortex of an adult rat (coronal slice at body temperature). Copyright ©1999. David Pinto, used with permission.
The action potential is terminated by two effects. First, the sodium channels spontaneously inactivate, reducing the influx of sodium ions. And second, the high membrane voltage activates voltage-dependent potassium channels, which open and permit K⁺ ions to flow out of the cell; this has the effect of hyperpolarizing the membrane.

After an action potential, the membrane voltage generally drops to below its resting value before recovering. Immediately after a spike is generated, the neuron enters a phase known as the refractory period, during which it is difficult or impossible to elicit another action potential. During the absolute refractory period, no amount of stimulation will generate a spike; this period is typically 2-3 ms in length [18]. Following this is the relative refractory period, during which a higher stimulus level is required to elicit an action potential than would be required in a resting neuron: this period may last on the order of 5-10 ms [18]. Note that the absolute refractory period places an upper limit on the maximum firing rate a neuron may achieve, regardless of the input intensity, meaning that neurons cannot act as high-frequency devices: with an absolute refractory period of 2 ms, for example, the spiking frequency is limited to less than 500 Hz.

The dynamics of action potential generation are well described by the Hodgkin-Huxley model: see section 1.5.2.

1.4.3 Synaptic coupling

In a chemical synapse, the increased membrane voltage associated with an incoming action potential causes synaptic vesicles to release their contents, chemicals called neurotransmitters, into the synaptic cleft. These chemicals diffuse across the gap and attach to receptors on the other cell, causing changes in the membrane voltage of the postsynaptic cell: see Figure 1.5.

1.4.4 Spike-frequency adaptation

Every chapter in this thesis relates, in one way or another, to the effects of a behaviour displayed by many neurons, called spike-frequency adaptation. Rather than responding to a constant stimulus with a constant rate of firing (or a constant average rate, allowing for noise), spike-frequency adaptation causes a neuron to respond with less and less frequent action potentials as the input is sustained. The main mechanism underlying this effect is thought to be calcium-dependent potassium currents [20, 21]: each action potential triggers an influx of Ca²⁺ ions, and the accumulation of calcium triggers K⁺ currents that slow down the rate at which the neuron approaches the threshold for action potential generation. Each neuron, then, maintains a trace of its past activity in the form of its current internal level calcium ions: this trace decays over time, as the Ca²⁺ ions leak back to their resting levels in the absence of new action potentials. Sections 2.3, 4.3 and 5.5 all discuss neural models incorporating, at increasing levels of biophysical detail, the dynamics of spike-frequency adaptation.
Figure 1.5: Structure of a chemical synapse. When an action potential arrives at the axon terminal of the presynaptic cell, the associated depolarization causes synaptic vesicles to release their contents (the neurotransmitters) into the synaptic cleft, a process called exocytosis. When the neurotransmitters bind to receptors on the postsynaptic cell, they have an excitatory or inhibitory effect on the postsynaptic cell’s membrane voltage, depending on the type of neurotransmitter involved. From Molecular Cell Biology by Lodish et al. [19] ©1986, 1990, 1996 by Scientific American Books, Inc. Used with permission by W. H. Freeman and Company.
### Table 1.3: Summary of some of the neural models that have been proposed. The table indicates two sets of divisions for the models: static vs. dynamic, and rate vs. spiking. Static models do not have internal dynamics, simply producing an output for a given input: dynamic models have internal states governed by some appropriately chosen dynamics. Rate models generate analog values as their output, representing the firing rate of a neuron (or the collective average firing rate of a group of neurons); spiking models produce individual spikes (of varying degrees of complexity) as their output. Binary units refer to highly simplified models in which the neuron’s state is given as either excited (1) or resting (0): it is also possible to employ dynamic binary models, where the state remains binary but is governed by some set of internal dynamics. Linear units use a linear relationship to map inputs to outputs. Nonlinear units replace this mapping with some nonlinear function, typically some form of sigmoid. The next five models in the table (analog neurons through to biochemical compartmental models) are discussed in the text. Note that the final two entries, although they involve actual biological neurons, are still “models”: isolated neurons in vitro, or even in slices sectioned out of a brain, do not have precisely the same behaviour observed in neurons inside a fully functioning nervous system.

### 1.5 Neural models

Various mathematical models have been used to represent neurons. One major division can be made between static and dynamic models. Static models (used mainly in artificial neural network (ANN) research) act as functions mapping inputs into outputs, while dynamic models have internal states governed some set of dynamical (differential or difference) equations. My thesis work has been entirely on the dynamic side of this division. Within dynamic models, a distinction may be made between rate-based (or analog) models and spiking models. In rate-based models, the output of each neuron is considered to be the rate (frequency) with which it produces spikes; this is a real-valued quantity, and the individual spiking times are not considered. Spiking models generate individual action potentials as their output. In this work, I have used both rate-based and spiking models. Table 1.3 lists a few common models, arranged approximately in order of increasing complexity.

<table>
<thead>
<tr>
<th>Model Type</th>
<th>Models</th>
</tr>
</thead>
<tbody>
<tr>
<td>Static</td>
<td>Binary units, Linear units, Nonlinear units</td>
</tr>
<tr>
<td>Dynamic</td>
<td>Analog neurons, Rate</td>
</tr>
<tr>
<td></td>
<td>Integrate-and-fire [1-D], FitzHugh-Nagumo [2-D], Hodgkin-Huxley [4-D]</td>
</tr>
<tr>
<td></td>
<td>Biochemical compartmental models [many-D]</td>
</tr>
<tr>
<td></td>
<td>Neurons in slice preparations</td>
</tr>
</tbody>
</table>

---

*Table 1.3: Summary of some of the neural models that have been proposed.*
1.5.1 Compartamental models

Some of the most elaborate neural models are based on breaking the neuron into many coupled regions known as compartments, then modelling the ionic flows and conductances in each compartment, along with appropriate coupling terms between compartments [22, 23]. The model by Traub et al. [24], for example, uses 19 compartments to represent a pyramidal cell in the CA3 region of the guinea pig hippocampus. Each compartment has up to six active ionic conductances, controlled by up to 10 ion channel variables, leading to a system with literally hundreds of dimensions. None of the models used in this thesis approach this level of detail.

The individual compartments in compartmental models often obey dynamics similar to those described in the next section.

1.5.2 Conductance-based (Hodgkin-Huxley) models

Hodgkin and Huxley [25], in work that ultimately won them the Nobel prize, carried out a series of experiments on the giant axon of the squid, measuring the conductances (inverse of resistance) associated with the Na\(^+\) and K\(^+\) ions under varying voltage conditions. They then constructed a model that fit the observed behaviour using a small number of dynamical variables: see Weiss [18] and Koch [26] for useful discussions.

The model consists of an equation for the membrane potential.

\[
C \frac{dV}{dt} = I_o + I_{Na} + I_K + I_L. \tag{1.2}
\]

where \(C\) is the membrane capacitance, \(I_o\) is the applied current, and

\[
I_{Na} = g_{Na}m^3h[V_{Na} - V]. \tag{1.3}
\]
\[
I_K = g_Kn^4[V_K - V]. \tag{1.4}
\]
\[
I_L = g_L[V_L - V]. \tag{1.5}
\]

\(I_{Na}\) is the current associated with flows of sodium ions across the membrane, with maximum conductance \(g_{Na}\) and reversal (Nernst) potential \(V_{Na}\) (see section 1.4.1). Similarly, \(I_K\) is the potassium current with conductance \(g_K\) and reversal potential \(V_K\), and \(I_L\) is a leak current with conductance \(g_L\) and reversal potential \(V_L\). The sodium and potassium conductances are modulated by the gating variables \(m, h,\) and \(n\), each of which is in the range [0, 1] and represents the degree to some hypothetical voltage-sensitive gate is open (0 being fully closed and 1 being fully open). The exponents on \(m\) and \(n\) in equations (1.3) and (1.4) represent the assumption that 3 and 4 such gates, respectively, must be simultaneously open for maximum conductance to occur. The gating variables are assumed to obey first-order kinetics; gating variable \(x\) makes
transitions from closed to open with rate constant $\alpha_x(V)$, and from open to closed with rate constant $\beta_x(V)$:

$$(1 - x) \xrightarrow{\alpha_x} x \xrightarrow{\beta_x} (1 - x). \quad (1.6)$$

These kinetics correspond to the following set of differential equations:

$$\frac{dm}{dt} = \alpha_m(V)(1 - m) - \beta_m(V)m = [m_\infty(V) - m]/\tau_m(V) \quad (1.7)$$

$$\frac{dn}{dt} = \alpha_n(V)(1 - n) - \beta_n(V)n = [n_\infty(V) - n]/\tau_n(V) \quad (1.8)$$

$$\frac{dh}{dt} = \alpha_h(V)(1 - h) - \beta_h(V)h = [h_\infty(V) - h]/\tau_h(V). \quad (1.9)$$

where $x_\infty \equiv \alpha_x/(\alpha_x + \beta_x)$ and $\tau_x \equiv 1/(\alpha_x + \beta_x)$. Thus, each of these variables asymptotically approaches the value $x_\infty(V)$, with time constant $\tau_x(V)$.

The voltage-dependent rate constants were obtained by fitting curves to experimentally measurable currents and conductances (see [18] for more information). The original equations obtained for the squid giant axon were [18, 25]:

$$\alpha_m(V) = \frac{-0.1(V + 35)}{\exp[-0.1(V + 35)] - 1} \quad (1.10)$$

$$\beta_m(V) = 4 \exp[-(V + 60)/18] \quad (1.11)$$

$$\alpha_h(V) = 0.07 \exp[-0.05(V + 60)] \quad (1.12)$$

$$\beta_h(V) = \frac{1}{\exp[-0.1(V + 30)] + 1} \quad (1.13)$$

$$\alpha_n(V) = \frac{-0.01(V + 50)}{\exp[-0.1(V + 50)] - 1} \quad (1.14)$$

$$\beta_n(V) = 0.125 \exp[-0.0125(V + 60)] \quad (1.15)$$

Using these expressions to find $x_\infty$ and $\tau_x$ for each of the variables yields the plots in Figure 1.6.

The Hodgkin-Huxley model captures the dynamics of action potential generation, as follows. Imagine that we have a cell sitting near its resting voltage, $V_{\text{rest}} < 0$. For $V < 0$, $m_\infty \to 0$, $h_\infty \to 1$, and $n_\infty \to 0$. From (1.3-1.4), we see that the sodium current $I_{Na} = g_Na m^3 h[V_{Na} - V] \to 0$ as $m \to 0$, and the potassium current $I_K = g_K n^4[V_K - V] \to 0$ as $n \to 0$. Near the resting voltage, then, the cell's behaviour is dominated by the leak current $I_L$, and any applied current $I_0$. If we now apply a depolarizing input to the cell, the membrane voltage increases towards 0 mV. This change has the effect of increasing $m_\infty$, and the variable $m$ rises as it tracks $m_\infty$; this leads to an
Figure 1.6: Asymptotic values (top) and time constants (bottom) of the channel variables in the Hodgkin-Huxley equations, as functions of the membrane voltage, $V$. The dynamics of each channel variable is of the form $\dot{x} = [x_\infty(V) - x]/\tau_x(V)$, for $x \in \{m, h, n\}$. 
increase in the magnitude of $I_{Na}$. $I_{Na}$ is positive for $V < V_{Na}$, and since $V_{Na} > 0$ (typically), the effect of increasing the sodium current at this point is to further depolarize the cell. This leads to a positive feedback cycle in which the increasing voltage leads to a larger sodium current (as $m$ increases), which increases $I_{Na}$ and causes the voltage to increase even faster; this generates the upward spike of the action potential. The positive feedback is terminated by two factors as $V$ increases: $h_{\infty}$ decreases and $h$ falls, which decreases $I_{Na}$ ($I_{Na} \to 0$ as $h \to 0$); and $n_{\infty}$ increases, causing an increase in the magnitude of $I_K$. $I_K$ is negative for $V > V_K$, and since $V_K < 0$, the effect of the potassium current during the upward spike is to hyperpolarize the cell, pulling the voltage back down towards the resting potential. Under the influence of the potassium current, the cell's voltage typically "overshoots," being reduced to somewhere below the original resting potential. At this point both the potassium and sodium currents are once again inactivated, and the cell converges back to its resting state. If a sustained depolarizing stimulus is applied, the cell will generate another action potential, and repeat this cycle with a frequency dependent on the magnitude of the stimulus current: if no sustained depolarization is present, the cell will remain in its resting state indefinitely, until another stimulus generates enough depolarization to start the positive feedback cycle leading to an action potential.

One obvious question is why the increase in $m_{\infty}$ and the decrease in $h_{\infty}$ do not simply cancel one another out. preventing the increase in $I_{Na}$ and shutting down the positive feedback cycle before it can begin. The answer lies in the time constants: $\tau_m \ll \tau_h$, so $m$ increases towards $m_{\infty}$ much more quickly than $h$ falls towards $h_{\infty}$. The upward stroke of the spike takes place in the time window during which $m$ has increased but $h$ and $n$ have not yet "caught up."

The Hodgkin-Huxley model was specific to the squid giant axon, but the same basic formulation is still commonly used as a model for other neurons. These are called "conductance-based" models; recent examples include [21, 27]. The spiking output from one such conductance-based model (from [21]) is shown in Figure 1.7.

### 1.5.3 FitzHugh-Nagumo model

The FitzHugh-Nagumo equations [28, 29] represent a reduction of the four-dimensional Hodgkin-Huxley dynamics (discussed in section 1.5.2) to a two-dimensional system. (Helpful discussions of the FHN equations are found in [30] and [31].) The first simplification is carried out by noting that, since $\tau_m \ll 1$ s, $m \approx m_{\infty}$; taking $m(t) = m_{\infty}(V)$ reduces equation (1.7) to an algebraic relationship. The next step is to take $h(t) = h_\sigma$. This is not biophysically realistic, but the reduced system still retains the desired characteristics: the system has a single fixed point for small inputs; it is excitable in the sense that a large perturbation can cause a large-magnitude excursion through phase space (corresponding to a single spike) before it returns to the fixed point; and there is a bifurcation to an oscillatory state for some sufficiently high input level.

A further simplification is then made, replacing the remaining $V$ and $n$ equations with the
Figure 1.1: Spiking in a conductance-based (Hodgkin-Huxley type) model. The plot shows membrane voltage versus time for a conductance-based model proposed by Wang [21]; see section 5.5.
CHAPTER 1. INTRODUCTION AND BACKGROUND

Figure 1.8: Schematic of integrate-and-fire (IF) model. An input current $I(t)$ is applied to a parallel resistance ($R$) and capacitance ($C$). The output is a membrane voltage $V$, which is reset when some threshold $V_{th}$ is reached. For finite $R$, this is known as a "leaky" IF neuron. As $R \to \infty$, the leak term disappears, and the unit becomes a "perfect" IF neuron.

qualitatively similar dimensionless equations

$$\frac{dv}{dt} = v(a - v)(v - 1) - w + I$$  \hspace{1cm} (1.16)

$$\frac{dw}{dt} = b v - \gamma w.$$  \hspace{1cm} (1.17)

where $0 < a < 1$, $b > 0$, and $\gamma > 0$.

The equations of the FitzHugh-Nagumo model appear very different from the original Hodgkin-Huxley dynamics, but the model retains the correct qualitative behaviours while being much more analytically tractable.

1.5.4 Integrate-and-fire model

The integrate-and-fire (IF) model was first discussed by Lapicque [32] (a helpful discussion is found in [33]). It is a very simple model that treats the cellular membrane as a parallel capacitance and resistance to which an input current is applied; see Figure 1.8. This leads to the differential equation

$$C \frac{dV}{dt} = -\frac{V}{R} + I(t)$$  \hspace{1cm} (1.18)

for the membrane voltage $V$. For finite $R$, the model is called a "leaky" integrate-and-fire neuron, since the $-V/R$ term makes the voltage into a leaky integrator of the input current; this simulates the presence of leakage currents passing through the membrane. As $R \to \infty$, the unit becomes a nonleaky or "perfect" IF model.
Spiking in the IF model is simulated by resetting the voltage to some value $V_{\text{reset}}$ when a threshold value $V_{\text{th}}$ is crossed. At this point, the neuron is considered to have produced an action potential. No attempt is made to replicate the action potential shape: in IF models, action potentials are instantaneous, point events, generally written as $\delta$-functions in mathematical descriptions.

Although it greatly simplifies the dynamics of real neurons, the IF model captures the two most crucial features of neural spiking dynamics: a prethreshold, integrating phase, followed by the generation of stereotypical, brief impulses once threshold is reached [33]. In this thesis, IF models will be discussed in chapters 4, 5, and 6.

1.5.5 Hopfield's analog model

In [34], Hopfield presents an analog neural model that uses essentially the same equation as the integrate-and-fire model.

$$C \frac{dx}{dt} = -\frac{x}{R} + I(t).$$

(1.19)

where $x$ may be seen as the mean membrane potential of a neuron (though Hopfield also discussed other possible interpretations, see [34]). $C$ and $R$ are capacitance and resistance values, and $I(t)$ is an input current. Rather than producing individual spikes with a threshold mechanism, the output is taken to be a firing rate, calculated as a nonlinear function $y = f(x)$, where $y$ is the firing rate output and $f(x)$ is called the firing rate function. Often a sigmoidal firing rate function is used, such as

$$f(x) = \frac{1}{1 + e^{-x}}.$$ 

(1.20)

Hopfield was able to show that coupled networks of these analog neurons possessed fixed-point attractors, and that desired attractors could be created using a simple algorithm [34]. This enables such networks, now often called “Hopfield networks,” to perform associative memory tasks: by inserting an attractor corresponding to each desired “memory,” the network will perform reconstruction on corrupted versions of the original pattern, usually converging to the stored pattern which the corrupt version most closely resembles. (See Hertz et al. [35] for a good discussion of the applications of the Hopfield model.)

1.6 Thesis overview

This document will address the following sequence of topics:

- A technique for adding a version of spike-frequency adaptation to any existing analog
neuron model is described; the resulting models are called "phasic analog neurons." When two phasic analog neurons are coupled in mutual inhibition, oscillatory solutions can emerge where otherwise only fixed-point solutions would be possible. An application of techniques from nonlinear dynamics reveals the conditions under which oscillations occur, and the stability properties of the oscillatory cycles which arise. Such a two-cell system is a very simple model of a common biological mechanism known as a central pattern generator. [Chapter 2]

- As an application of the simple pattern generators analyzed above, a network of phasic analog neurons is used to generate the gait for a hexapod walking robot. Simple insights from biology help structure the network, leading to an architecture that generates the appropriate phase relationships among the six legs, and recovers the gait quickly when the legs are perturbed. [Chapter 3]

- Moving from analog to individually-spiking models, I consider the behaviour of populations of integrate-and-fire neurons coupled in mutual inhibition and displaying spike-frequency adaptation. As in the phasic analog neuron case, oscillatory behaviour occurs for sufficiently strong coupling, and it is possible to analyze the system's behaviour at the population level despite the large numbers of individual elements involved. Reasonably accurate predictions are made for the point of onset of oscillations, the period and amplitude of the oscillations, and the point at which oscillator death occurs. [Chapter 4]

- Networks of coupled neurons can carry out a signal-processing operation known as noise-shaping, in which noise is shifted from low to high frequencies. The addition of spike-frequency adaptation improves noise-shaping in networks of integrate-and-fire neurons; this extends previous work by Mar et al. [36]. Networks consisting of more complex conductance-based neurons also show the noise-shaping behaviour. In the conductance-based case, the noise-shaping performance is not directly improved by introducing adaptation, but adaptation does offer an advantage in terms of distributing the signal representation more evenly across a heterogeneous network. [Chapter 5]

- Random reset is a popular method of introducing variability into the otherwise fully deterministic firing of integrate-and-fire models. Two types of random reset are commonly used: random voltage reset, in which the membrane voltage is reset to a stochastic initial value after each spike; and random threshold reset, in which the firing threshold is chosen stochastically after each spike. At low firing frequencies, the two forms of random reset have opposite effects on the level of variability seen in the neuron's firing record: in the presence of spike-frequency adaptation, this difference is seen even at higher firing rates. A few simple calculations serve to demonstrate why this is the case. [Chapter 6]
1.7 Local abbreviations

In some of the chapters of this thesis, the algebraic expressions become unwieldy unless terms are collected into conveniently defined groups. In some cases these groupings have clear meanings, and have been named appropriately. Other groups have no obvious physical meaning, and are defined purely for algebraic convenience; I have used the symbols $Z_i$, $i$ an integer, for all such definitions. Each $Z_i$ is defined at an appropriate place in the text, and also reproduced in a table at the start of each chapter, along with any other definitions used in the chapter.

I use the term local abbreviations because each $Z_i$ (or other definition) applies only within its chapter of origin. Thus, the definition of $Z_1$ used in chapter 2 is not the same as that in chapter 4. This allows a standard format to be used for all such abbreviations, without requiring the reader to search through large global lists to find a particular definition.
Chapter 2

Phasic analog neurons

2.1 Local abbreviations

The following table lists the abbreviations used for convenience in this chapter: as discussed in section 1.7, they are “local” in the sense that they apply only within this chapter.

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Definition</th>
<th>Section</th>
</tr>
</thead>
<tbody>
<tr>
<td>$Z_1$</td>
<td>$k\tau$</td>
<td>2.1</td>
</tr>
<tr>
<td>$Z_2$</td>
<td>$\sqrt{1 + Z_1 + Z_1^2}$</td>
<td></td>
</tr>
<tr>
<td>$Z_3$</td>
<td>$Z_2(Z_1 + 1)$</td>
<td></td>
</tr>
<tr>
<td>$Z_4$</td>
<td>$Z_2(Z_1 - 1)$</td>
<td></td>
</tr>
<tr>
<td>$Z_5$</td>
<td>$Z_3 - (Z_1 + 1)^2$</td>
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<td>$Z_4 + Z_1^2 + 1$</td>
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<td>$Z_7$</td>
<td>$Z_4 - Z_1^2 - 1$</td>
<td></td>
</tr>
<tr>
<td>$Z_8$</td>
<td>$Z_3 + (Z_1 + 1)^2$</td>
<td></td>
</tr>
<tr>
<td>$Z_9$</td>
<td>$[\gamma f''(\theta)(1 + Z_1)]/[f'(\theta)Z_1 Z_2(16Z_1^2 + 41Z_1 + 16)]$</td>
<td></td>
</tr>
<tr>
<td>$\chi_1$</td>
<td>$\gamma \left(\frac{Z_1 - Z_2}{1 + Z_1} q_1 + \frac{Z_1 - Z_2}{1 + Z_1} q_2 - \frac{1}{\sqrt{Z_1}} q_3\right) + \theta$</td>
<td></td>
</tr>
<tr>
<td>$\chi_2$</td>
<td>$\gamma \left(\frac{Z_1 - Z_2}{1 + Z_1} q_1 + \frac{Z_1 - Z_2}{1 + Z_1} q_2 + \frac{1}{\sqrt{Z_1}} q_3\right) + \theta$</td>
<td></td>
</tr>
<tr>
<td>$J_{12}$</td>
<td>$f(\chi_1) + f(\chi_2) - 2f(\theta)$</td>
<td></td>
</tr>
<tr>
<td>$J_{21}$</td>
<td>$f(\chi_2) - f(\chi_1)$</td>
<td></td>
</tr>
<tr>
<td>$N_1$</td>
<td>$f'''(\theta)f'(\theta)(16(k\tau)^3 + 57(k\tau)^2 + 57k\tau + 16)$</td>
<td></td>
</tr>
<tr>
<td>$N_2$</td>
<td>$8[f''(\theta)]^2((k\tau)^3 + 3(k\tau)^2 + 3k\tau + 1)$</td>
<td></td>
</tr>
<tr>
<td>$D_1$</td>
<td>$[f'(\theta)]^2(16(k\tau)^2 + 4k\tau + 16)$</td>
<td></td>
</tr>
</tbody>
</table>
2.2 Introduction and background

As discussed in the introductory chapter, neurons respond to stimuli by generating action potentials, voltage spikes which travel down the cell's axon. Arriving at synaptic junctions, these spikes influence, typically through neurotransmitters diffused across a synaptic gap, the states of other neurons (or of muscles or other tissues). To model the behavior of a neuron, we may work at the level of the biochemistry of the cell, or we may propose simplified models which capture various aspects of the dynamics. Several popular neural models are, in order of increasing abstractness: the Hodgkin-Huxley equations [25], the FitzHugh-Nagumo equations [28, 29] (see also discussions in [30] and [31]), and integrate-and-fire models [32, 37, 38]; refer back to the discussions in chapter 1 for more detail.

At a higher level of abstraction, we may replace the individual spiking times with a time-averaged firing rate. Information is lost in this process (see [39] for a discussion of this point), but the result is a considerably simplified model in which each neuron may be considered to output an analog value, its spiking rate. Such "analog" or "graded-response" neural models have been proposed by Hopfield [34] and Cohen and Grossberg [40], and may be applied in cases where the time scale of interest is long relative to the typical interspike time, or when each analog neuron is taken to model a population of individually spiking neurons rather than a single cell. Analog models may be explicitly derived from spiking-time models by carrying out the time averaging process [38, 41]. Analog neurons have proven useful in modelling associative memory [34, 42], as behavior controllers for autonomous robots [43, 44], and in solving optimization problems [45].

In associative memory or optimization problems, networks of analog neurons produce their "answer" by converging to a fixed-point attractor. In the memory problem, we create an attractor corresponding to each stored pattern, and expect the network to recover the original pattern when presented with a noisy version of it. For such applications, we always want the network to converge to a fixed point, and oscillatory solutions are to be avoided. Extensive analysis has been performed on networks of the types introduced in [34, 40], and it has been shown (see, in addition to the original papers, [46, 47, 48, 49, 50, 51]) that they do indeed have the property of always converging to a fixed point.

There are many biological situations, however, in which oscillations are necessary, for example to drive autonomic functions and in locomotion (see [52] and references therein). It is thus of interest to examine situations in which the much-studied analog neuron models may be made to generate oscillatory solutions. Many of the oscillatory neural signals seen in biology are generated by central pattern generators (CPGs): networks of neurons whose interconnections are such that the neurons collectively produce rhythmic outputs. CPGs often work on the principle of mutual inhibition, in which neurons (or groups of neurons) are reciprocally connected so that the output of each neuron inhibits the other [52]. Perhaps the earliest description of a CPG of the type
Figure 2.1: “Half-center” oscillator. Each analog neuron receives a constant input $I_i$ and is coupled to the other in mutual inhibition ($w_{ij} < 0$).

shown in Figure 2.1 was Brown’s “half-center model” [53, 54]. As Brown noted, oscillations in two mutually inhibitory neurons can occur if the inhibition is limited in duration. If an initial asymmetry allows the first neuron to dominate, it will “gain the upper hand,” suppressing the other while firing strongly itself. If this inhibition is of limited duration, the second neuron will eventually cease to be suppressed, allowing it to dominate and inhibit the first, and so on, yielding a cycle of alternating bursts of activity in the two neurons. Despite its simplicity, the half-center model does capture the essential dynamics of CPGs actually observed in biology: Satterlie [55], for example, describes the signals used in swimming in the pteropod mollusc Clione limacina as being generated by this mechanism.

What causes the limited duration of inhibition which the half-center model assumes? There are several possible neurophysiological mechanisms, including fatigue, post-inhibitory rebound, and spike-frequency adaptation [52, 56]. This chapter will focus on the last of these. While some biological neurons are “tonic,” responding with a steady firing rate output when stimulated with a constant input. many others are “phasic” or “adapting,” initially responding to a constant stimulus, but gradually ceasing to respond as the stimulation persists [11]. (Figure 2.2 shows the different responses of tonic and phasic analog neurons to a constant input.) Clearly if the two neurons in Figure 2.1 are phasic, oscillations become possible; once a given neuron has come to dominate, its input becomes constant and it will eventually “adapt out,” reducing its output and allowing the other neuron to take over.

Suppose that we wish to model the half-center CPG using analog neurons. If we use two standard analog neurons [34, 40], the system will converge to a fixed point, and no oscillations will occur. If we wish this simple two-neuron system to oscillate, we must introduce some mechanism to limit the inhibitory duration. We shall do this by proposing a simple means by which the
Figure 2.2: Firing rate outputs for single phasic and tonic analog neurons with no self-connections. The dynamics are $\tau \dot{x} = -x + I$, $\dot{\alpha} = k(x - \alpha)$. The firing rate output is given by $y = f(\gamma(x - \alpha) + \theta)$, where $\gamma = 4$ and $\theta = -2$ are shifting and scaling parameters and $f(x) = 1/(1 + e^{-x})$. (Dashed line) Tonic neuron: $k = 0$; $\tau = 1$; $I = 1$. (Solid line) Phasic neuron: $k = 1$; $\tau = 1$; $I = 1$. 
qualitative dynamics of neural adaptation may be added to existing analog neuron models. Beyond simply allowing us to model the half-center CPG, the addition of neural adaptation to existing analog neurons enriches their dynamics and extends the range of neurological phenomena to which they may be applied.

I will begin by introducing the units I have called "phasic analog neurons." then proceed to discuss a model of the half-center CPG formed by connecting two such neurons with mutual inhibition. A Hopf bifurcation analysis of the model will enable us to calculate the inhibitory connection strength at which oscillations begin to occur, and show us how to tune the system parameters to yield cycles with desired characteristics.

2.3 Phasic analog neurons

Networks of the Hopfield or Cohen-Grossberg type capture the essential dynamics of temporal summation: biological neurons maintain a decaying trace of their past excitation levels [11]. Models of this type generally omit, however, the dynamics of spike-frequency adaptation (but see [57, 58]): many real neurons (called "phasic" or "adapting") respond only at the onset of a constant or slowly-varying stimulus. then cease responding as the stimulus persists [11]; biological neurons which respond steadily to constant input also exist, and are called "tonic." (Adaptation is most often discussed in relation to sensory neurons, so it is perhaps worth pointing out that motor neurons can also display this behaviour. Atwood and Nguyen [59], for example, discuss phasic and tonic motor neurons in crayfish.)

I propose a simple, computationally efficient method by which a form of neural adaptation may be added to existing analog neuron models. Consider a variant of the equations introduced by Hopfield [34] (the tonic version of these equations has also been used by Beer and Gallagher [43, 44]), and augment each neuron's description with a second linear differential equation. The dynamics of a network are then written as

\[
\begin{align*}
\tau_i \dot{x}_i &= -x_i + \sum_j w_{ij} y_j + I_i \\
\dot{y}_i &= k_i (x_i - \alpha_i),
\end{align*}
\]  

for \( i = 1, \ldots, n \). The \( x_i \) represent activation levels (corresponding to a membrane potential) with time constants \( \tau_i > 0 \). The \( \alpha_i \) represent firing thresholds, with rate constants \( k_i \geq 0 \). The \( y_i \) represent output firing rates, and are functions of the difference between \( x \) and \( \alpha \): we use

\[ y_i = f(\gamma(x_i - \alpha_i) + \theta), \]

where \( f(\cdot) \) is the firing rate function and \( \gamma > 0 \) and \( \theta \) are scaling and shifting parameters. I will not specify the form of firing function at this point; in section 2.4 I will discuss the effects of two different forms. I take the connection strengths \( w_{ij} \) from neuron \( i \) to neuron \( j \) to be constant. Each neuron receives an external input \( I_i \), which may be time-varying. Figure 2.2 shows the result of integrating (2.1) for a single node with no self connection.
Matsuoka [57, 58] proposes a similar approach to adding neural adaptation to an analog model, but one in which an adaptation term is incorporated directly into the activation equation; in this model, the $\dot{\alpha}$ equation may be appended to any form of activation equation (for concreteness, we will use the form in (2.1) throughout this chapter). Horn and Usher [60] describe a form of adaptation for discrete-time, binary-state neurons, as does Halperin [61].

The addition of the $\dot{\alpha}$ equation is equivalent to passing $x$ through an RC high-pass filter circuit, with $k = 1/RC$. Since the effect of temporal summation is low-pass filtering of the input [62, 63], a phasic neuron acts as a band-pass filter. Consider a single neuron of the type given in (2.1), with no self-connection: $\tau \ddot{x} = -x + I(t)$, $\dot{\alpha} = k(x - \alpha)$. With input $I(t) = \cos \omega t$, the steady-state output may be shown to be $(x - \alpha)(t) = A \cos(\omega t - \psi)$, with

$$A = \omega [\omega^2 (1 + k\tau)^2 + (k - \omega^2 \tau)^2]^{-1/2}$$  \hspace{1cm} (2.2)

and

$$\psi = \tan^{-1} \left[ \frac{k - \omega^2 \tau}{\omega (1 + k\tau)} \right].$$  \hspace{1cm} (2.3)

The amplitude $A$ drops to zero as $\omega \to 0$ and as $\omega \to \infty$, reaching a maximum value of $A = 1/(1 + k\tau)$ at $\omega = \sqrt{k/\tau}$. The phase $\psi$ is zero at $\omega = \sqrt{k/\tau}$, approaches $\pi/2$ as $\omega \to 0$, and approaches $-\pi/2$ as $\omega \to \infty$. See Figure 2.3.

### 2.4 Oscillatory solutions: Hopf bifurcation

I will now consider the behaviour of two phasic neurons, reciprocally connected as shown in Figure 2.1. This represents the dynamics of a simple CPG, the half-center model [52, 53, 56], and I will show that oscillatory solutions arise for sufficiently strong mutual inhibition. Consider the case of two identical neurons ($\tau_1 = \tau_2 = \tau$, $k_1 = k_2 = k$) with a symmetric connection ($\omega_{12} = \omega_{21} = \omega$) and no self-connections ($\omega_{11} = \omega_{22} = 0$). The system has a single fixed point; shifting this point to the origin, the equations become

$$\tau \dot{\tilde{x}}_1 = -\tilde{x}_1 + w f(\gamma(\tilde{x}_2 - \tilde{\alpha}_2) + \theta) - w f(\theta)$$  \hspace{1cm} (2.4)

$$\dot{\tilde{\alpha}}_1 = k(\tilde{x}_1 - \tilde{\alpha}_1)$$  \hspace{1cm} (2.5)

$$\tau \dot{\tilde{x}}_2 = -\tilde{x}_2 + w f(\gamma(\tilde{x}_1 - \tilde{\alpha}_1) + \theta) - w f(\theta)$$  \hspace{1cm} (2.6)

$$\dot{\tilde{\alpha}}_2 = k(\tilde{x}_2 - \tilde{\alpha}_2)$$  \hspace{1cm} (2.7)

where I have defined $\tilde{x}_i = x_i - I_i - w f(\theta)$ and $\tilde{\alpha}_i = \alpha_i - I_i - w f(\theta)$. 
Figure 2.3: Frequency response of a single phasic analog neuron (with no self-connection) to an input $I(t) = \cos \omega t$. (Top) Steady-state output amplitude, $A$, from equation (2.2). (Bottom) Phase, $\psi$, from equation (2.3). Parameters: $\tau = k = 1$. 
Treating \( w \) as a bifurcation parameter, I will perform a Hopf bifurcation analysis of (2.4-2.7) using standard techniques. (See [64] for a discussion of Hopf bifurcations in a general class of coupled nonlinear oscillators, and [37] for a demonstration of the bifurcation in a pair of asymmetrically connected neurons with self-connections.) For the reader's convenience, I will reproduce the Hopf theorem here (the following is a slightly modified version of the statements given in [65] and [66]):

**Theorem 2.1 (Hopf bifurcation theorem)** Suppose that \( \dot{x} = F(x, y, \mu), \dot{y} = G(x, y, \mu) \) (where \( \mu \) is a parameter such that the bifurcation point occurs at \( \mu = 0 \)), with \( F(0,0,\mu) = G(0,0,\mu) = 0 \) and that the Jacobian matrix \( \begin{pmatrix} \frac{\partial F}{\partial x} & \frac{\partial F}{\partial y} \\ \frac{\partial G}{\partial x} & \frac{\partial G}{\partial y} \end{pmatrix} \) evaluated at the origin when \( \mu = 0 \) is
\[
\begin{pmatrix} 0 & -\omega \\ \omega & 0 \end{pmatrix}
\]
for some \( \omega \neq 0 \); this implies that the Jacobian has the purely imaginary eigenvalues \( \pm i\omega \). If
\[
F_{\mu x} + G_{\mu y} \neq 0 \tag{2.8}
\]
and
\[
a \neq 0, \tag{2.9}
\]
where
\[
a = \frac{1}{16} (F_{xx} + F_{yy} + G_{xz} + G_{yy}) \\
+ \frac{1}{16\omega} [F_{xy} (F_{xx} + F_{yy}) - G_{xy} (G_{xz} + G_{yy}) - F_{xx} G_{xx} + F_{yy} G_{yy}] \tag{2.10}
\]
is a constant [all partial derivatives (\( F_x = \partial F/\partial x \), and so on) in (2.8) and (2.10) are evaluated at \( (0,0,0) \)], then a curve of periodic solutions bifurcates from the origin into \( \mu < 0 \) if \( a(F_{\mu x} + G_{\mu y}) > 0 \) or into \( \mu > 0 \) if \( a(F_{\mu x} + G_{\mu y}) < 0 \). The origin is stable for \( \mu > 0 \) (resp. \( \mu < 0 \)) and unstable for \( \mu < 0 \) (resp. \( \mu > 0 \)) if \( F_{\mu x} + G_{\mu y} < 0 \) (resp. \( F_{\mu x} + G_{\mu y} > 0 \)). If \( a < 0 \) the periodic solutions are stable, while if \( a > 0 \) the periodic solutions are repelling; the bifurcation is supercritical if the bifurcating periodic orbits are stable, otherwise it is subcritical. The amplitude of the periodic orbits grows as \( |\mu|^{1/2} \) whilst their periods tend to \( \frac{2\pi}{|\omega|} \) as \( |\mu| \) tends to zero.

(Note that the Hopf theorem addresses a two-dimensional system. Higher-dimensional systems may be reduced to two dimensions by considering only the dynamics on the center manifold; see Theorem 2.2 on page 33. All of the calculations relevant to the Hopf bifurcation analysis may be carried out in this reduced system; see [65, 66] for more information on this point.)
Evaluating the Jacobian of (2.1-2.7) at the origin, the first condition of the Hopf theorem is that we must have a pair of purely imaginary eigenvalues. This condition is satisfied at \( w = \pm w^* \), where \( w^* = (1 + k\tau)/\sqrt{f'(\theta)} \) with \( f'(x) = \partial f(x)/\partial x \): at these points, we have the eigenvalues \( \lambda_{1,2} = \frac{-1 + k\tau \pm \sqrt{1 + k\tau + (k\tau)^2}}{\tau} \) and \( \lambda_{3,4} = \pm \sqrt{-k/\tau} = \pm i\omega \), where \( \omega \equiv \sqrt{k/\tau} \). Note that \( \lambda_1 \) and \( \lambda_2 \) are real and both strictly negative for \( k > 0 \). Setting \( w = -w^* - \mu \) (or \( w = w^* + \mu \)) puts the bifurcation point at \( \mu = 0 \), as in the statement of Theorem 2.1.

To examine the second condition of the theorem \( (F_{\mu x} + G_{\mu y} \neq 0) \), let us apply a linear change of coordinates to (2.4-2.7), bringing the system into the normal form

\[
\begin{pmatrix}
\dot{q}_1 \\
\dot{q}_2 \\
\dot{q}_3 \\
\dot{q}_4
\end{pmatrix} =
\begin{pmatrix}
\lambda_1 & 0 \\
0 & \lambda_2
\end{pmatrix}
\begin{pmatrix}
q_1 \\
q_2
\end{pmatrix} +
\begin{pmatrix}
\phi_1(q) \\
\phi_2(q)
\end{pmatrix}
\tag{2.11}
\]

where the \( \phi_i \) contain all the nonlinear terms. The following set of local abbreviations allow the \( \phi_i \) to be expressed compactly: \( Z_1 \equiv k\tau; Z_2 \equiv \sqrt{1 + Z_1 + Z_1^2}; Z_3 \equiv Z_2(Z_1 + 1); Z_4 \equiv Z_2(Z_1 - 1); Z_5 \equiv Z_3 - (Z_1 + 1)^2; Z_6 \equiv Z_4 + Z_2^2; Z_7 \equiv Z_4 - Z_2^2 + 1; Z_8 \equiv Z_3 + (Z_1 + 1)^2; \)

\[\chi_1(q) \equiv \gamma \left( \frac{Z_1 - Z_2}{1 + Z_1} q_1 + \frac{Z_1 + Z_2}{1 + Z_1} q_2 - \frac{1}{\sqrt{Z_1}} q_3 \right) + \theta;\]

\[\chi_2(q) \equiv \gamma \left( \frac{Z_1 - Z_2}{1 + Z_1} q_1 + \frac{Z_1 + Z_2}{1 + Z_1} q_2 + \frac{1}{\sqrt{Z_1}} q_3 \right) + \theta;\]

\[J_{12}(q) \equiv f(\chi_1(q)) + f(\chi_2(q)) - 2f(\theta); \text{ and } J_{21}(q) \equiv f(\chi_2(q)) - f(\chi_1(q)).\]

The nonlinear terms in (2.11) may then be written as

\[\begin{align*}
(2\tau Z_2)\phi_1(q) &= Z_5 q_1 + Z_6 q_2 + wJ_{12}(q)(Z_3 - 1)/2 \\
(2\tau Z_2)\phi_2(q) &= Z_7 q_1 + Z_8 q_2 + wJ_{12}(q)(Z_2 + 1)/2 \\
\tau\phi_3(q) &= -q_3(Z_1 + 1) + wJ_{21}(q)\sqrt{Z_1}/2 \\
\phi_4(q) &= 0.
\end{align*}\]

The second condition of the Hopf bifurcation theorem, inequality (2.8), then becomes

\[\frac{\partial^2 \phi_3}{\partial \mu \partial q_3} + \frac{\partial^2 \phi_4}{\partial \mu \partial q_4} = \frac{\partial^2 \phi_3}{\partial \mu \partial q_3} \neq 0.\]

where the partial derivatives are evaluated at the origin and the \( \phi_4 \) derivative vanishes since \( \phi_4 = 0 \). Using (2.14), the partial derivative may be found to be
\[
\frac{\partial^2 \phi_3}{\partial \mu \partial \theta} \bigg|_{w=\pm w^*, q_3=0} = \pm \frac{\gamma f'(\theta)}{2\tau}.
\] (2.17)

Since \( \gamma > 0 \) and \( \tau > 0 \), condition (2.16) is satisfied for \( f'(\theta) \neq 0 \). Thus, as long as the firing rate function \( f(\cdot) \) and the shifting parameter \( \theta \) are such that \( f'(\theta) \neq 0 \), the first two conditions of the Hopf theorem are satisfied for \( w = \pm w^* \). The next step is to consider the stability coefficient \( a \), given by equation (2.10); recall that the sign of this coefficient tells us whether the periodic solutions are attracting or repelling.

Since the half-center model relies on mutual inhibition, let us consider \( w = -w^* - \mu \), and examine what occurs as \( \mu \) crosses from negative to positive values. Examining the expression for \( a \) in equation (2.10), we see that in the nomenclature of (2.11), we have \( F = \phi_3 \) and \( G = \phi_4 \); since \( G = \phi_4 = 0 \), equation (2.10) simplifies considerably, yielding

\[
a = \frac{1}{16} \left( \frac{\partial^2 \phi_3}{\partial q_3^2} + \frac{\partial^2 \phi_3}{\partial q_3 \partial q_4^2} \right) + \frac{1}{16 \omega} \left[ \frac{\partial^2 \phi_3}{\partial q_3 q_4^2} \left( \frac{\partial^2 \phi_3}{\partial q_3^2} + \frac{\partial^2 \phi_3}{\partial q_4^2} \right) \right].
\] (2.18)

where as before the partials are evaluated at the origin.

To evaluate the partial derivatives in (2.18), we need to find a local expression for an invariant manifold called the center manifold. The center manifold theorem [65, 66] is a well-known result that allows the center manifold for a nonlinear system to be calculated (in some local region of interest) from a linearized version of the system. I reproduce the theorem here for the convenience of the reader (this statement of the theorem is from [65]):

**Theorem 2.2 (Center manifold theorem)** Let \( \mathcal{F} \in C^r(\mathbb{R}^n) \) with \( \mathcal{F}(0) = 0 \). Divide the eigenvalues, \( \lambda \), of \( D\mathcal{F}(0) \) (the Jacobian matrix evaluated at the origin) into three sets, \( \sigma_u \), \( \sigma_s \), and \( \sigma_c \), where \( \lambda \in \sigma_u \) if \( \text{Re}(\lambda) > 0 \), \( \lambda \in \sigma_s \) if \( \text{Re}(\lambda) < 0 \), and \( \lambda \in \sigma_c \) if \( \text{Re}(\lambda) = 0 \). Let \( E^u \), \( E^s \), and \( E^c \) be the corresponding generalized eigenspaces. Then there exist \( C^r \) unstable and stable manifolds \( (W^u \text{ and } W^s) \) tangential to \( E^u \) and \( E^s \) respectively at \( x = 0 \) and a \( C^{r-1} \) center manifold, \( W^c \), tangential to \( E^c \) at \( x = 0 \). All are invariant, but \( W^c \) is not necessarily unique.

Equation (2.11) indicates that our system has a two-dimensional stable eigenspace, the \( q_1 - q_2 \) plane in the transformed coordinates (recall that \( \lambda_1 < 0 \), \( \lambda_2 < 0 \)). It also has a two-dimensional center eigenspace, the \( q_3 - q_4 \) plane. The center manifold is an invariant subspace of the full four-dimensional space, which from Theorem 2.2 is tangent to the center eigenspace at the origin.

I will approximate the center manifold in the vicinity of the origin using

\[
q_1 \simeq \pi_1(q_3, q_4) = a_1 q_3^2 + b_1 q_3 q_4 + c_1 q_4^2
\] (2.19)

and
Note that zero- and first-order terms have been omitted, since the requirement that the center manifold is tangent to the $q_3 - q_4$ plane at the origin means that all these terms must vanish. The coefficients are obtained by comparing terms in

$$\dot{q}_1 = \lambda_1 q_1 + \phi_1(q) \simeq \lambda_1 \pi_1 + \phi_1(\pi_1, \pi_2, q_3, q_4)$$

[from equation (2.11)] with those in

$$\dot{q}_1 = \frac{\partial \pi_1}{\partial q_3} \dot{q}_3 + \frac{\partial \pi_1}{\partial q_4} \dot{q}_4,$$

and similarly for $\dot{q}_2$. The algebra involved in determining the coefficients in (2.19-2.20) is cumbersome but straightforward: I will not reproduce the full details here. Defining the local abbreviation

$$Z_9 = [\gamma f''(\theta)(1 + Z_1)]/[f'(\theta)Z_1Z_2(16Z_1^2 + 41Z_1 + 16)],$$

the coefficients are:

$$a_1 = Z_9[4 - 23Z_1Z_2 - 12Z_2 - 12Z_2 + 8Z_1^2 - 19Z_1^2 - 8Z_1^2 Z_3]/4$$
$$a_2 = Z_9[-4 - 23Z_1Z_2 - 12Z_2 + 4Z_1 + 8Z_1^2 + 19Z_1^2 - 8Z_1^2 Z_2]/4$$
$$b_1 = -Z_9[2Z_1^2 + 4Z_1 + 2Z_1Z_2 - 3 + 5Z_2]/2\sqrt{Z_1}$$
$$b_2 = -Z_9[-2Z_1^2 - 2Z_1 + 2Z_1Z_2 + 3 + 5Z_2]/2\sqrt{Z_1}$$
$$c_1 = -Z_9[2 + 6Z_1 + 2Z_1 + 11Z_1^2 + 9Z_1Z_2 + 4Z_1^3 + 4Z_1^2Z_2]/2$$
$$c_2 = Z_9[2 + 6Z_1 + 2Z_1 + 11Z_1^2 + 9Z_1Z_2 + 4Z_1^3 - 4Z_1^2Z_2]/2$$

With these coefficients in hand, it is possible to evaluate the partial derivatives in (2.18): when this is done (once again, the algebra is lengthy but uninteresting), we obtain

$$a = \frac{\gamma^2}{16(k\tau)} \frac{N_1 - N_2}{D_1}.$$  \hspace{1cm} (2.21)

using the local abbreviations

$$N_1 \equiv f''(\theta)f'(\theta)(16k\tau)^3 + 57(k\tau)^2 + 57k\tau + 16,$$
$$N_2 \equiv 8[f'(\theta)]^2[(k\tau)^3 + 3(k\tau)^2 + 3k\tau + 1],$$
$$D_1 \equiv [f'(\theta)]^2(16k\tau)^2 + 41k\tau + 16).$$

Both supercritical ($a < 0$; stable oscillatory solutions) and subcritical ($a > 0$; unstable oscillatory solutions) Hopf bifurcations occur for the half-center oscillator model, depending on the choice of the parameters $k$, $\tau$, $\gamma$ and $\theta$. The sign of $a$ is a function only of $\theta$ and the product $k\tau$, which reflects the ratio of the time scales of the activation and adaptation equations. The
Figure 2.4: Sigmoidal firing rate function \( f_1(x) = 1/(1 + e^{-x}) \). The plot shows \( f_1(\gamma x + \theta) \), where \( \gamma \) and \( \theta \) are scaling and shifting parameters. (Dashed line) The unscaled, unshifted function \((\gamma = 1, \theta = 0)\). (Dash-dotted line) Scaled but not shifted \((\gamma = 4, \theta = 0)\). (Solid line) Shifted and scaled \((\gamma = 4, \theta = -2)\).

The details of the stability of the oscillatory solutions depend on the form of the firing rate function, \( f(-) \). First, consider a common sigmoidal choice, \( f_1(x) = 1/[1 + \exp(-x)] \) (see Figure 2.4). Since \( f'_1(\theta) > 0 \) for all \( \theta \), and the condition (2.8) is satisfied for all parameter settings. The stability boundaries for this case are shown in Figure 2.5. Note that for this firing rate function, whether the bifurcation is supercritical or subcritical depends mainly on the value of \( \theta \), which is associated with the spontaneous firing rate of each neuron. Figure 2.6 shows a plot of \( a(\theta) \) with the other parameters fixed at \( k = \tau = 1, \gamma = 4 \).
Figure 2.5: Stability boundaries for the sigmoidal activation function $f_1(x) = 1/[1 + \exp(-x)]$. The periodic solutions arising at the Hopf bifurcation point are stable in the central region ($a < 0$), and unstable above and below it ($a > 0$).
Figure 2.6: Stability coefficient $a$ vs. shifting parameter $\theta$, for sigmoidal activation function $f_1(x) = 1/[1 + \exp(-x)]$. The other system parameters are fixed at $\tau = k = 1, \gamma = 4$. A transition from stable ($a < 0$) to unstable ($a > 0$) oscillations occurs at $\theta = \pm 1.68$. 
Although the sigmoidal firing rate function is a popular choice in work with analog neurons, and in particular with the Hopfield equations, it does not reproduce the form of the firing rate function often seen in real neurons. The \( f-I \) curves (firing rate vs. applied current) of biological neuron frequently have the general shape seen in Figure 2.7; many conductance-based model neurons also have this form, as does the refractory integrate-and-fire model. (See Figure 5.8 and [33, 38].) A simplified function which has the desired qualitative form is \( f_2(x) = \mathcal{H}(x)/(1 + \ln(1 + 1/x)) \), where \( \mathcal{H}(x) \) is the Heaviside step function \( (\mathcal{H}(x) = 1 \text{ for } x > 0, \mathcal{H}(x) = 0 \text{ otherwise}) \). For this choice of firing function, we must consider only \( \theta > 0 \); otherwise we have \( f'(\theta) = 0 \), which renders the bifurcation value \( w^* = (1 + k\tau)/\gamma f'(\theta) \) undefined, and violates the second condition of the Hopf theorem. inequality (2.8). Figure 2.8 shows the stability boundaries when this firing rate function is used. Note that this choice of function makes the product \( k\tau \) the main factor in determining the stability of solutions: this product represents the ratio of the time scales of the excitation and adaptation processes.

Numerical simulation has been used to test the algebraic results. (All numerical results have been generated using the sigmoidal firing rate function \( f_1(\cdot) \), introduced above.) With \( k = \tau = 1, \gamma = 4, \theta = 0, \) we find \( w^* = 2, a = -1 \). With \( w = -w^* - \mu \), the Hopf theorem predicts circular trajectories on the center manifold (here, the \( q_3-q_4 \) plane) with radius \( r = \sqrt{-\gamma f'(\theta)\mu/2\tau a} = \sqrt{\mu/2} \). Setting \( \mu = 0.02 \) and integrating (2.11) numerically, we find that the projection of the trajectory onto the \( q_3-q_4 \) plane converges to a circle of radius \( r = \sqrt{0.02/2} = 0.1 \). as expected. Note that Hopf bifurcation analysis is strictly local: it tells us that oscillatory solutions will arise in the vicinity of the origin of (2.4-2.7) when \( |w| \) exceeds \( w^* \). It does not guarantee that oscillatory solutions will not occur for smaller values of \( w \). With \( a > 0 \), a large-magnitude limit cycle appears: this cycle is globally stable for \( \mu > 0 \), while for \( \mu < 0 \) the system becomes multistable, with some solutions converging to the origin and some to the limit cycle. Figure 2.9 shows such a case. With \( a < 0 \), the simulations indicate that the origin is globally stable for \( \mu < 0 \).

This analysis allows us to choose the system parameters in (2.4-2.7) to yield the type of oscillatory solutions we desire. Selecting parameters for which \( a < 0 \) and taking \( \mu \) to be small, we obtain small limit cycles in the vicinity of the fixed point, corresponding to small fluctuations in the base firing rate of the two neurons, as shown in Figure 2.10. With \( a > 0 \) and \( \mu > 0 \), the oscillatory solutions near the origin are unstable, and the trajectories are repelled outwards, finally being intercepted by a larger limit cycle in which the two neurons are alternately strongly activated and strongly inhibited: see Figure 2.11. Either of these cases may be used to represent a half-center CPG.

I have examined only the vicinity of \( -w^* \), but another Hopf bifurcation with identical stability properties occurs for \( w = w^* + \mu \). The oscillatory solutions arising for this case have the two neurons becoming active in phase with each other, rather than being activated in alternation as
Figure 2.7: Nonsigmoidal firing rate function \( f_2(x) = \Theta(x)/[1 + \ln(1 + 1/x)] \). The plot shows \( f_2(\gamma x + \theta) \), where \( \gamma \) and \( \theta \) are scaling and shifting parameters. (Dashed line) The unscaled, unshifted function (\( \gamma = 1, \theta = 0 \)). (Dash-dotted line) Scaled but not shifted (\( \gamma = 4, \theta = 0 \)). (Solid line) Shifted and scaled (\( \gamma = 4, \theta = 2 \)).
Figure 2.8: Stability boundaries for the nonsigmoidal activation function $f_2(x) = \Theta(x)/(1 + \ln(1 + 1/x))$. Oscillatory solutions are stable to the right of the boundary line ($a < 0$) and unstable to the left ($a > 0$).
Figure 2.9: Multistability in the half-center oscillator. The plot shows trajectories projected onto the $q_3 - q_4$ plane, for $\tau = k = 1$, $\gamma = 4$, and $\theta = -4$; the sigmoidal firing rate function $f_1(x)$ has been used. The bifurcation point is $w^* = 28.308$, and the stability coefficient is $a = 0.973$. With $w = -w^* - \mu$, the plot shows trajectories (obtained by numerical integration) for $\mu = -15$. The system is multistable, with coexistence between a stable fixed point and a stable limit cycle.
Figure 2.10: Neural outputs vs. time, obtained by numerical integration with parameters: \( \tau = k = 1, \gamma = 4, \theta = 0, w^* = 2, w = -2.1 (\mu = 0.1) \). The stability coefficient in this case is \( a = -1 < 0 \), so the oscillatory solutions in the vicinity of the origin are attracting; past the bifurcation point, small limit cycles appear in the region around the origin, and the trajectories remain on these cycles; movement on such a limit cycle causes the fluctuations in firing rate output seen here.
Figure 2.11: Neural outputs vs. time, obtained by numerical integration with parameters: $\tau = k = 1, \gamma = 4, \theta = 0, w^* = 28.308, w = -28.408 (\mu = 0.1)$. The stability coefficient in this case is $a = 0.973 > 0$, so the oscillatory solutions in the vicinity of the origin are repelling; trajectories move away from the origin and are captured by a large-magnitude limit cycle similar to the one shown in Figure 2.9. Motion on this large limit cycle causes the alternating bursts of activity seen here.
in the half-center model.

2.5 Discussion

I have introduced a simple means of adding the qualitative dynamics of neural adaptation to any existing analog (also known as graded-response) neuron model. Using these phasic analog neurons, I have shown that one may model the dynamics of the simplest central pattern generator, the half-center model: two phasic neurons connected in a mutually inhibitory fashion, producing alternating bursts of activity. A Hopf bifurcation analysis shows the inhibitory strength past which oscillatory solutions will certainly arise, and allows oscillations of a desired type to be produced by tuning the system parameters.

In the absence of neural adaptation, two mutually inhibitory neurons will end up with one neuron fully inhibiting the other, a situation known as "oscillator death." This has been discussed in the context of both analog and integrate-and-fire neural models, by Atiya and Baldi [37] and by Bressloff and Coombes [38]. As we have seen in this chapter, mutual inhibition can in fact lead to oscillatory behavior in a pair of neurons, provided that the inhibitory effect is of limited duration. This limited duration is the key to the appearance of oscillatory solutions, and thus I would not expect the details of the time course of the neural adaptation to affect the existence of a bifurcation to oscillatory solutions.

Adaptation in biological neurons is a complex process, depending on the details of the biochemical dynamics. The firing thresholds introduced in (2.1), while yielding the qualitative dynamics of adaptation, are not proposed as a physiologically realistic model. More realistic adaptation equations could replace the linear firing threshold equations with physiologically motivated nonlinear equations; see chapters 4 and 5 for more physiologically detailed models of adaptation.

Analog neurons have proven to be a useful tool both in modelling some of the functions of the brain and in attempting to reproduce animal behavior in the context of robotics and artificial intelligence. The addition of neural adaptation to these models may enhance their usefulness in each of these areas.

2.6 Future directions

In the model presented here, a constant input leads to complete adaptation: that is, the fully-adapted firing rate is the same as the rate in the absence of any input, namely $f(\theta)$. As the more detailed models used in chapters 4 and 5 will show, it would be more realistic to assume only partial adaptation, in which the neuron drops to some fraction of its initial rate, but not all the way to $f(\theta)$. I have made one attempt along these lines [67], modifying the network dynamics.
(equation (2.1)) to have the form

\[ \tau_i \dot{x}_i = -x_i + \sum_j w_{ji} y_j + I_i, \]  
\[ \dot{\alpha}_i = k_i [x_i \mathcal{H}(x_i) - \alpha_i] - r \alpha_i, \]

with the firing rate output being \( y_i = f(\gamma(x_i - \alpha_i) + \theta) \) as before. The varying threshold \( \alpha \) now has two new properties: it is always positive (due to the Heaviside step function \( \mathcal{H}(x) \)); and it has a "leak" term with rate \( r > 0 \). For a fixed point \( \bar{x} > 0 \), the fully-adapted firing rate now drops only to \( y = f[\gamma \bar{x}(\frac{r}{\bar{\beta} + r}) + \theta] > f(\theta) \).

This new model is somewhat more physiologically realistic than the model discussed above, but it is no longer so amenable to complete mathematical analysis. It may be worthwhile to pursue this line of inquiry further. But as chapter 4 will show, it is also possible to start from populations of individual neurons and generate a set of analog equations corresponding to the aggregate behavior of the whole population. The phasic analog model is valuable for its simplicity, but for biological realism I believe that the approach of chapter 4 will be the more fruitful one to pursue.
Chapter 3

Walking gait generation

3.1 Introduction

For getting around over a wide variety of terrains, legs have a substantial advantage over wheels or tracks: animals can walk or run over ground on which wheeled machines would quickly become stuck. Only a small percentage of the Earth’s surface is reachable with wheeled vehicles, and of course other planets are notorious for their lack of adequately paved roads. The robustness of legged locomotion has led to an interest in the robotics community in constructing walking machines [68, 69, 70, 71, 72, 73] and in studying the control systems used by biological organisms to coordinate the motions of their legs [74, 75, 76, 77, 78, 79, 80, 81]. Work aimed at transferring principles of biological locomotion to robotics has been one of most productive areas in biologically inspired robotics.

A great deal of work has been done on the generation of locomotor patterns in biological organisms, both experimental [55, 82, 83, 84, 85, 86] and theoretical/computational [80, 81, 87, 88, 89, 90, 91, 92]. In this chapter, I do not propose to offer any new theoretical insight on walking gait generation, but simply to apply some principles from biology to create a surprisingly simple network which robustly generates the leg position commands for a six-legged robot. The work illustrates: 1) the usefulness of the phasic analog neurons discussed in chapter 2, which allow an elegant network architecture; and 2) the fact that drawing on simple principles from biology can aid in the implementation of a control system for a robot.

The standard picture of how rhythmic locomotor patterns arise in animals is a combination of central pattern generators (CPGs) and sensory feedback [93, 94, 95]. CPGs are groups of neurons which produce oscillatory output in the absence of any sensory input, and these are thought to provide the main rhythm in locomotor tasks such as swimming, flying, and walking. The rhythm is modulated by sensory feedback from the limbs involved, which adjusts the stepping (or flapping/swimming) pattern to compensate for perturbations from the outside world. The
simplest form of sensory feedback is the stretch reflex, wherein a muscle contracts when it is stretched, pulling the limb back towards a central position.

Here, I describe a simple network which generates a gait for a hexapod robot, using these two principles: central pattern generators to produce the main rhythmic pattern, combined with a stretch reflex to compensate for perturbations. The network produces the tripod gait (see below), and recovers neatly from perturbations to the legs, quickly reestablishing the proper gait.

3.2 Gaits

When an animal (or robot) walks, each of its legs cycle through two main types of motion: the stance phase, wherein the leg is in contact with the ground, supporting the animal and propelling it forward; and the swing phase, wherein the leg is off the ground, moving forward to prepare for the next stance phase. If we view each leg as an oscillator and choose some reference point in the stance-swing cycle (for example, the start of the stance phase), then each leg may be assigned a phase based on how far along the cycle it is. Then, different possible gaits may be described simply as different sets of phase relationships among the legs [64, 82, 96, 97]. Six-legged insects use a variety of gaits [98], two of which are shown in Figure 3.1. By far the most common hexapod gait is the tripod gait [99], in which one group of three legs (forming a triangle across the body) is swung forward while the other three legs remain on the ground, propelling the body forward and providing a tripod of support for the body so that static equilibrium is maintained at all times. See Figure 3.2.

This work considers only the problem of generating the appropriate phases for the six legs of a hexapod walking with the tripod gait. For a complete control system, additional commands are of course required to control the detailed position of each leg, and in particular to raise and lower the legs appropriately. However, once the correct phases have been obtained, it is a simple matter to generate a function that maps the phase into both front/back and up/down positions [72, 100].

3.3 Coupled neural oscillators

3.3.1 Individual oscillators

The spontaneously oscillating output corresponding to the central pattern generator portion of the neural control system is produced by two coupled phasic analog neurons, as described in chapter 2; the relevant equations are:

$$ \tau \dot{x}_1 = -x_1 + w y_2 + I_o $$ (3.1)
Figure 3.1: Phase relationships for two common hexapod gaits: the tripod gait (left) and the metachronal gait (right). Relative phases for each leg are shown inside the circle representing the leg, and are given as a fraction of unity (with 0 and 1 being identical). (Left) In the tripod gait, legs L1, R2, and L3 are in phase with one another, and half a cycle out of phase with legs R1, L2, and R3. The tripod gait is shown again in Figure 3.2. (Right) In the metachronal gait, a “wave” of stepping proceeds from back to front along one side of the body, then from back to front along the other side.

Figure 3.2: Base of support in the tripod gait. Dashed circles indicate legs off the ground (swing phase); filled circles indicate legs in contact with the ground (stance phase). The body’s base of support is indicated by a triangle connecting the stance-phase legs. The gait alternates between the left-hand state (legs L1, R2, and L3 down) and the right-hand state (legs R1, L2, and R3 down).
where \( y_i = f(\gamma(x_i - \alpha_i) + \theta) \), \( i = 1, 2 \), is the firing rate output of each analog neuron. The function \( f(\cdot) \) maps the net activation level \( (x_i - \alpha_i) \) to a firing rate output, with \( \gamma > 0 \) and \( \theta \) as scaling and shifting parameters. Throughout this chapter the firing rate function is taken to be the sigmoid function \( f(x) = \frac{1}{1 + \exp(-x)} \), which maps all values into the range \([0, 1]\). For \( w < 0 \) and \( |w| \) sufficiently large, equations (3.1-3.4) have a stable limit cycle with \( y_1 \) and \( y_2 \) out of phase with one another [92]; see chapter 2 for more detail.

The firing rates of the two analog neurons are used the control signals driving the leg positions in a very simple simulated hexapod. In fact, it is a hexapod only in the sense that six leg positions are simulated; no kinematics or dynamics are included to represent the body these legs should be carrying.

3.3.2 Neural coupling

To generate gaits, an oscillator unit is employed to produce the back and forth rhythm for each leg; these oscillators are then coupled together so that the tripod gait emerges from the network. Since gaits are defined by a set of phase relationships among the legs, the problem of designing a gait-generating network becomes one of coupling the individual oscillators such that they produce the desired phase relationships between the six legs. In the tripod gait, the requirement is that every neighbouring pair of legs have an antiphase relationship (one-half of a cycle out of phase). Coupling between nonlinear oscillators often leads to antiphase relationships [87]. Numerical simulations of two coupled oscillators of the type described in section 3.3.1 confirm that antiphase behaviour is quite easily obtained in a pair of oscillators by presenting the firing rate outputs \( (y_i) \) of each oscillator as inputs to the other in the arrangement shown in Figure 3.3.

3.4 Single leg

We move on now to the problem of using the oscillator outputs to drive the position of a leg. The leg of an insect, or even of a walking robot, is a complex piece of machinery, and would require detailed modelling to represent accurately. This will not be addressed here; instead, a very simple representation of each leg will be employed: a position variable \( \phi \), ranging over \([-1, 1]\), with the boundaries of the range representing the extreme positions ("back as far as possible" and "forward as far as possible") attainable by the limb.

The motor neurons in insects often output velocity commands rather than positions or torques [101]. In this form of control, the firing rate outputs of an individual oscillator \( (y_1 \)
Figure 3.3: Oscillators coupled to give anti-phase oscillations. Two oscillators are shown, in horizontal pairs: one pair has its member neurons drawn with solid lines, the other with dashed lines. In the synaptic connections, filled circles indicate excitatory connections, while open circles indicate inhibitory connections. Each individual oscillator is driven by constant excitatory input $I_0$, and has an internal mutual inhibition of strength $w$. Coupling between the oscillators consists of inhibitory and excitatory connections with strength $w_c$.

and $y_2$) are used as velocity commands: the leg dynamics are then

$$\dot{\phi} = V(y_1 - y_2).$$

where $V$ is a rate parameter.

Equation (3.5) is open loop: it contains no feedback from the leg's position. Animal walking is believed [93] to depend on a combination of the open-loop signal from a neural oscillator and proprioceptive feedback based on the limb's position. A simple form of feedback thought to be significant in leech swimming, locust flying, and cockroach walking [93] is known as a stretch reflex. When a muscle is stretched past a certain length, this reflex acts to oppose further lengthening of the muscle. This may be added to our simple leg model as follows:

$$\dot{\phi} = V([y_1 - gS(\phi)] - [y_2 - gS(-\phi)]),$$

where

$$S(\phi) = \max\{\phi - \phi_{sr}, 0\}.$$  

$\phi_{sr} \geq 0$ is the position at which the stretch reflex is activated and $g$ is the gain. Note that $\phi = 0$ is the limb's neutral position, and that the reflex is activated both for $\phi > \phi_{sr}$ and for $\phi < -\phi_{sr}$; in each case it acts to move the leg back towards $\phi = 0$. The reflex becomes more strongly
Figure 3.4: Single leg position vs. time. A perturbation is introduced by fixing $\dot{\phi} = 0$ between $t = 20$ and $t = 22$. *(Top)* Using the open-loop equation (3.5), we see that the perturbation permanently alters the oscillatory range of the leg. *(Bottom)* Using the stretch reflex equation (3.6), the perturbation is rapidly erased, and the original oscillatory range is recovered. *Parameters:* $\tau = k = 1; \gamma = 4; \theta = 0; w^* = 2; w = -3; I_o = 1; \phi_{sr} = 0.5; g = 2; V = 1.$

activated the further forward or back the leg is moved.

This simple addition stabilizes the system, allowing rapid recovery from perturbations. Figure 3.4 shows the results of numerical integration of equations (3.5) and (3.6). In each case, a perturbation has been introduced by setting $\dot{\phi} = 0$ between $t = 20$ and $t = 22$. As the upper plot demonstrates, the open-loop equations may be pushed into a new range by such a perturbation, while the lower plot shows the system recovering to the original cycle under the influence of the stretch reflex.
3.5 Two legs

To obtain antiphase coupling between two legs, the legs’ oscillators may be coupled in the manner indicated in Figure 3.3. However, this is an open-loop form of coupling: each oscillator receives information only about the command signals being sent to each leg, not about the leg’s actual position. To close the loop, the legs are coupled through their actual positions \( \phi \), using a mixture of excitatory and inhibitory connections as shown in Figure 3.5. (The idea of coupling oscillating limbs through their current positions rather than through their command signals is discussed in [102].) Labelling the two legs \( L \) and \( R \) (for “left” and “right,” since we want this type of antiphase coupling for legs on opposite sides of the body), the relevant ODEs are as follows. Denote the leg positions by \( \phi_l \), with \( l \in \{ L, R \} \). Each leg has its own individual neural oscillator, with variables \( x_{l,1}, x_{l,2}, \alpha_{l,1}, \) and \( \alpha_{l,2} \), and associated with each neuron in the oscillator is its firing rate output, \( y_{l,i} = f(\gamma(x_{l,i} - \alpha_{l,i}) + \theta) \) for \( i = 1, 2 \). There are ten equations in total:

\[
\begin{align*}
\tau \dot{x}_{L,1} &= -x_{L,1} + wy_{L,2} + I_o - w_c \phi_R \\
\dot{\alpha}_{L,1} &= k(x_{L,1} - \alpha_{L,1}) \\
\tau \dot{x}_{L,2} &= -x_{L,2} + wy_{L,1} + I_o + w_c \phi_R \\
\dot{\alpha}_{L,2} &= k(x_{L,2} - \alpha_{L,2}) \\
\dot{\phi}_L &= V([y_{L,1} - gS(\phi_L)] - [y_{L,2} - gS(-\phi_L)]) \\
\tau \dot{x}_{R,1} &= -x_{R,1} + wy_{R,2} + I_o - w_c \phi_L \\
\dot{\alpha}_{R,1} &= k(x_{R,1} - \alpha_{R,1}) \\
\tau \dot{x}_{R,2} &= -x_{R,2} + wy_{R,1} + I_o + w_c \phi_L \\
\dot{\alpha}_{R,2} &= k(x_{R,2} - \alpha_{R,2}) \\
\dot{\phi}_R &= V([y_{R,1} - gS(\phi_R)] - [y_{R,2} - gS(-\phi_R)]).
\end{align*}
\]

where \( S(x) \) is as defined in equation (3.7), and the other parameter values are as previously discussed.

As Figure 3.6 shows, the closed-loop stretch reflex coupling both maintains an antiphase relationship between the two legs and recovers quickly from perturbations.

3.6 Six legs

The tripod gait is generated simply by extending the coupling described in the previous sections to a set of six legs. In the tripod gait, each leg has an antiphase relationship to its ipsilateral (same side) and contralateral (opposite side) neighbours. The coupling described in section 3.5
Figure 3.5: Antiphase coupling for two legs. Filled circles indicate excitatory connections, open circles indicate inhibitory connections; symbols next to connections represent coupling strengths. The upper oscillator produces a pair of outputs $y_{L.1}, y_{L.2}$, which drives the leg position $\phi_L$ according to equation 3.6. Similarly, the lower oscillator drives leg position $\phi_R$. 
Figure 3.6: Trajectories of two legs, coupled as indicated in Figure 3.5. Perturbations are introduced by setting $\dot{\phi}_1 = 0$ for $t \in [20, 22]$ and setting $\dot{\phi}_2 = 0$ for $t \in [35, 40]$. Note that the system recovers rapidly from these disturbances, returning to the original oscillatory solution. 

Parameters: $\tau = k = 1; \gamma = 4; \theta = 0; w^* = 2; w = -3; w_c = 0.25; I_o = 1; \phi_{sr} = 0.5; g = 2; \text{ and } V = 1.$
Figure 3.7: Coupling pattern for tripod gait generating network. The dashed lines indicate antiphase coupling of the type shown in Figure 3.5: this is made explicit in equations (3.18-3.23).

is used to connect each neighbouring pair of legs, as shown in Figure 3.7.

The full set of equations used to generate the tripod gait is as follows. Each leg is represented by a position $\phi_l$, with $l \in \{L_1, L_2, L_3, R_1, R_2, R_3\}$. The neural oscillator associated with each leg has four variables, denoted $x_{l,1}$, $x_{l,2}$, $\alpha_{l,1}$, and $\alpha_{l,2}$; associated with each analog neuron in the oscillator is its firing rate output. $y_{l,i} = f(\gamma(x_{l,i} - \alpha_{l,i}) + \theta)$ for $i = 1, 2$. There are thirty differential equations in total, five for each leg:

\[
\tau \dot{x}_{l,1} = -x_{l,1} + wy_{l,2} + I_o - w_c I_l
\]
\[
\dot{\alpha}_{l,1} = k(x_{l,1} - \alpha_{l,1})
\]
\[
\tau \dot{x}_{l,2} = -x_{l,2} + wy_{l,1} + I_o + w_c I_l
\]
\[
\dot{\alpha}_{l,2} = k(x_{l,2} - \alpha_{l,2})
\]
\[
\dot{\phi}_l = V([y_{l,1} - gS(\phi_l)] - [y_{l,2} - gS(-\phi_l)]).
\]

where as before, $\tau$ is the neural activation time constant, $k$ is the adaptation rate, $w < 0$ is the strength of the mutual inhibition within each neural oscillator, $I_o$ is the tonic (constant) excitation to which the neurons are subjected, $w_c > 0$ is the strength of the coupling between legs, the stretch reflex function $S(z)$ is as defined in equation (3.7), $g$ is the stretch reflex gain, and $V$ scales the rate of leg movement. The actual coupling between legs occurs through the $I_l$ terms, whose values define which neighbouring legs influence leg $l$'s position:
Figure 3.8 shows the result of numerically integrating the thirty equations defined by (3.18-3.22), with the legs started at random positions, \( \phi_i \in [-1, 1] \) and the other variables initialized to small random values. The differential equations have been implemented in Simulink, a MATLAB package for ODE solving. Runs consistently show that a tripod gait is quickly established, and the same gait is rapidly resumed if the legs are perturbed. Note that we do not need to use a different value of coupling strength \( w_c \) for the central legs \( L_2 \) and \( R_2 \), even though they have three neighbours where the other legs have only two.

Figure 3.9 shows the result of adding white noise to the velocity of each leg, so that the ODE for each leg position becomes

\[
\dot{\phi}_i = V([y_{i,1} - gS(\phi_i)] - [y_{i,2} - gS(-\phi_i)]) + \sigma(t)
\]

where \( \sigma(t) \) is a stochastic band-limited white noise term (supplied, in this case, by the Simulink ODE solver). The proper phase relationships are maintained despite the presence of the noise.

### 3.7 Future directions

The outputs of the network described in this chapter have been used to control the walking of a six-legged robot named Kafka, built during my Master’s work [72]; the implementation was carried out by Joseph Yang [100]. Unfortunately, technical difficulties prevented the use of real-time feedback, so that the legs were controlled in an open-loop manner. It should be possible to correct this difficulty and feed the leg position feedback into the differential equations.

In [100], the set of ODEs were numerically integrated on a 486 computer in real time. However, the form of the equations is such that it should be possible to implement them in analog electronics directly [103, 104], meaning that no external computer would be required to carry out the numerical integration.

In terms of analysis, the basic features of the individual oscillators would be preserved if they were replaced by two-dimensional van der Pol oscillators (see [65]), or some other simple form of relaxation oscillator; this would halve the dimensionality of the system and facilitate more detailed analysis of issues such as the global stability of the limit cycles seen in the network.
Figure 3.8: Tripod gait, generated by six coupled oscillators. The upper plot shows one set of three legs, \( \{\phi_{L1}, \phi_{R2}, \phi_{L3}\} \), while the lower plot shows the other tripod, \( \{\phi_{R1}, \phi_{L2}, \phi_{R3}\} \). Perturbations are introduced by setting \( \phi_{L1} = \phi_{R1} = 0 \) for \( t \in [15, 20] \) and \( \phi_{L2} = 0 \) for \( t \in [30, 35] \). Parameters: \( \tau = k = 1; \gamma = 4; \theta = 0; w^* = 2; w = -3; w_c = 0.1; \phi_{sr} = 0.5; g = 2; \) and \( V = 1 \).
First tripod: legs L1, R2, L3
Second tripod: legs R1, L2, R3

Figure 3.9: Tripod gait, generated by six coupled oscillators, in the presence of noise. White noise with power 0.005 has been added to the velocity commands; see text. The upper plot shows one set of three legs, \( \{ \phi_{L1}, \phi_{R2}, \phi_{L3} \} \), while the lower plot shows the other tripod, \( \{ \phi_{R1}, \phi_{L2}, \phi_{R3} \} \). Perturbations are introduced by setting \( \phi_{L1} = \phi_{R1} = \sigma(t) \) for \( t \in [15, 20] \) and \( \phi_{L2} = \sigma(t) \) for \( t \in [30, 35] \); during these periods, the affected legs drift under the influence of the white noise \( \sigma(t) \) (independent noise sources are provided for each leg). Parameters: \( \tau = k = 1; \gamma = 4; \theta = 0; \quad w^* = 2; \quad w = -3; \quad w_c = 0.1; \quad \phi_{cr} = 0.5; \quad g = 2; \) and \( V = 1 \).
Chapter 4

Oscillations in pools of coupled neurons

4.1 Local abbreviations

The following table lists the abbreviations used in this chapter; as discussed in section 1.7, they are “local” in the sense that they apply only within this chapter.

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Definition</th>
<th>Section</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \bar{\tau}_i ) (= \bar{\tau}(C_i) )</td>
<td>( [1 + k_r C_i]^{-1} )</td>
<td>4.4.1</td>
</tr>
<tr>
<td>( \bar{I}_i ) (= \bar{I}(C_i) )</td>
<td>( I_o - k_l C_i )</td>
<td>↓</td>
</tr>
<tr>
<td>( \tau_{ad} )</td>
<td>( [\beta (k_l + k_r) + \frac{1}{\tau_c}]^{-1} )</td>
<td>↓</td>
</tr>
<tr>
<td>( \bar{I}_1 )</td>
<td>( I_o - k_l C_1 - F_2 K_o \tau_s )</td>
<td>4.4.2</td>
</tr>
<tr>
<td>( \bar{I}_2 )</td>
<td>( I_o - k_l C_2 - F_1 K_o \tau_s )</td>
<td>↓</td>
</tr>
<tr>
<td>( Z_1 )</td>
<td>( k_l + k_r/2 )</td>
<td>↓</td>
</tr>
<tr>
<td>( Z_2 )</td>
<td>( (I_o - 1/2)/(1 + K_o \tau_s) )</td>
<td>↓</td>
</tr>
<tr>
<td>( Z_3 )</td>
<td>( Z_1/(1 - K_o^2 \tau_s^2) )</td>
<td>↓</td>
</tr>
<tr>
<td>( Z_4 )</td>
<td>( K_o \tau_s Z_1/(1 - K_o^2 \tau_s^2) )</td>
<td>↓</td>
</tr>
<tr>
<td>( Z_5 )</td>
<td>( [\beta Z_4 + 1/\tau_c]^{-1} )</td>
<td>↓</td>
</tr>
<tr>
<td>( Z_6 )</td>
<td>( \frac{1}{2} (I_o k_r + k_l) - (I_o - 1)/\tau_c )</td>
<td>4.5</td>
</tr>
<tr>
<td>( Z_7 )</td>
<td>( \beta (I_o - 1/2) )</td>
<td>4.6</td>
</tr>
<tr>
<td>( Z_8 )</td>
<td>( I_o - \frac{1}{2} - (I_o - 1)/K_o \tau_s ) ( /Z_1 )</td>
<td>↓</td>
</tr>
<tr>
<td>( Z_9 )</td>
<td>( (k_l + k_r)/K_o \tau_s Z_1 )</td>
<td>↓</td>
</tr>
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</table>
4.2 Introduction and background

Chapter 2 presented the concept of half-center oscillators, groups of neurons which oscillate due to mutual inhibition combined with a limited duration of inhibitory activity. The limited duration of inhibition was provided by a form of spike-frequency adaptation, and an analog neuron model was used.

Analog neurons, while useful for many analytical and practical purposes, are not generally regarded as realistic models for individual neurons [39, 105], since many significant neural processing events happen on such short time scales that only a small number of action potentials is involved, making the notion of an "average firing rate" problematic [33]. Rather than viewing an analog neuron as a single neuron whose output is its own individual firing rate, we can conceive of an analog neuron as representing a population of individually spiking neurons, and view its output as a firing rate averaged over the entire population [41, 105, 106, 107]; a time average for a single neuron is replaced with an ensemble (population) average for a group of neurons, thus avoiding the problem of the long averaging times required to define a meaningful rate in the single-neuron case [105].

In this chapter, this population-averaging approach will be used to create and analyze a half-center oscillator made up of two populations of individually spiking neurons. For simplicity, I will consider only the case in which each population of neurons has no internal connections, and has all-to-all connections between the pools, as shown in Figure 4.1. Once again, the factor which limits the duration of inhibition will be spike-frequency adaptation, this time implemented at the level of individual neurons rather than in the analog neuron equations.

Two pools coupled as shown in Figure 4.1 will, given that the individual neurons display spike-frequency adaptation and that the mutual inhibition is sufficiently strong, produce alternating bursts of firing in the two pools, as shown in Figure 4.2.

For large populations of individual neurons, each of which has its own internal dynamics, the complete system representing the two coupled pools has a large number of dimensions. In this chapter, I will show that it is possible to reduce this high-dimensional system to a two-dimensional set of dynamics, a drastic simplification. These simplified dynamics allow quite accurate predictions to be made about the behaviour of the full, high-dimensional system, including the period of the oscillations and the approximate range of coupling strengths for which oscillations will occur.

The connection to robotics is the prospect of using oscillators of the type described in this chapter to produce rhythms required for locomotion (or other functions) in a robot. Such oscillators would enjoy one of the central advantages of biological systems, namely redundancy: since the oscillations come from a large number of individual units, loss of any single element would not halt the oscillations.
Figure 4.1: Two coupled pools of individually spiking neurons. There are no connections within each pool. Between the pools, coupling is all-to-all: each neuron in pool 1 sends its output to every neuron in pool 2 with an inhibitory coupling strength $K$, and vice versa. Each pool receives a constant (tonic) input, $I_0$; see section 4.3.

4.3 Neuron model

4.3.1 Dimensional form

The model neurons used in this chapter are of the integrate-and-fire (IF) type (see section 1.5.4). The model described in [108], includes spike-frequency adaptation, an effect wherein an accumulating ionic concentration causes the neuron to fire less rapidly; the equations for an individual neuron are

\[
\begin{align*}
C \frac{dV}{d\tilde{t}} &= -g(V - V_r) + \tilde{I}(\tilde{t}) - G[Ca^{2+}](V - V_K) - (V_{th} - V_{reset})\delta(V - V_{th}) \quad (4.1) \\
d[Ca^{2+}] / d\tilde{t} &= \Delta Ca \delta(V - V_{th}) - [Ca^{2+}] / \tau_{Ca}, \quad (4.2)
\end{align*}
\]

where $V$ is the membrane voltage (see section 1.4), $[Ca^{2+}]$ is the concentration of calcium ions inside the neuron, $\delta(\cdot)$ is the Dirac delta function, and $\tilde{t}$ is the (dimensional) time, marked with a tilde to distinguish it from the dimensionless “time” appearing below in section 4.3.2. The meanings and typical ranges of values for the other parameters appearing in (4.1-4.2) are given in Table 4.1. Note that a very similar adaptation model for IF neurons is described in [33].

In the model, each time the membrane potential $V$ hits some threshold value $V_{th}$, a spike is generated and $V$ is reset to $V_{reset}$; here, I will always take $V_{reset} = V_r$, so that the voltage is reset to the resting potential after each spike. Equation (4.2) shows that each spike produces an increment of size $\Delta Ca$ in the concentration of calcium inside the cell. Clearly this is an
Figure 4.2: Alternating bursts of firing in two coupled pools of 100 neurons each. This is what is known as a "raster plot": each horizontal line represents one of the 200 neurons in the population, with spiking times indicated by dots. One pool of neurons fires rapidly, suppressing firing in the other pool, then reduces its firing rate due to spike-frequency adaptation. When the firing rate drops far enough, the previously suppressed pool is able to recover and dominate the previously firing pool. The figure shows the output from the nondimensionalized model presented in section 4.3.2; the quantity $t$ is thus a dimensionless time, scaled by the membrane time constant. Parameters: $I_o = 10; K_o = 48; \tau_s = 0.025 (K_o \tau_s = 1.2); k_r = 1.2; k_l = 0.75; \beta = 0.2; \tau_c = 2.5$. (These parameters will be discussed in section 4.3.2.) All numerical simulations in this chapter are carried out using the fourth-order Runge-Kutta method with maximum step size $h = 10^{-4}$ (dimensionless).
approximation, since real cells do not change ionic concentrations instantaneously. However, the influx of calcium in real cells is sufficiently rapid that it may be approximated by a step; see [21, 108]. The effect of incoming calcium is to activate a calcium-dependent potassium current: the presence of calcium acts to open potassium channels in the cellular membrane, making the membrane more permeable to potassium ions and thus inducing a current flow across the membrane. (See section 1.4.1 for more information about flows of ions across membranes.)

For a constant input current $\bar{I}(\bar{t}) = \bar{I}_0$, an adapting neuron fires at some initial rate that decreases as the calcium accumulates: see Figure 4.3. Denoting the initial rate $f_{init}$ and the final steady-state rate $f_{ss}$, the degree (or “strength”) of the adaptation effect may be summarized as

$$F_{adap} = \frac{f_{init} - f_{ss}}{f_{init}} = 1 - \frac{f_{ss}}{f_{init}}.$$  \hfill (4.3)

A value of $F_{adap} = 0$ indicates no adaptation, while $F_{adap} = 1$ indicates “complete” adaptation, in which the neuron ceases firing entirely after it has adapted. Biological neurons rarely exhibit complete adaptation; typical experimental values for $F_{adap}$ range from 0 to 0.6 [21, 108].

### 4.3.2 Dimensionless form

Converting equations (4.1-4.2) to a dimensionless form helps to clarify the analysis, and the remainder of this chapter will deal only with dimensionless quantities.

Let us nondimensionalize $V$ by setting $v = (V - V_r)/(V_{th} - V_r) = (V - V_r)/\theta$, where $\theta \equiv V_{th} - V_r$. This maps the rest (and reset) voltage $V_r$ to $v = 0$, and the spiking voltage $V_{th}$ to $v = 1$. A sensible time scale is provided by the membrane time constant, $\tau_m \equiv C/g$; define a dimensionless “time” $\bar{t} = t/\tau_m$. The calcium concentration $[\text{Ca}^{2+}]$ may be nondimensionalized with respect to

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Meaning</th>
<th>Value</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>$C$</td>
<td>membrane capacitance</td>
<td>1 $\mu$F</td>
<td>1 $\mu$F is standard</td>
</tr>
<tr>
<td>$g$</td>
<td>membrane conductance</td>
<td>0.05 mS</td>
<td>$S = \text{Siemens} = 1/\text{Ohms}$</td>
</tr>
<tr>
<td>$\tau_m$</td>
<td>membrane time constant</td>
<td>20 ms</td>
<td>$\tau_m \equiv C/g$</td>
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<tr>
<td>$V_r$</td>
<td>resting potential</td>
<td>-70 mV</td>
<td></td>
</tr>
<tr>
<td>$V_{reset}$</td>
<td>reset potential</td>
<td>-70 mV</td>
<td>Here, $V_{reset} = V_r$</td>
</tr>
<tr>
<td>$\bar{I}(\bar{t})$</td>
<td>input current</td>
<td>0 to 10 $\mu$A</td>
<td></td>
</tr>
<tr>
<td>$G$</td>
<td>Ca-dependent K conductance</td>
<td>0 to .1 mS/$\mu$M</td>
<td></td>
</tr>
<tr>
<td>$V_K$</td>
<td>K reversal potential</td>
<td>-80 mV</td>
<td></td>
</tr>
<tr>
<td>$\Delta_{Ca}$</td>
<td>$[\text{Ca}^{2+}]$ increment</td>
<td>.2 $\mu$M</td>
<td></td>
</tr>
<tr>
<td>$\tau_{Ca}$</td>
<td>$[\text{Ca}^{2+}]$ decay time constant</td>
<td>50 to 100 ms</td>
<td></td>
</tr>
<tr>
<td>$V_{th}$</td>
<td>spiking threshold</td>
<td>-54 to -40 mV</td>
<td></td>
</tr>
</tbody>
</table>

Table 4.1: Parameters for adapting integrate-and-fire model. dimensional form. The values cited are taken from [108].
Figure 4.3: Response of adapting IF neuron to constant input current. (Top) The "instantaneous firing rate." $f$, calculated as $1/t^*$, where $t^*$ is the interval between successive spikes. The initial rate $f_{init} = 374$ Hz, while the steady-state rate $f_{ss} = 218$ Hz; the strength of adaptation is thus $F_{adap} = 1 - f_{ss}/f_{init} = 0.42$. (Bottom) The calcium concentration in the neuron. Note that $[Ca^{2+}]$ rises to cycle around a steady state value. Parameters: $C = 1 \mu F; \tau_m = 20$ ms; $V_r = -70$ mV; $V_{th} = -54$ mV; $\Delta Ca = 0.2 \mu M; V_K = -80$ mV; $I_o = 6.4 \mu A; G = .06$ mS/\mu M; $\tau_Ca = 50$ ms.
CHAPTER 4. OSCILLATIONS IN POOLS OF COUPLED NEURONS

<table>
<thead>
<tr>
<th>$V_{th}$ [mV]</th>
<th>$\tau_{Ca}$ [ms]</th>
<th>$G$ [mS/µM]</th>
<th>$k_r$</th>
<th>$k_I$</th>
<th>$\beta$</th>
<th>$\tau_c$</th>
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<td>-54</td>
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<td>.03</td>
<td>.6</td>
<td>.375</td>
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<td>2.5</td>
</tr>
<tr>
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<td>.75</td>
<td>.2</td>
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<td>1.5</td>
<td>.9375</td>
<td>.2</td>
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<tr>
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<td>.06</td>
<td>1.2</td>
<td>.75</td>
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<td>5</td>
</tr>
<tr>
<td>-40</td>
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<td>1.2</td>
<td>.4</td>
<td>.2</td>
<td>5</td>
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</table>

Table 4.2: Values of the dimensionless constants for various choices of the dimensional parameters. The dimensional parameters not listed are $C = 1 \mu F$, $\tau_m = 20$ ms. $V_{rest} = V_{reset} = -70$ mV. $\alpha = 0.2 \mu M$. and $V_K = -80$ mV.

an arbitrary reference level. $[Ca^{2+}]_{ref}$: set $c = [Ca^{2+}]/[Ca^{2+}]_{ref}$, with $[Ca^{2+}]_{ref} = 1 \mu M$.

These definitions lead to the single-neuron equations

\[
\dot{v} = -[1 + k_r c] v + I(t) - k_I c - \delta (v - 1) \tag{4.4}
\]
\[
\dot{c} = \beta \delta (v - 1) - c/\tau_c, \tag{4.5}
\]

where $\dot{x} = dx/dt$. The dimensionless parameters are given by $k_r = G[Ca^{2+}]_{ref} \tau_m/C$, $k_I = k_r (V_r - V_K)/\theta$. $\beta = \Delta Ca/[Ca^{2+}]_{ref}$. and $\tau_c = \tau_{Ca}/\tau_m$; all of these are positive, except in the nonphysiological case where $V_r < V_K$. The dimensionless input current is given by $I(t) = I(t) (\tau_m/C \theta) - (V_{reset} - V_r)/\theta$, which becomes just $I(t) = I(t) (\tau_m/C \theta)$ since $V_{reset} = V_r$. Table 4.2 shows typical values for the dimensionless parameters.

The dimensionless voltage rises until it reaches $v = 1$, at which point it is reset to $v = 0$. A convenient way to rewrite equations (4.4-4.5) is

\[
\dot{v} = -v/\tau + \bar{I} - \delta (v - 1) \tag{4.6}
\]
\[
\dot{c} = \beta \delta (v - 1) - c/\tau_c, \tag{4.7}
\]

where $\bar{\tau}(c) = [1 + k_r c]^{-1}$ and $\bar{I}(t, c) = I(t) - k_I c$. These definitions cast the integrate-and-fire model into its most basic form, making the between-spikes solution particularly clear. Setting $t = 0$ when $v$ is reset to $v = 0$, and taking a constant current $\bar{I} = \bar{I}_o$, the solution to (4.6) for a given (constant) $c$ is:

\[
v(t) = \bar{I}_o \bar{\tau}(1 - e^{-t/\bar{\tau}}). \tag{4.8}
\]

The solution asymptotically approaches $v_\infty = \bar{I}_o \bar{\tau}$. Provided that $v_\infty > 1$, $v(t)$ will reach the
firing threshold \( v = 1 \) at some time \( t^* \), which is easily determined from (4.8) to be

\[
t^* = -\frac{\tau}{I_0} \ln \left( 1 - \frac{1}{I_0} \right).
\]  (4.9)

Using this, we can define an “instantaneous firing rate” as the reciprocal of the interspike interval \( t^* \):

\[
f = \frac{1}{t^*} = \left[ -\frac{\tau}{I_0} \ln \left( 1 - \frac{1}{I_0} \right) \right]^{-1} \mathcal{H}(I_0 - 1),
\]  (4.10)

where \( \mathcal{H}(\cdot) \) is the Heaviside step function (\( \mathcal{H}(x) = 1 \) if \( x \geq 1 \), otherwise \( \mathcal{H}(x) = 0 \)).

### 4.3.3 Synaptic coupling

To form networks of the above-described IF neurons, I coupled them using simulated synaptic currents. Each time a neuron outputs a spike, its associated synaptic current is increased by a synaptic kernel \( \gamma(s) \) for all times \( t > s \). If \( \{t_i^m\} \) is the set of times at which neuron \( i \) has fired, its synaptic output current at time \( t \) is

\[
I_i^{\text{syn}}(t) = \sum_m \gamma(t - t_i^m).
\]

This is a common method of simplifying the intricacies of synaptic dynamics: see [105, 107, 36].

Here I will use the synaptic kernel \( \gamma(s) = e^{-s/\tau_s} \mathcal{H}(s) \), where \( \tau_s \) is the synaptic decay time constant. (Since I am working with the nondimensionalized model, \( \tau_s \) is also dimensionless.)

This kernel corresponds to a synapse with a very rapid onset time and an exponential decay: the modelled “neurotransmitters” begin to diffuse across the synaptic cleft instantaneously after the neuron generates a spike, and the process persists for some (typically brief) time, with the synaptic influence decaying exponentially.

In the numerical simulations, I have generally taken \( \tau_s = 0.025 \). Assuming a membrane time constant of 20 ms, this corresponds to a dimensional synaptic time constant of 0.5 ms, which implies a quite rapid decay in synaptic influence, meaning spikes in the distant past have a negligible effect on the present value of the synaptic current. Figure 4.4 shows the synaptic current output for a single IF neuron.

### 4.4 Population activity

The collective firing of a group (“pool”) of neurons produces a net activity for the pool: if we observe the spike train formed by superimposing the spiking times of all neurons in a pool, we can average over a short time window and define an average firing rate for the pool. Following
Figure 4.4: Synaptic current output from a single neuron. $I_{\text{syn}}(t) = \sum_{m} \gamma(t - t_{m}^{\text{sp}})$, with synaptic kernel $\gamma(s) = e^{-s/\tau_{s}} H(s)$. The rapid synaptic decay time means that, for this firing rate, the synaptic output is essentially a function only of the most recent spike time. Parameters: $I_{0} = 10$; $\tau_{s} = 0.025$. 
Gerstner [105], I define this average firing rate, or "activity," as

\[
F_i(t) = \frac{1}{\Delta t} \frac{\sum n_{\text{spike}}^{(i)}(t; t + \Delta t)}{N_i},
\]

where \( F_i \) is the population activity for pool \( i \), \( N_i \) is the number of neurons in the population, \( \Delta t \) is some small time interval, and \( n_{\text{spike}}^{(i)}(t; t + \Delta t) \) is the total number of spikes generated by the pool in the interval from \( t \) to \( t + \Delta t \). Figure 4.5 shows a raster plot (the stacked firing records of all neurons in a population, plotted against time) illustrating this definition. For very large populations, we may formally consider the limit

\[
F_i(t) = \lim_{N_i \to \infty} \lim_{\Delta t \to 0} \frac{1}{\Delta t} \frac{\sum n_{\text{spike}}^{(i)}(t)}{N_i}.
\]

In this limit, \( F_i(t) \) is independent of the choice of \( \Delta t \), and at every time an "instantaneous" firing rate is defined. For the smaller networks I will consider here, with population sizes in the hundreds of neurons, the choice of \( \Delta t \) does make a difference when calculating \( F_i \) from the output of a simulation: \( F_i \) becomes highly discontinuous if \( \Delta t \) is made too small, since in a finite population some small slices of time will contain no firings at all. However, it is possible to analyze the system's behaviour under the assumption that pool activities are smooth functions: as we will see, this approach yields reasonable results despite the finite population size.

### 4.4.1 Activity in a single population

The activity observed in a population of neurons depends on whether the population is displaying asynchronous or synchronous behaviour. In asynchronous firing, the firing times of the individual neurons are uncorrelated, and we may assume that the population activity is a simple average of the individual firing rates. When the population becomes synchronized, the firing times of the neurons are strongly correlated; in the case of a fully synchronized system, all neurons fire simultaneously. The population activity in the synchronous case is a series of brief pulses of high activity, separated by intervals of no activity (see [105, 109]).

For a single pool of uncoupled neurons, the population could maintain a synchronous state only if all neurons had identical initial conditions and no noise was present in the system; since noise is in fact present in all real neural systems, synchrony is not realistic for a pool of uncoupled neurons.

Given that the population is in an asynchronous state, we may calculate the population activity as the average of the instantaneous firing rates of the individual neurons:

\[
F_i = \frac{1}{N_i} \sum_{j \text{ in pool } i} f_j.
\]
Figure 4.5: Raster plots illustrating population activity. (Top) Each horizontal line represents one of the 100 neurons in the population, with spiking times indicated by dots. (Bottom) If we consider the slice of time from $t = 2$ to $t = 2.01$ ($\Delta t = 0.01$), we see that 16 neurons fire in the interval; the population activity at that instant is thus $F = 16/(100 \cdot 0.01) = 16$ Hz. Considering the interval between $t = 2.01$ and $t = 2.02$ illustrates the effect of a finite population size: the activity in this instant is only $F = 4$ Hz, implying that $F(t)$ is a very jagged function. It is generally necessary to take a larger sampling time (for example, $\Delta t = 0.1$), to obtain a smooth $F(t)$. 

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where \( N_i \) is the number of neurons in the \( i \)-th pool, and \( f_j \) is the firing rate of the \( j \)-th neuron. For a constant input current \( I_o \), each neuron has a firing rate given by (4.10). If we define the pool-averaged value of \( c \) as

\[
C_i = \frac{1}{N_i} \sum_{j \text{ in pool } i} c_j.
\]

(4.14)

then the population activity in a pool of identical neurons is just

\[
F_i(C_i) = \left[ -\tau(C_i) \ln \left( 1 - \frac{1}{\bar{I}_o(C_i)\tau(C_i)} \right) \right]^{-1} \mathcal{H}(\bar{I}_o(C_i)\tau(C_i) - 1).
\]

(4.15)

where \( \bar{\tau}(C_i) = [1 + k_r C_i]^{-1} \) and \( \bar{I}_o(C_i) = I_o - k_f C_i \) are the pool-averaged membrane time constant and effective input current, respectively.

From (4.7), the dynamics of the pool-averaged calcium level is given by

\[
\dot{C}_i = \frac{1}{N_i} \beta \sum_{j=1}^{N_i} \delta(v_j - 1) - \frac{1}{N_i} \sum_{j=1}^{N_i} c_j / \tau_c.
\]

(4.16)

For firing which is rapid relative to the calcium decay rate, as is generally the case, we may replace the individual delta functions in (4.16) with the firing rate itself [108], obtaining

\[
\dot{C}_i \approx \frac{1}{N_i} \beta \sum_{j=1}^{N_i} f_j - \frac{1}{N_i} \sum_{j=1}^{N_i} c_j / \tau_c.
\]

(4.17)

which yields

\[
\dot{C}_i \approx \beta F_i(C_i) - C_i / \tau_c.
\]

(4.18)

Note that the population activity is a function of the pool-averaged calcium level. The problem of determining the activity of pool \( i \) is thus reduced to finding \( C_i \). Equation (4.15) may be made more tractable using the approximation [108]

\[
[-\ln(1 - x)]^{-1} = \frac{1}{x} - \frac{1}{2} + O(x)
\]

(4.19)

for \( x \ll 1 \). For input \( I_o \), if \( \bar{I}_o \bar{\tau} \gg 1 \), we may use (4.19) in (4.15) to obtain

\[
F_i(C_i) \approx \frac{1}{\bar{\tau}} \left( \bar{I}_o \bar{\tau} - \frac{1}{2} \right) \mathcal{H}(\bar{I}_o \bar{\tau} - 1)
\]

(4.20)

\[
= \left( \bar{I}_o - \frac{1}{2\bar{\tau}} \right) \mathcal{H}(\bar{I}_o \bar{\tau} - 1)
\]

(4.21)
\[ C_i(t) = \beta \tau_{ad} \left( I_o - \frac{1}{2} \right) \left( 1 - e^{-t/\tau_{ad}} \right). \quad (4.25) \]

where it has been assumed that \( \bar{I}_o \bar{r} > 1 \) throughout, allowing us to drop the Heaviside step functions.

Equations (4.22) and (4.25) predict the time course of the population activity for a pool of uncoupled identical neurons with constant input \( I_o \). As \( t \to \infty \),

\[ C_i(t) \to C_i^{ss} = \beta \tau_{ad} \left( I_o - \frac{1}{2} \right). \quad (4.26) \]

and the activity approaches its steady-state value

\[ F_i^{ss} = F_i(C_i^{ss}) \approx \left( I_o - \frac{1}{2} \right) \left[ 1 - \beta \tau_{ad} \left( k_I + \frac{k_r}{2} \right) \right]. \quad (4.27) \]

Figure 4.6 shows the population activity and pool-averaged calcium level from a numerical simulation of a pool of uncoupled neurons; the simulation values are compared with the predictions of equations (4.22) and (4.25).

Note that the time courses of the pool activity and calcium level shown in Figure 4.6 resemble the firing rate and calcium concentration plots for a single neuron, shown in Figure 4.3. An uncoupled pool of neurons is equivalent to a single neuron in which the effect of the noise in the individual firing rates has been smoothed by averaging over the population. In fact, a pool of neurons behaves in many ways like an analog neuron, producing a collective firing rate output that may be seen as an analog value [103]. The average calcium concentration in the population effectively encodes the firing rate; this has been discussed in [20, 21].
Figure 4.6: Time course of the activity and average calcium level in an uncoupled population. 
(Top) The population activity decays exponentially with time constant \( \tau_{ad} \) to a steady-state value \( F_i^{ss} \). Dashed line: results from a numerical simulation. Solid line: predicted time course, from equation (4.22). Finite size effects mean that the population rate fluctuates in the numerical run. (Bottom) The pool-averaged calcium level increases with the same time constant, \( \tau_{ad} \), to a steady-state value \( C_i^{ss} \). Dashed line: results from a numerical simulation. Solid line: predicted time course, from equation (4.25). Parameters: \( N = 500; I_0 = 15; k_r = 1.2; k_I = 0.75; \beta = 0.2; \tau_c = 2.5. \)
4.4.2 Activity in two coupled populations

When coupling is added to the system, as described in section 4.3.3 and illustrated in Figure 4.1, the pools may no longer be in an asynchronous state. The coupling allows the possibility that synchronization will occur within or across pools. Here, I will assume that enough noise is injected into the individual neurons that any synchronizing tendencies of the coupling are overcome, and each pool remains in an asynchronous state. (In all numerical simulations in this chapter, a random voltage reset in the range \([-3, 3]\) is used, along with Poisson noise with \(\lambda^+ = \lambda^- = 50\) and \(\Delta V = 0.01\) (see sections 5.4.2 and 6.4); this high noise level does indeed suppress all synchronization effects, and the population is kept in an asynchronous state.) Asynchronous firing will allow me to calculate the activity in two coupled pools using the same approximations as in section 4.4.1.

Define an effective input current for pool \(i\), \(\tilde{I}_i\), combining the effect of its own adaptation (calcium) level with the coupling from the other pools:

\[
\tilde{I}_i(t) = I_o - k_t C_i - \sum_j N_j K_{ji} \int_0^\infty \gamma(s) F_j(t - s) ds.
\]  
(4.28)

where \(K_{ji}\) is the coupling strength from pool \(j\) to pool \(i\), \(\gamma(s)\) is the synaptic coupling kernel, and \(F_j\) is the activity in pool \(j\). If the pool activities vary slowly compared to the synaptic time constant, we may write

\[
\tilde{I}_i(t) \approx I_o - k_t C_i - \sum_j F_j K_{ji} \int_0^\infty e^{-s/\tau_s} ds
\]

where \(\gamma(s) = e^{-s/\tau_s}\mathcal{H}(s)\). Here I will only consider the case of two pools with no internal coupling \((K_{ii} = 0)\), symmetric coupling between the pools \((K_{12} = K_{21} = K)\), and identical population sizes \(N_1 = N_2 = N\). For this case, the effective input currents are

\[
\begin{align*}
\tilde{I}_1 &= I_o - k_t C_1 - F_2 K_o \tau_s, \\
\tilde{I}_2 &= I_o - k_t C_2 - F_1 K_o \tau_s.
\end{align*}
\]  
(4.31)
(4.32)

where \(K_o = NK\). Note that this is essentially Gerstner’s spike response method, see [107, 109]. In [105], Gerstner carries out the analysis of the population activity of a single asynchronously firing population of neurons, and sketches the approach for multiple populations; he does not consider spike-frequency adaptation effects.
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Using the currents (4.29-4.30) in equation (4.15) gives

\[
F_1 = \left[ -\tilde{\tau}_1 \ln \left( 1 - \frac{1}{\tilde{I}_1 \tilde{\tau}_1} \right) \right]^{-1} \mathcal{H}(\tilde{I}_1 \tilde{\tau}_1 - 1), \tag{4.33}
\]

\[
F_2 = \left[ -\tilde{\tau}_2 \ln \left( 1 - \frac{1}{\tilde{I}_2 \tilde{\tau}_2} \right) \right]^{-1} \mathcal{H}(\tilde{I}_2 \tilde{\tau}_2 - 1), \tag{4.34}
\]

where for brevity I have defined \( \tilde{\tau}_i = \tilde{\tau}(C_i) = (1 + k_r C_i)^{-1} \) for \( i = 1, 2 \). For instantaneous values of \( C_1 \) and \( C_2 \), (4.33) and (4.34) provide a set of simultaneous equations to be solved for \( F_1 \) and \( F_2 \). (Note that \( \tilde{I}_1 = \tilde{I}_1(F_2) \) and \( \tilde{I}_2 = \tilde{I}_2(F_1) \).) In their full nonlinear form the equations are not soluble analytically, though a solution may of course be found numerically. To obtain a closed-form solution, I use the linear approximation given in equation (4.22), and write

\[
F_1 \approx \left[ I_o - \frac{1}{2} - F_2 K_o \tau_s - \left( k_I + \frac{k_r}{2} \right) C_1 \right] \mathcal{H}(\tilde{I}_1 \tilde{\tau}_1 - 1), \tag{4.35}
\]

\[
F_2 \approx \left[ I_o - \frac{1}{2} - F_1 K_o \tau_s - \left( k_I + \frac{k_r}{2} \right) C_2 \right] \mathcal{H}(\tilde{I}_2 \tilde{\tau}_2 - 1). \tag{4.36}
\]

Note that these equations are not linear, due to the presence of the step functions: they are, however, piecewise linear. Figure 4.7 shows the intersection of the \( F_1 \) and \( F_2 \) equations for a case with weak coupling, and illustrates the difference between the full nonlinear equations and the piecewise linear approximation.

For sufficiently weak coupling (see section 4.5), there is a single solution to equations (4.35-4.36), given by the intersection of the two lines in Figure 4.7 (bottom plot). Defining the local abbreviations \( Z_1 \equiv k_I + k_r / 2 \), \( Z_2 \equiv (I_o - 1/2)/(1 + K_o \tau_s) \), \( Z_3 \equiv Z_1/(1 - K_o^2 \tau_s^2) \), and \( Z_4 \equiv K_o \tau_s Z_1/(1 - K_o^2 \tau_s^2) \), this solution is given by

\[
\hat{F}_1 = Z_2 - Z_3 C_1 + Z_4 C_2, \tag{4.37}
\]

\[
\hat{F}_2 = Z_2 - Z_3 C_2 + Z_4 C_1. \tag{4.38}
\]

Assuming \( F_1(t) = \hat{F}_1 \) and \( F_2(t) = \hat{F}_2 \), we can substitute into equation (4.18) and reduce the system’s dynamics to a two-dimensional system on the \( C_1 - C_2 \) plane.

\[
\dot{C}_1 = \beta \hat{F}_1 - C_1 / \tau_c, \tag{4.39}
\]

\[
\dot{C}_2 = \beta \hat{F}_2 - C_2 / \tau_c. \tag{4.40}
\]
Figure 4.7: Determination of the activity in two coupled pools, weak coupling. (Top) The full nonlinear equations (4.33-4.34) are shown. The quantities $F_i^{\text{max}}$ refer to the maximum possible firing rate of pool $i$, given the current input level and value of $C_i$. The values $F_i^*$ refer to the firing rate in pool $i$ past which the opposite pool is completely suppressed. These quantities will be referred to later, in sections 4.5 and 4.6. (Bottom) The piecewise linear approximations, equations (4.35-4.36). Parameters: $K_0 \tau_s = 0.5$; $I_o = 10$; $k_r = 1.2$; $k_I = 0.75$; $\beta = 0.2$; $\tau_c = 2.5$; $C_1 = \hat{C}_1$, $C_2 = \hat{C}_2$. 
Defining another abbreviation, \( Z_5 \equiv [\beta Z_4 + 1/\tau_c]^{-1} \), we may write the fixed points \( \tilde{C}_i \) (at which \( \dot{C}_i = 0 \)) as

\[
\tilde{C}_1 = \tilde{C}_2 = \frac{\beta Z_2 Z_5}{1 - \beta Z_3 Z_5} = \frac{\beta \tau_c (I_o - \frac{1}{2})}{\beta \tau_c Z_1 + K_o \tau_s + 1}.
\] (4.41)

Shifting this fixed point to the origin by defining \( \tilde{C}_i = C_i - \tilde{C}_i \), we find

\[
\begin{pmatrix}
\dot{C}_1 \\
\dot{C}_2
\end{pmatrix} = 
\begin{pmatrix}
-i/Z_5 & \beta Z_3 \\
\beta Z_3 & -1/Z_5
\end{pmatrix}
\begin{pmatrix}
C_1 \\
C_2
\end{pmatrix}.
\] (4.42)

The matrix eigenvalues are \( \lambda_1 = -\beta Z_3 - 1/Z_5 \), \( \lambda_2 = \beta Z_3 - 1/Z_5 \); substituting and simplifying, these expressions become

\[
\lambda_1 = -\frac{1}{\tau_c} - \frac{\beta (k_I + k_r/2)}{1 - K_o \tau_s},
\]
\[
\lambda_2 = -\frac{1}{\tau_c} - \frac{\beta (k_I + k_r/2)}{1 + K_o \tau_s}.
\] (4.43, 4.44)

Note that the eigenvalues depend on the coupling only through the product \( K_o \tau_s \). \( \lambda_2 \) is always negative for inhibitory coupling (\( K_o > 0 \)). \( \lambda_1 \) is negative for \( K_o \tau_s < 1 \); at \( K_o \tau_s = 1 \) it has a singularity, with

\[
\lim_{K_o \tau_s \to 1^-} \lambda_1 = -\infty
\] (4.45)

and

\[
\lim_{K_o \tau_s \to 1^+} \lambda_1 = +\infty.
\] (4.46)

For \( 1 < K_o \tau_s < 1 + \beta (k_I + k_r/2) \tau_c \), \( \lambda_1 > 0 \). Figure 4.8 shows \( \lambda_1 \) and \( \lambda_2 \) plotted against \( K_o \tau_s \).

For the remainder of this section, I will consider \( K_o \tau_s < 1 \), in which case the fixed point \( \tilde{C}_1 = \tilde{C}_2 \) is locally stable: \( \lambda_1 < 0 \) and \( \lambda_2 < 0 \). The linearized dynamics indicate that the system will converge to these values starting from any point in the \( C_1 - C_2 \) plane (linear systems, of course, cannot have multiple fixed points or multistable solutions). Unfortunately, the corresponding nonlinear system is not tractable; to solve it, I would need to be able to solve for \( F_1 \) and \( F_2 \) in their full nonlinear form, equations (4.33-4.34). Therefore I cannot make rigorous claims about the behaviour of the nonlinear equations, except to argue that their behaviour will be locally similar to the behaviour of the piecewise linear equations. Based on extensive numerical simulations, it appears to be reasonable to claim that all nonoscillatory solutions in the \( C_1 - C_2 \) plane do in fact converge to the near vicinity of a fixed point that is well approximated by (4.41). See Figure 4.9 for the results of several simulations, starting with various initial values of \( C_1 \) and
CHAPTER 4. OSCILLATIONS IN POOLS OF COUPLED NEURONS

Figure 4.8: Eigenvalues for calcium dynamics. Note the singularity in $\lambda_1$ at $K_o\tau_s = 1$: the eigenvalue is undefined at this point. For $1 < K_o\tau_s < 1 + \beta (k_I + k_r/2) \tau_c = 1.675$, $\lambda_1 > 0$, after which it becomes negative once more. Parameters: $k_r = 1.2$; $k_I = 0.75$; $\beta = 0.2$; $\tau_c = 2.5$. 
Figure 4.9: Movement in \( C_1 - C_2 \) space, weak coupling. The results of several numerical simulations are shown, using different initial values for \( C_1 \) and \( C_2 \). (Recall that \( C_1 \) and \( C_2 \) are quantities averaged over each pool. Random initial values of \( c \) were assigned to each member of each pool, with the means chosen to yield approximately the desired \( C_1(0) \) and \( C_2(0) \).) Results are from simulations with two pools of 500 neurons each \( (N_1 = N_2 = 500) \). Parameters: \( I_o = 10; K_o = 20, \tau_s = 0.025 \) \( (K_o \tau_s = 0.5) \); \( k_r = 1.2; k_I = 0.75; \beta = 0.2; \tau_c = 2.5. \)

\( C_2 \), and Table 4.3 for a comparison of the predicted and actual values of \( \bar{C}_1 \) and \( \bar{C}_2 \) at various values of the coupling strength.

### 4.5 Onset of oscillations

In Figure 4.7, there is only one solution for \( F_1 \) and \( F_2 \). But as we increase the coupling strength, additional solutions appear. Figure 4.10 shows a situation with multiple intersections of the \( F_1 \)
Table 4.3: Values of $\bar{C}_1$ and $\bar{C}_2$. The “Theory” column gives the value of equation (4.41) for each coupling strength. The “Simulation” columns show results from numerical runs with two pools of 500 neurons each. Since fluctuations exist in the $C_i$ in the simulations, the value reported is the averaged over 15 time units, after the system has converged to its steady state; the numbers in square brackets are the associated standard deviations. Parameters: $I_o = 10$; $k_T = 1.2$; $k_I = 0.75$; $\beta = 0.2$; $\tau_c = 2.5$.

<table>
<thead>
<tr>
<th>$K_o\tau_s$</th>
<th>Theory $\bar{C}_1 = \bar{C}_2$</th>
<th>Simulation $C_1$ [$\sigma$]</th>
<th>Simulation $C_2$ [$\sigma$]</th>
<th>Percent error</th>
</tr>
</thead>
<tbody>
<tr>
<td>.1</td>
<td>2.676</td>
<td>2.606 [.003]</td>
<td>2.606 [.004]</td>
<td>2.7</td>
</tr>
<tr>
<td>.2</td>
<td>2.533</td>
<td>2.470 [.004]</td>
<td>2.469 [.004]</td>
<td>2.5</td>
</tr>
<tr>
<td>.3</td>
<td>2.405</td>
<td>2.347 [.004]</td>
<td>2.349 [.004]</td>
<td>2.4</td>
</tr>
<tr>
<td>.4</td>
<td>2.289</td>
<td>2.236 [.005]</td>
<td>2.238 [.005]</td>
<td>2.4</td>
</tr>
<tr>
<td>.5</td>
<td>2.184</td>
<td>2.138 [.005]</td>
<td>2.135 [.005]</td>
<td>2.2</td>
</tr>
</tbody>
</table>

and $F_2$ curves. The exact nonlinear equations (4.33–4.34) can have up to five intersections, but two of these occur only in a narrow range of parameters, and I will be concerned only with the three that also arise in the piecewise linear equations (4.35–4.36). In addition to the $F_1 = F_2$ intersection point, we now have one point with $F_1 = F_1^{\text{max}} > 0, F_2 = 0$, and another with $F_2 = F_2^{\text{max}} > 0, F_1 = 0$. The quantities $F_i^{\text{max}}$ refer to the maximum firing rate a pool may attain: this occurs in the absence of coupling, or when the other pool is completely suppressed. The other significant points marked on Figure 4.10 are $F_i^*$: these are the firing rates above which pool $i$ completely suppresses the opposite pool.

Which of the three possible solutions for the $F_i$ applies is a function of the $C_i$: when the system moves away from $C_1 = C_2$, it can enter a regime in which one pool fully suppresses the firing of the other. The firing rate solution then becomes one of the two corner points, with either $F_1 = 0$ or $F_2 = 0$. Once this occurs, oscillatory behaviour arises in the system, as follows (for concreteness, consider the case where the system moves first to a state with $F_2 = 0$):

- Owing to an imbalance in the $C_i$ levels ($C_2 > C_1$), pool 2 is fully suppressed by pool 1.
  Thus, we have $F_1 = F_1^{\text{max}} > 0, F_2 = 0$.
  - The pool dynamics become $\dot{C}_1 = \beta F_1 - C_1/\tau_c$ and $\dot{C}_2 = -C_2/\tau_c$.
  - Under these dynamics, $C_1$ increases, while $C_2$ decreases.

- At some point, $F_1^{\text{max}}$ drops below $F_1^*$, and the firing rate solution $F_1 = F_1^{\text{max}}, F_2 = 0$ no longer exists. The system moves quickly towards the only other stable solution, $F_2 = F_2^{\text{max}} > 0, F_1 = 0$.
  - The pool dynamics become $\dot{C}_2 = \beta F_2 - C_2/\tau_c$ and $\dot{C}_1 = -C_1/\tau_c$. 

Figure 4.10: Strong coupling leads to multiple intersections in $F_1, F_2$ curves. (Top) Full nonlinear equations (4.33-4.34) are shown. Note the three intersection points: the lower right ($F_1 = F_1^{\text{max}}, F_2 = 0$); the upper left ($F_1 = 0, F_2 = F_2^{\text{max}}$); and the centre ($F_1 = F_2$). The quantities $F_i^{\text{max}}$ refer to the maximum possible firing rate of pool $i$, given the current input level and value of $C_i$. The values $F_i^{*}$ refer to the firing rate in pool $i$ past which the opposite pool is completely suppressed. Note that here, $F_i^{\text{max}} > F_i^{*}$: compare this to the weak-coupling case in Figure 4.7. (Bottom) The piecewise linear approximations, equations (4.35-4.36). Parameters: $K_0\tau_s = 1.2; I_o = 10; k_r = 1.2; k_I = 0.75; \beta = 0.2; \tau_c = 2.5; C_1 = C_2 = \bar{C}_1 = \bar{C}_2$. 
This time, $C_2$ increases while $C_1$ decreases.

- When $F_2^{\text{max}}$ falls below $F_2^*$, the system transfers back to the $F_1 = F_1^{\text{max}}$, $F_2 = 0$ point, and the cycle repeats.

The existence of oscillations of the form described depends on two conditions. First, we must have $F_1^{\text{max}} > F_1^*$ at $C_1 = \bar{C}_1$; if this condition is not satisfied, there is only one possible solution for $F_1$ and $F_2$, namely the $F_1 = F_2$ intersection discussed above, and the system will tend to that fixed point. Once $F_1^{\text{max}} > F_1^*$ at this fixed point, the two new solutions for $F_1$ appear, and oscillations become possible. This condition is necessary but not sufficient: if the $F_1 = F_2$ fixed point is stable, the system may never visit the other fixed points, and oscillations may not occur. I will discuss these two conditions in turn.

At the steady-state calcium level $\bar{C}_1 = \bar{C}_2 = \bar{C}$, we have $F_1^{\text{max}} = I_0 - \frac{1}{2} - Z_1 \bar{C}$ and $F_1^* = [I_0 - 1 - (k_t + k_r)\bar{C}] / K_0 \tau_s$. $\bar{C}$ is a function of $K_0 \tau_s$—see equation (4.41)—and with some manipulation we may rearrange $F_1^{\text{max}}(K_0 \tau_s) > F_1^*(K_0 \tau_s)$ to yield

$$K_0 \tau_s > \frac{-\frac{1}{2} + \sqrt{\frac{1}{4} - 4 \tau_c (I_0 - \frac{1}{2}) Z_6}}{(I_0 - \frac{1}{2})}$$

where I have defined the local abbreviation $Z_6 \equiv \frac{3}{2}(I_0 k_r + k_t) - (I_0 - 1) / \tau_c$. The value of the right hand side, above, is less than 1 for typical parameter values. (For $I_0 = 10$, $k_r = 1.2$, $k_t = 0.75$, $\beta = 0.2$, and $\tau_c = 2.5$, we have that $F_1^{\text{max}} > F_1^*$ if $K_0 \tau_s > 0.76$.)

The point at which the fixed point $C_1 = C_2 = \bar{C}$ becomes unstable is determined simply by examining Figure 4.8. At $K_0 \tau_s = 1$, the local (linearized) dynamics around the fixed point change from a node (both eigenvalues negative) to a saddle (one negative and one positive eigenvalue). Thus, for $K_0 \tau_s > 1$, fluctuations (which are always present in the system due to the finite population size) will drive the system away from $C_1 = C_2 = \bar{C}$ and towards one of the two other fixed points, which exist provided that $K_0 \tau_s$ satisfies the inequality (4.47).

Thus, we expect that oscillations will certainly occur for $K_0 \tau_s > 1$, and this is confirmed by numerical simulations. However, oscillations in the simulations still occur for $K_0 \tau_s < 1$, and in fact tiny oscillations around $\bar{C}$ are seen even for $K_0 \tau_s$ less than the right-hand side in inequality (4.47); this mismatch between the theory and the observed behaviour is due to the linearizing approximations used to derive (4.47), and also to the presence of fluctuations in a finite population. At the other extreme, for large $K_0 \tau_s$ one pool permanently suppresses the other and oscillations cease; see section 4.7.

Figure 4.11 shows the results from a numerical simulation with $K_0 \tau_s = 1.2$, and as expected, there is an oscillatory solution (this is the same simulation whose raster plot was shown in Figure 4.2).
Figure 4.11: Oscillatory solution for two coupled pools. Results are from a simulation with two pools of 100 neurons each. (Top) $C_1$ and $C_2$ versus $t$. Note that the oscillation consists of the average calcium levels in the two pools, $C_1$ and $C_2$, rising to a high value (when the pool is active) and falling to a low value (when the pool is suppressed). (Bottom) $F_1$ and $F_2$ versus $t$, where the $F_i$ are as defined in (4.11), using a sliding window with $\Delta t = 0.15$. Note the fluctuations in firing rate; these are due to the finite population size. Parameters: $I_o = 10; K_o = 48, \tau_s = 0.025 (K_o \tau_s = 1.2); k_r = 1.2; k_f = 0.75; \beta = 0.2; \tau_e = 2.5$. 
4.6 Period of oscillations

As Figure 4.11 shows, oscillations in the two coupled pools consist of the average calcium concentration levels in the two pools \( (C_1 \text{ and } C_2) \) cycling back and forth between a high value (which I shall call \( C_h \)) and a low level (called \( C_l \)); the period of this cycle is also the period of the oscillatory bursts of firing. I will denote by \( T \) the length of a single pool's burst of firing; the period of the oscillations is thus approximately \( 2T \). To find the period, I will solve for the three unknown quantities \( (C_h, C_l, \text{ and } T) \) in terms of the system parameters.

I will assume that one pool always suppresses the other, and neglect the time during which both pools have nonzero firing rates. (As Figure 4.11 shows, this time is very brief.) To solve for \( T \), I will examine the time course of the \( C_i \) during one burst of firing; for concreteness, consider the case where \( F_1 > 0 \) and \( F_2 = 0 \). I set time \( t = 0 \) at the beginning of this period and time \( t = T \) as the instant at which the system changes back to \( F_1 = 0, F_2 > 0 \). Since pool 1 has been quiescent immediately before \( t = 0 \), its calcium level has been falling while the level in pool 2 has been rising. This implies the following set of boundary conditions:

\[
C_1(0) = C_l \quad (4.48) \\
C_2(0) = C_h \quad (4.49) \\
C_1(T) = C_h \quad (4.50) \\
C_2(T) = C_l. \quad (4.51)
\]

Since \( F_2 = 0 \), the dynamics of pool 2 become \( \dot{C}_2 = -C_2/\tau_c \), which gives \( C_2(t) = C_h e^{-t/\tau_c} \)

Applying the boundary condition at \( t = T \) gives \( C_1 = C_h e^{-T/\tau_c} \), allowing me to write an expression for \( T \) in terms of the other two variables.

\[
T = \tau_c \ln \left( \frac{C_h}{C_l} \right). \quad (4.52)
\]

The calcium dynamics of the active pool are only slightly more involved. Since pool 2 is not firing, pool 1 receives no input from the synaptic coupling between the pools. With only the global input \( I_o \) influencing it, pool 1's average firing rate is given by

\[
F_1 \simeq (I_o - 1/2) - (k_I + k_f/2) C_1. \quad (4.53)
\]

Substituting this into (4.18), we obtain

\[
\dot{C}_1 \simeq \beta F_1 - C_1/\tau_c \quad (4.54)
\]
\[
\begin{align*}
\beta (I_0 - 1/2) & - \left[ \beta (k_I + k_r/2) + \frac{1}{\tau_e} \right] C_1 = (4.55) \\
Z_7 - C_1/\tau_{ad} & = (4.56)
\end{align*}
\]

using the local abbreviations \( Z_7 \equiv \beta (I_0 - 1/2) \) and \( \tau_{ad} \equiv \left[ \beta (k_I + k_r/2) + \frac{1}{\tau_e} \right]^{-1} \). Solving (4.56) gives \( C_1(t) = Z_7\tau_{ad} + (C_1 - Z_7\tau_{ad})e^{-t/\tau_{ad}} \); applying the final condition. \( C_h = Z_7\tau_{ad} + (C_1 - Z_7\tau_{ad})e^{-T/\tau_{ad}} \). Using equation (4.52) to eliminate \( T \) gives
\[
(C_h - Z_7\tau_{ad}) = (C_1 - Z_7\tau_{ad}) \left( \frac{C_h}{C_1} \right)^{-\tau_e/\tau_{ad}} .
\]

(4.57)

One more equation is required to be able to solve the system, and it is obtained by examining the condition that causes the termination of a burst of firing in one pool (in this case, pool 1). At \( t = 0 \), \( F_1^{\text{max}} > F_1^* \): pool 1’s firing rate in the absence of inhibition is greater than the rate required to fully suppress pool 2’s firing. As \( C_1 \) rises, \( F_1^{\text{max}} \) falls; at the same time, \( C_2 \) falls and \( F_1^* \) rises. When \( F_1^{\text{max}} < F_1^* \), the fixed point of the firing rate dynamics disappears, and the system moves rapidly towards the only remaining stable fixed point (the state with \( F_2 > 0 \), \( F_1 = 0 \)). The final equation is obtained using the simplifying assumption that this transition occurs instantaneously when the two critical values of \( F_1 \) cross: \( F_1^{\text{max}}(T) = F_1^*(T) \).

The value of \( F_1^{\text{max}} \) is given by (4.33) evaluated at \( t = T \); since \( C_1(T) = C_h \), this gives
\[
F_1^{\text{max}}(T) = \left( I_0 - \frac{1}{2} \right) - (k_I + k_r/2)C_h .
\]

(4.58)

\( F_1^* \) is found by calculating the effective input current to pool 2. \( \tilde{I}_2 \equiv I_0 - k_I C_2 - F_1 K_o \tau_s \) (see equation (4.32) in section 4.4.2) and the effective membrane time constant. \( \tilde{\tau}_2 \equiv [1 + k_r C_2]^{-1} \).

Pool 2 is fully suppressed when \( \tilde{I}_2\tilde{\tau}_2 \leq 1 \) taking \( \tilde{I}_2\tilde{\tau}_2 = 1 \) at \( t = T \) yields
\[
F_1^*(T) = \frac{1}{K_o \tilde{\tau}_2} [I_0 - 1 - (k_I + k_r)C_i] .
\]

(4.59)

Equating (4.58) and (4.59) provides a simple linear relationship between \( C_h \) and \( C_i \),
\[
C_h = Z_8 + Z_9 C_i ,
\]

(4.60)

using the local abbreviations \( Z_8 \equiv \left[ I_0 - \frac{1}{2} - \frac{(I_0 - 1)}{K_o \tilde{\tau}_2} \right] / Z_1 \) and \( Z_9 \equiv (k_I + k_r)/K_o \tau_s Z_1 \).

We now have the required three equations, namely (4.52), (4.57), and (4.60). Using (4.60) we may eliminate \( C_h \) in (4.57), obtaining an expression only in terms of \( C_i \):
\[
(Z_8 + Z_9 C_i - Z_7\tau_{ad}) = (C_i - Z_8\tau_{ad}) \left( \frac{C_i}{Z_8 + Z_9 C_i} \right)^{\tau_e/\tau_{ad}} .
\]

(4.61)

Equation (4.61) is not analytically tractable, but we may write a function based on the above
Figure 4.12: Plot of $g(C_l)$ against $C_l$ for typical parameter values: the zero crossing of this function gives the solution for $C_l$. **Solid line:** Exact function, from equation (4.62); the zero crossing is at $C_l = 1.24$. **Dashed line:** First-order approximation given by equations (4.66-4.68), expanding about $C_l = 1/2$; the zero crossing is at $C_l = 1.19$. **Parameters:** $I_o = 15$; $K_o = 50$, $\tau_s = 0.025$ ($K_o\tau_s = 1.25$); $k_r = 1.2$; $k_l = 0.75$; $\beta = 0.2$; $\tau_c = 2.5$.

To determine if there exists a unique solution for $C_l$, we must examine the behaviour of the function $g(C_l)$. Negative values of $C_l$ are unphysical, so we consider only $C_l \geq 0$; at $C_l = 0$
we have $g(0) = Z_7\tau_{ad} - Z_8$. This value is generally greater than zero; more specifically, if we consider $K_o\tau_s = 1$, we may write that

$$g(0) > 0 \text{ iff } I_o > 1 + \frac{1}{2\beta r(c_1 + k_T/2)}.$$  \hfill (4.63)

For $k_T = 1.2$, $k_l = 0.75$, $\beta = 0.2$, and $\tau_c = 2.5$ this gives $I_o > 1.74$, a moderate value. (Recall that we must have $I_o > 1$ to have any activity at all in the population.) The above criterion is conservative; if $K_o\tau_s > 1$, the value of $I_o$ required to make $g(0)$ positive will be smaller. Thus, for most reasonable parameter regimes we will have $g(0) > 0$.

The first derivative of $g(C_l)$ with respect to $C_l$ is

$$\frac{\partial g(C_l)}{\partial C_l} = g'(C_l) = -Z_9 + \left(\frac{C_l}{Z_8 + Z_9C_l}\right)^{\tau_c/\tau_{ad}} \left[1 + \frac{\tau_c(C_l - Z_7\tau_{ad})Z_8}{\tau_{ad}C_l(Z_8 + Z_9C_l)}\right].$$  \hfill (4.64)

Figure 4.13 shows a plot of this function for typical parameter values. The derivative is negative at $C_l = 0$: $g'(0) = -Z_9$ and $Z_9 > 0$. As $C_l$ grows, $g'(C_l)$ approaches an asymptotic value given by

$$\lim_{C_l \to \infty} g'(C_l) = -Z_9 + Z_9^\tau_c/\tau_{ad}.$$  \hfill (4.65)

This asymptotic value is negative if $Z_9^{1+\tau_c/\tau_{ad}} > 1$, or simply if $Z_9 > 1$ since $\tau_c/\tau_{ad} > 0$. $Z_9 > 1$ for $K_o\tau_s < 1 + [1 + 2k_l/k_T]^{-1}$: for $k_T = 1.2$ and $k_l = 0.75$, this becomes $K_o\tau_s < 1.44$.

To summarize: near $K_o\tau_s = 1$, the function $g(C_l)$ has $g(0) > 0$, and $g'(C_l) < 0$ for all $C_l$. This implies that the function will cross zero in only one place, and thus there exists a unique solution for $C_l$. We obtain this solution by finding the zero crossing of equation (4.62). This is easily done numerically, and routines to do so are available in many software packages (for example, both MATLAB and Maple have zero-finding routines). If a closed-form solution is desired, the function in (4.62) may be expanded as a power series. A singularity in the second derivative means that the function is not analytic at $C_l = 0$, so we must expand about another point, $C_l = a$. Expanding to first order gives

$$g(C_l) \simeq m(a)C_l + b(a),$$  \hfill (4.66)

where the slope $m(a)$ and intercept $b(a)$ are functions of our choice of point of expansion, $a$. Defining the abbreviations $\gamma(a) = Z_8 + aZ_9$ and $\alpha(a) = [a/\gamma(a)]^{\tau_c/\tau_{ad}}$, we may write the slope and intercept as

$$m(a) = -Z_9 + \alpha(a) \left[1 + \frac{\tau_c}{\tau_{ad}} \left(1 + \frac{Z_7\tau_{ad}Z_9}{\gamma(a)} - \frac{Z_7\tau_{ad}}{a} - \frac{aZ_9}{\gamma(a)}\right)\right]$$  \hfill (4.67)

and
Figure 4.13: Plot of $g'(C_i)$ against $C_i$. The asymptotic value is $Z_9^{-\tau_c/\tau_{ad}} - Z_9 = -0.37$. Parameters: $I_o = 15; K_o = 50. \tau_s = 0.025 (K_o \tau_s = 1.25); k_r = 1.2; k_I = 0.75; \beta = 0.2; \tau_c = 2.5.$
CHAPTER 4. OSCILLATIONS IN POOLS OF COUPLED NEURONS

With the slope and intercept in hand, the approximate solution of equation (4.61) is given by

\[ C_i(a) = -\frac{b(a)}{m(a)} \] (4.69)

This solution is a function of \( a \), and a poor choice of expansion point could lead to inaccurate results. However, choosing \( a = 1/2 \), for example, provides a reasonable approximation over most nonpathological parameter settings.

Once a value of \( C_i \) has been obtained, \( C_h \) is then found from \( C_h = Z_8 + Z_9 C_i \), and finally the half-period is \( T = \tau_c \ln(C_h/C_i) \). Figure 4.14 compares the theoretical prediction to results from a series of numerical simulations, plotting the half-period \( T \) against \( K_0 \tau_s \), with all other parameters fixed. A good match between theory and numerical results is obtained, across a broad range of values of \( K_0 \tau_s \). The theory predicts that oscillator death (see the next section) will set in earlier than is actually the case in the numerical simulations, and therefore the theory diverges from the simulation results as the theoretical oscillator death point is approached.

4.7 Oscillator death

If the coupling strength becomes too large, oscillations will cease because the fully-adapted firing rate of one pool will still be enough to completely suppress the other, so one pool will permanently dominate; this is known as “oscillator death” [37, 38]. The theory in the above sections provides a prediction for when this should occur for this system.

As discussed in section 4.6, the transition from one pool firing to the other firing occurs when \( F_i^{\text{max}} \) drops below \( F_i^* \); that is, when the maximum firing rate of the active pool drops below the rate required to fully suppress the other pool. (Recall that this statement neglects the (small) finite time during which both pools have nonzero activity.) Using the terminology introduced above, the condition for oscillator death is

\[ F_i^* < F_i^{\text{max}}. \] (4.70)

If pool \( i \) fully dominates, then \( C_i \rightarrow C_i^{**} = \beta \tau_a \left( I_o - \frac{1}{2} \right) \), from equation (4.26) on page 71. Thus,

\[ F_i^{\text{max}} \rightarrow F_i^{**} = F_i(C_i^{**}) \approx \left( I_o - \frac{1}{2} \right) \left[ 1 - \beta \tau_a \left( k_l + \frac{k_r}{2} \right) \right], \] (4.71)
Figure 4.14: Comparison of theory with simulation results: $T$ vs. $K_0\tau_s$. Two synaptic decay rates were used, $\tau_s = 0.025$ and $\tau_s = 0.05$. The other parameters were fixed at: $I_o = 10; k_T = 1.2; k_I = 0.75; \beta = 0.2; \tau_c = 2.5$. The simulation results are for two pools of 100 neurons each. Note that the theory predicts that no oscillations will occur for $K_0\tau_s > 1.587$; see equation (4.73) in section 4.7. The vertical dotted line marks $K_0\tau_s = 1.587$; note that while the theory has $T \to \infty$ as it approaches this point, the simulations still have oscillatory solutions for $K_0\tau_s = 1.6$. By the time we reach $K_0\tau_s = 1.7$, however, no oscillatory period can be defined, since the two pools are making essentially random transitions, driven by fluctuations rather than deterministic dynamics; see Figure 4.15 in section 4.7. The numerical simulation results shown here were generated by keeping $\tau_s$ fixed at one of two values and varying $K_0$; other runs (not shown), in which $\tau_s$ was varied, indicate that the product $K_0\tau_s$ is the significant factor, as predicted. For large values of $\tau_s$ the theory begins to break down, since the assumption of approximately constant population activity over the synaptic coupling time scale, used in equations (4.31-4.32), is violated.
from equation (4.27). If pool $j$ is fully suppressed, $C_j \to 0$, and from equation (4.59),

$$F_i^* = (I_o - 1)/K_o \tau_s.$$  \hspace{1cm} (4.72)

Substituting (4.71) and (4.72) into (4.70) and rearranging, the condition for oscillator death becomes

$$K_o \tau_s > \frac{I_o - 1}{K_{iys}} \quad \text{or} \quad \frac{I_o - 1}{(I_o - \frac{1}{2}) [1 - \beta \tau_{ad} (k_l + \frac{k_r}{2})]}.$$  \hspace{1cm} (4.73)

For $I_o = 10$, $k_r = 1.2$, $k_l = 0.75$, $\beta = 0.2$, $\tau_c = 2.5$, this becomes $K_o \tau_s > 1.587$. As Figure 4.14 shows, this underestimates the true value somewhat, since oscillations still occur for $K_o \tau_s = 1.6$. The reason is the finite population size, which causes fluctuations in the firing rates. When the fully adapted firing rate of one pool is only marginally high enough to suppress the other pool, small fluctuations in firing rate will be able to cause transitions, allowing the other pool to become dominant. As $K_o \tau_s$ grows, these transitions are expected to become less and less regularly spaced in time, since their timing is dominated by random fluctuations rather than by the deterministic population dynamics. Figure 4.15 shows a numerical simulation with $K_o \tau_s = 1.7$, and we do in fact see irregular transition intervals. By $K_o \tau_s = 1.8$, oscillations have halted completely, and one pool consistently comes to dominate despite the fluctuations; the precise value at which this occurs should be a function of the population size, since fluctuations vanish as the number of individual elements expands to infinity.

### 4.8 Future directions

Although synaptic coupling within each pool has been neglected in this chapter ($K_{ii} = 0$), adding internal coupling does not fundamentally change the nature of the calculations. Consider the case with all-to-all coupling between pools ($K_{12} = K_{21} = K$, as before), and all-to-all coupling within each pool ($K_{11} = K_{22} = W$). The effective current equations (4.31–4.32) then become

$$\dot{I}_1 = I_o - k_l C_1 - F_1 W_o \tau_s - F_2 K_o \tau_s,$$

$$\dot{I}_2 = I_o - k_l C_2 - F_2 W_o \tau_s - F_1 K_o \tau_s,$$  \hspace{1cm} (4.74)

$$\dot{V}_1 = \dot{V}_2 = \frac{1}{K_o} (I_o - 1),$$

where $K_o = NK$ and $W_o = NW$. The calculations in sections 4.4.1 through 4.7 should be repeatable for this expanded system, at the cost of more cumbersome algebra. In fact, any number of pools of neurons could be analyzed, representing each population’s activity using effective synaptic input currents and population-averaged calcium levels.

Throughout this chapter I have assumed that asynchronous firing prevails in the populations,
Figure 4.15: Fluctuation-induced transitions in a simulation with parameters $I_0 = 10$, $k_T = 1.2$, $k_I = 0.75$, $\beta = 0.2$, and $\tau_c = 2.5$. For these values, oscillator death is predicted for $K_o \tau_s > 1.587$; here, $K_o \tau_s = 1.7$. The plot of $C_t$ against $t$ shows that transitions are in fact still possible, but that they are irregular and induced by fluctuations in the population firing rates (due to the finite population size) rather than by the deterministic population dynamics.
with any potential synchrony being dispersed by noise. In [105], Gerstner carries out an elegant
derivation of the stability of the asynchronous and synchronous states in a single population,
in terms of the noise level and the axonal delay time (I have not considered axonal delays
in this work). It would be interesting to extend Gerstner's work to the coupled pools, and
consider the effect of spike-frequency adaptation on the stability boundaries between synchrony
and asynchrony.

The calculations presented here have been based on the simple integrate-and-fire neuron
model, but it is possible to carry out essentially the same derivations for more complex models.
The only requirement is that an expression must be available which relates an individual neuron's
firing rate to its current input and its internal calcium concentration, as equation (4.15) does
for the integrate-and-fire case. With such an expression in hand, much of the subsequent would
be essentially unchanged. One difficulty that might arise would be if the model to be analyzed
had no reasonable linear approximation in the regime of interest, equivalent to equation (4.22);
this could make the system intractable, though computational studies could still be carried out
in such a case.
Chapter 5

Noise-shaping in populations of coupled neurons

5.1 Acknowledgement

This chapter presents work carried out in the Applied Biodynamics Laboratory, part of the Center for Biodynamics at Boston University, headed by Prof. James Collins. The research is an extension of previous work on neural noise-shaping, presented in Mar et al. [36]. I wish to acknowledge the collaborative input of Dr. Douglas Mar, Prof. Carson Chow, and Prof. James Collins; however, everything presented here is primarily my own work.

5.2 Background: Analog to digital conversion

In this chapter, we move from locomotion to sensory processing, and consider some signal-processing characteristics of networks of spiking neurons. The ability to distinguish signals from noise will be as vital to biologically inspired robots as it is to animals, and the work in this chapter suggests some ways in which spike-frequency adaptation may help improve signal processing in networks of neuron-like spiking units.

A common task in electronic signal processing is converting signals from analog form (for example, the voltage output of a device such as a microphone) into digital form (for example, a bit string encoding the sound registered by the microphone); this is known as A/D conversion. As Figure 5.1 shows, the process typically introduces two forms of discretization into the original continuous (in both time and amplitude) signal. First, the signal is sampled at discrete times, with the individual samples still being continuous values (producing signal $f_2(x)$ in the figure). Provided that the sampling frequency is at least double the highest frequency component of the original signal, no information is lost in this process; this is the content of the Nyquist sampling
Figure 5.1: Effect of quantization on a signal. Function $f_1(x)$ is the original, analog signal. In function $f_2(x)$, this signal has been sampled at discrete times; as long as this sampling occurs at twice the highest frequency occurring in $f_1(x)$, no information is lost in this process. The function $f_3(x)$ is formed by quantizing $f_2(x)$, in this case by rounding all sampled values to either 0 or 1. Information is lost in this last process. Put another way, noise (called quantization noise) is introduced into the original signal.

Electronics designers want their A/D converters to work as accurately as possible, so they attempt to reduce the level of quantization noise as much as possible; the lower the quantization noise, the more faithfully the doubly-discretized digital signal will reproduce the original analog signal.
input. One method of reducing the quantization noise is oversampling. If the original signal is contained in a band \( 0 \leq f \leq f_o \), oversampling means using a sampling frequency \( f_s \gg 2f_o \). The oversampling ratio (OSR) is defined as

\[
\text{OSR} = \frac{\text{sampling frequency}}{\text{Nyquist frequency}} = \frac{f_s}{2f_o}
\]

For oversampling to be effective, the signal must be sufficiently "busy": it must have some chance of changing quantized levels between successive sampling times. If this is not inherently the case, it may be achieved by "dithering" the signal, adding enough external noise to allow changes in quantization state to occur. Busy signals will have, to a good approximation, quantization noise with a flat power spectrum, the same at all frequencies (white noise) [111]. In this case, the amount of noise in the signal band, \( n_o \), may be shown to fall off with the square root of the oversampling ratio.

\[
n_o \propto (\text{OSR})^{-1/2}.
\]

This is the standard sampling effect: when \( M \) independent, identically distributed random variables are averaged, the standard deviation of the new random variable thus obtained is proportional to \( M^{-1/2} \) [110, 112].

It is, however, possible to do better than simple oversampling. Electronic devices called delta-sigma (\( \Delta \Sigma \)) converters employ a technique known as noise-shaping to improve the effect of oversampling. Figure 5.2 shows a schematic of a \( \Delta \Sigma \) quantizer. A first-order delta-sigma converter reduces the noise in the signal band more rapidly than simple averaging:

\[
n_o \propto (\text{OSR})^{-3/2}.
\]

The reduction in noise power is achieved by altering the shape of the power spectrum. With simple oversampling, the quantization noise is approximately white: it has equal power at all frequencies. \( E(f) = E \), a constant. When the delta-sigma scheme is used, the quantization noise power spectrum goes as \( N(f) = 2E \sin(\pi f/f_s) \) [111]; see Figure 5.3. The power spectrum is "shaped": it has lower power at the low frequencies which comprise the signal band, and higher power in the high frequencies. This noise shaping is what enables the faster reduction of in-band quantization noise with oversampling ratio.
Figure 5.2: Schematic of a first-order delta-sigma converter. The analog input \( x(t) \) is converted into a series quantized outputs \( y_i \), where \( i \) indexes the discrete sampling times. Rather than simply oversampling at the clock frequency \( f_s \), the delta-sigma converter subtracts its own output from the input signal, and operates on an integrated version of \( x(t) \) rather than on the signal itself. The effect of these two factors is to reduce the amount of quantization noise present in the signal band.

5.3 Neural noise-shaping

Neurons may use a form of noise-shaping in their signal-processing functions. Mar et al. [36] and Adams [113] have demonstrated that a network of integrate-and-fire (IF) neurons, when coupled in all-to-all mutual inhibition, displays first-order noise shaping.

Consider the schematic of an IF neuron shown in Figure 5.4, and compare this with the delta-sigma modulator shown in Figure 5.2. Comparing the two figures points out the similarities between a self-inhibiting IF neuron and a \( \Delta\Sigma \) modulator: both accept an analog input, integrate it, and apply negative feedback using a discretized version of the integrated signal. It is important to note, however, that although the IF neuron does perform a sort of discretization of the input through its spiking behaviour, it remains an entirely analog device; in particular, time is not discretized in Figure 5.4.

In [36], a network of \( N \) integrate-and-fire neurons coupled in all-to-all inhibition is used to produce noise-shaping. The basic operation of the network is once again illustrated by Figure 5.4, replacing the individual integrator with a set of \( N \) integrators receiving similar inputs, and replacing the single-neuron synaptic current with a collective current generated by all of the neurons in the network. The number \( N \) is then analogous to the oversampling ratio in the discrete-time case. A coupled network shows an improved signal-to-noise ratio (SNR), as well as
Figure 5.3: Effect of a ΔΣ modulator on the noise power spectrum. The signal of interest has a maximum frequency component of $f_o$, indicated by the vertical dotted line. *(Dashed line)* The result of simple oversampling: the noise is flat with frequency, $E(f) = E$. The in-band noise power, $n_o$, is the area under the dashed line in the range $0 \leq f \leq f_o$, and this decreases with oversampling ratio as $n_o \propto (\text{OSR})^{-1/2}$. *(Solid line)* The result of applying a ΔΣ modulator: the noise is “shaped,” with less power in the low frequencies and more in the high frequencies, $N(f) = 2E \sin(\pi f/f_s)$; total noise power is the same as in the simple oversampling case. The in-band noise power is the area under the solid line in the range $0 \leq f \leq f_o$, which decreases with oversampling ratio as $n_o \propto (\text{OSR})^{-3/2}$. *(Compare the area of the triangle formed by the solid line to the area of the rectangle formed by the dashed line).*
Figure 5.4: Schematic of an integrate-and-fire neuron. Each output spike generates a decaying exponential pulse of synaptic output current. \( I_{syn} = \sum_m \gamma(t - t^{(m)}) \), where \( \{t^{(m)}\} \) is the set of firing times and \( \gamma(s) = e^{-s^2/\tau^2} \mathcal{H}(s) \) is the synaptic kernel. The figure shows the low-leak limit, where the neuron acts as a perfect integrator of the input current: \( \dot{v}(t) = I(t) - K I_{syn}(t) \), for synaptic coupling strength \( K \).

an extended dynamic range (DR). (At a given frequency of interest, SNR measures the ratio of the signal power to the noise level, while DR is the ratio between the maximum attainable signal power in the system and the noise level: for a fixed maximum signal power, decreasing the noise level increases the dynamic range.)

This work has demonstrated several points related to noise-shaping in networks of coupled neurons:

- The noise-shaping effect persists in the integrate-and-fire model with a source of noise different from the one used in the previous work. In [36], a random reset of the voltage was used to introduce noise into the firing times of the individual neurons; we shall see that a network using random Poisson processes as the noise source also shows the noise-shaping effect.

- The dynamic range and signal-to-noise ratio are improved by the addition of spike-frequency adaptation to the integrate-and-fire model used in [36].

- A more complex conductance-based model (with Hodgkin-Huxley type dynamics) also demonstrates the noise-shaping effect.

- In the conductance-based model, the improvement in dynamic range caused by adding spike-frequency adaptation to the integrate-and-fire model disappears. There is, however, a signal-processing benefit conferred by adaptation in the conductance-based case, namely that it evens out the firing rates in a heterogeneous population, helping to prevent the fastest-spiking neurons from completely suppressing the slowest.
I will discuss these points in sections 5.4 and 5.5.

### 5.3.1 Calculation of power spectra

To assess noise-shaping behaviour of a network of neurons, one must convert a list of firing times (generated by an integrate-and-fire model or by a conductance-based model, in sections 5.4 and 5.5, respectively) into a power spectrum. This is done by replacing each delta-function spike with a narrow rectangular pulse of unit height, centred at the firing time. When this operation is carried out over a full run, it converts a list of firing times into a continuous function of time; the autocorrelation of this function may then be found with standard techniques [114], and taking the Fourier transform of the autocorrelation function yields the power spectral density, which I shall refer to simply as the power spectrum. Code in C to carry out this operation was provided courtesy of Douglas Mar, Boston University.

### 5.4 Adapting integrate-and-fire neurons

#### 5.4.1 Neuron model

The adapting IF model uses the same equations presented in section 4.3.2:

\[ \dot{v}_i = -[1 + k_T \alpha_i] v_i + \alpha_i I(t) - k_I c_i - K \sum_{j=1}^{N} I_j^{syn}(t) - \delta(v_i - 1). \]  
\[ \dot{c}_i = \beta \delta(v_i - 1) - c_i / \tau_c. \]  

for \( i = 1, \ldots, N \). (The parameters \( k_T, k_I, \beta, \) and \( \tau_c \) are discussed in section 4.3.2.) All neurons receive the same input current \( I(t) = I_0 + S(t) \), combining a constant DC input and a time-varying signal. \( S(t) = A \sin 2\pi f_o t \) with amplitude \( A \) and frequency \( f_o \). This input is weighted by a heterogeneity factor \( \alpha_i \), representing a variation in intrinsic firing rates across the population; the \( \alpha_i \) are chosen from a uniform distribution over some range, typically \( \alpha_i \in [1, 1.25] \). The coupling is all-to-all (each neuron couples to every other neuron in the network, including itself), and uses the same form as described in section 4.3.3: \( I_j^{syn}(t) = \sum_{m} \gamma(t - t_j^m) \), where \( \{t_j^m\} \) is the set of firing times for neuron \( j \) and \( \gamma(s) = e^{-s/\tau_s} \mathcal{H}(s) \) is the synaptic kernel, \( \tau_s \) being the synaptic decay time constant. The coupling strength is given by the constant \( K \), with \( K > 0 \) representing inhibitory coupling.

Since equations (5.5-5.6) are nondimensional, all frequencies cited in this section will also be dimensionless (note, however, that the conductance-based model of section 5.5 is dimensional). A frequency of 1 therefore implies one cycle per membrane time constant. The conversion to a dimensional frequency depends on the value of the membrane time constant assumed in the
process of nondimensionalization. Here, I have in mind a neuron with a low level of leak and a long membrane time constant, perhaps \( \tau_m > 50 \text{ ms} \); the exact value is not critical to the results.

The average network frequency over each numerical run will be denoted \( F \). In all cases shown here, the values of \( I_o \) have been adjusted to make \( F \approx 1000 \).

All numerical simulations are carried out using a fourth-order Runge-Kutta method with maximum step size \( h = 10^{-4} \) (dimensionless). When two neurons happen to generate spikes within the same interval, the step size is halved until only one of them fires; the exact time at which the firing threshold is reached is then found by interpolation between \( v(t) \) and \( v(t + h) \).

### 5.4.2 Effect of Poisson noise

In [36], noise is introduced into the firing times of the neurons via a random reset of the voltage after each spike [109, 115, 116]; rather than being reset to 0 once the threshold \( v = 1 \) is reached, the reset voltage \( v_o \) is chosen with a uniform probability distribution over some range. Noise is necessary for several reasons. A sufficient level of noise prevents synchronization, which often occurs in coupled networks, especially with inhibitory coupling [27, 91, 117, 118]. A synchronized network can only represent one signal, namely the synchronized network frequency; it is thus better for the network to remain in the asynchronous state, in which signals may be encoded in the network activity at any arbitrary frequency. Another related reason for adding noise is to suppress the harmonics at multiples of input signal frequency. In a network of purely deterministic neurons, even in the absence of synchronization, the signal will have strong peaks at multiples of the input frequency; we want a clear peak at the signal frequency, and no other harmonics, and adding noise accomplishes this. In addition to these factors, of course, is the fact that real biological neurons tend to be quite variable in their firing times [31, 39, 119, 120, 121, 122], so a model incorporating noise is more biologically reasonable than a purely deterministic one.

Rather than using a random reset, another common method of introducing noise into a deterministic neuron model is to simulate the effect of incoming synaptic inputs from neurons not forming part of the modelled network [31, 115, 123, 124]. Since these external neurons are not explicitly represented in the network, their spiking times may be treated as effectively random and modelled as a Poisson process with some rate \( \lambda \). Considering two separate Poisson processes, one for excitatory inputs and one for inhibitory inputs, the noisy version of (5.5) becomes, for an individual, uncoupled neuron,

\[
\dot{v} = -[1 + k_r c]v + I(t) - k_f c - \delta(v - 1) + \Delta v^+ \eta^+(t) - \Delta v^- \eta^-(t),
\]

where \( \eta^+(t) \) and \( \eta^-(t) \) are Poisson processes with rates \( \lambda^+ \) and \( \lambda^- \), respectively. Since the output of a Poisson process is a series of \( \delta \)-functions, the effect of the additional terms in (5.7) is to
“kick” the voltage up and down instantaneously by $\Delta v^+$ or $\Delta v^-$ as each point event occurs. This discontinuous change in membrane voltage is not, of course, physically realistic, but it reasonably reproduces the effect of incoming synaptic inputs where the synaptic time constants are small; simulating the synaptic dynamics of each incoming spike is more computationally expensive, and gives essentially the same results in numerical simulations (results not shown). Setting $\lambda^+ = \lambda^- = \lambda$ and $\Delta v^+ = \Delta v^- = \Delta v$ makes the noise terms average out to zero, so that the average firing rate is the same as in the noiseless equations.

Figure 5.5 shows a comparison of the noise-shaping seen with Poisson noise to that seen with random reset noise: the results are qualitatively similar, though the slope of the curve in the Poisson noise case is slightly more shallow than in the random reset case. The improvement in dynamic range and signal-to-noise ratio seen in the random reset case (described in the next section) is also present for the Poisson noise case, though the improvement is slightly smaller (results not shown).

The near agreement in the power spectra for the two cases indicates that the noise-shaping effect is not a unique feature of the random reset method of adding noise to the firing rates.

5.4.3 Effect of adaptation on DR and SNR

Incorporating spike-frequency adaptation into the integrate-and-fire model has the effect of increasing the network’s dynamic range and signal-to-noise ratio in the lower frequency ranges. Figure 5.6 compares power spectra obtained for networks with and without spike-frequency adaptation. At the signal frequency of 100 Hz, there is a gain of 2.6 dB in dynamic range (the noise level decreases by this amount, which for a constant maximum signal power corresponds to an increase in dynamic range). There is also a gain in signal-to-noise ratio of 3.3 dB compared to the no-adaptation case, again at a signal frequency of 100 Hz.

No theoretical derivation of the effect of adaptation on the power spectrum has been completed to date. Calculations using a first-order approximation for the influence of the $c_i$ terms on the firing rates of the individual neurons indicate that the effect is due to some higher-order influence, since the increased dynamic range does not come out of the first-order version (these calculations will not be reproduced here). Work is currently in progress on a higher-order approach to the theory; this is being carried out in collaboration with Dr. Douglas Mar of Boston University and Prof. Carson Chow of the University of Pittsburgh.
Figure 5.5: Power spectra with Poisson noise vs. random reset ($\beta = 0$, no adaptation). No input signal has been provided to the network ($A = 0$). (Dotted line) Uncoupled network, $K = 0$. Poisson noise with $\lambda = 250$, $\Delta v = 0.025$. $I_0 = 18.36$, $\alpha_i \in [1, 1.25]$, $F = 1000$. (Dashed line) Coupled network, $K = 50$. Random reset noise, $v_o$ reset into the range $[-.3, .3]$. $I_0 = 60.8$, $\alpha_i \in [1, 1.25]$, $F = 999.6$. (Solid line) Coupled network, $K = 50$. Poisson noise with $\lambda = 250$, $\Delta V = 0.025$. $I_0 = 61.5$, $\alpha_i \in [1, 1.25]$, $F = 1001$. The power spectrum in the Poisson noise case is similar to that in the random reset case (except for a slightly shallower slope with frequency), indicating that the results reported in [36] are not specific to their choice of noise model.
Figure 5.6: Power spectra with and without spike-frequency adaptation, integrate-and-fire model. An input signal with $A = 3$ and $f_o = 100$ has been provided to the system in each of the following cases. In all cases, the heterogeneity factor $\alpha_i \in [1, 1.25]$, and noise is introduced via a random voltage reset noise, $v_o$ reset in the range $[-.3,.3]$. (Dotted line) Uncoupled network: $K = 0$; $I_o = 18.455$; $F = 1001$. (Dashed line) Coupled network, no adaptation: $\beta = 0$; $K = 50$; $I_o = 61.5$; $F = 1002$. (Solid line) Coupled network with adaptation: $k_r = 1.5$; $k_l = 0.9375$; $\beta = 0.2$; $\tau_c = 2.5$; $K = 50$; $I_o = 77.2$; $F = 999.9$. 

Chapter 5. Noise-Shaping in Populations of Coupled Neurons
Table 5.1: Currents used in the conductance-based model. \( V, m, h, n, [Ca^{2+}], \) and \( m^{Ca} \) are dynamical variables, while the quantities \( V_X \) and \( g_X \) (e.g. \( V_K \) and \( g_K \)) are parameters, giving the reversal potential and conductance, respectively, associated with ion \( X \). The values of the parameters used are shown in Table 5.2.

5.5 Conductance-based neurons

5.5.1 Neuron model

In this section we will consider noise-shaping using more complex model neurons than the integrate-and-fire neurons used in section 5.4. Here, the individual neurons will be described by a conductance-based neuron (see section 1.5.2) described in [21]. In the original paper, a two-compartment approach is taken, with one set of equations for the soma and another for the dendrites: here, only a single compartment has been used.

The equations are of the standard conductance-based type, with all-to-all synaptic coupling between members of the network. The rate of change of the membrane voltage is

\[
C \frac{dV_i}{dt} = I_L + I_{Na} + I_K + I_{Ca} + I_{AHP} + \alpha_i I_o - K \sum_{j=1}^{N} I_{syn}^j(t).
\] (5.8)

The definitions of the various ionic currents are summarized in Table 5.1. As in the integrate-and-fire case, heterogeneity in the population is introduced by multiplying the current \( I_o \) by a factor \( \alpha_i \) for each neuron. The synaptic coupling current is, as before, \( I_{syn}^j(t) = \sum_m \gamma(t - t_{jm}) \), where \( \{t_{jm}\} \) is the set of firing times for neuron \( j \) and \( \gamma(s) = e^{-s/\tau_s} H(s) \) is the synaptic kernel, \( \tau_s \) being the synaptic decay time constant. Unlike the integrate-and-fire model, the conductance-based neuron has no explicit threshold at which the voltage is discontinuously reset. Firing times are therefore assigned as the times at which the voltage crosses some fixed value such as \(-45 \text{ mV}\); when this crossing occurs, a spike is generated and the neuron's synaptic current output is increased. The coupling strength is given by the constant \( K \), with \( K > 0 \) representing inhibitory coupling.

Three variables summarize the action potential generating dynamics of the neuron: \( m \), the sodium channel activation; \( h \), the sodium channel inactivation; and \( n \), the potassium channel
activation. (See section 1.5.2). All of these have dynamics of the form \( \dot{x} = \phi_x [x_\infty(V) - x]/\tau_x(V) \), with

\[
x_\infty = \alpha_x(V)/[\alpha_x(V) + \beta_x(V)]
\]

and

\[
\tau_x = 1/[(\alpha_x(V) + \beta_x(V)].
\]

The sodium activation is assumed to be fast, so that

\[
m = m_\infty(V) = \frac{\alpha_m(V)}{\alpha_m(V) + \beta_m(V)}.
\]

with

\[
\begin{align*}
\alpha_m(V) &= \frac{-1(V + 33)}{\exp[-1(V + 33)] - 1} , \\
\beta_m(V) &= 4 \exp[-(V + 58)/12].
\end{align*}
\]

Note that \( V \) is expressed in mV throughout these equations. Equations of the form (5.12-5.13) are the result of fitting curves to experimental data on the ion channel kinetics in particular neurons: the neurons described here and in \[21\] are cortical pyramidal neurons, but all conductance-based models have a similar form.

The sodium inactivation variable obeys

\[
\dot{h} = \phi_h [h_\infty(V) - h]/\tau_h(V),
\]

where \( \phi_h \) is a rate scaling parameter, and the channel kinetics are given by

\[
\begin{align*}
\alpha_h(V) &= 0.07 \exp[-(V + 50)/10], \\
\beta_h(V) &= \frac{1}{\exp[-1(V + 20)] + 1}.
\end{align*}
\]

The potassium activation obeys

\[
\dot{n} = \phi_n [n_\infty(V) - n]/\tau_n(V),
\]

with channel kinetics
\begin{align*}
\alpha_n(V) &= \frac{-0.01(V + 34)}{\exp[-0.01(V + 34)] - 1}, \\
\beta_n(V) &= 0.125 \exp[-(V + 44)/25].
\end{align*}

Equations (5.8), (5.11), (5.14), and (5.17) define the usual four-dimensional system of equations used in conductance-based models; here, a common simplification has been made, reducing the system to three ordinary differential equations by replacing the ODE for \( m \) with the algebraic relationship (5.11). Wang [21] adds spike-frequency adaptation to the basic model by considering a calcium-dependent potassium current: as the concentration of calcium, \([Ca^{2+}]\), rises, a potassium current called \( I_{AHP} \) is activated, causing the cell to fire less rapidly. (The subscript "AHP" stands for "after hyperpolarization," and refers to the fact that after a cell has fired a burst of spikes, its resting potential is hyperpolarized compared to the resting potential in the absence of calcium.) The calcium dynamics (again, from [21]) are given by

\[
\frac{d[Ca^{2+}]}{dt} = \rho I_{Ca} - \frac{[Ca^{2+}]}{\tau_{Ca}},
\]

where \( \tau_{Ca} \) is the decay time constant, and \( \rho \) sets the rate of influx of calcium ions. The kinetics of the calcium channels are assumed to be fast, so that

\[
m^{(Ca)} = m^{(Ca)}_\infty = \frac{1}{\exp[-(V + 20)/9] + 1}.
\]

At the resting potential (\( V_r = -64.6 \)), \( m^{(Ca)}_\infty \) is near zero. During a spike, as \( V \) rapidly increases to the vicinity of 50 mV, and \( m^{(Ca)}_\infty \rightarrow 1 \) for a brief period, causing \( I_{Ca} \) to become nonzero. By equation (5.20), this causes an increase in calcium concentration, which then begins to decay away once the spike ends and \( m^{(Ca)}_\infty \rightarrow 0 \) again.

The neural model is thus made up of four ODEs: (5.8); (5.14); (5.17); and (5.20). The values of the parameters appearing in the equations are listed in Table 5.2.

With a constant applied current, the neuron spikes regularly, and the calcium concentration builds up until a steady state is reached, in which \([Ca^{2+}]\) is increased by each spike, then decays back to its original level before the next spike occurs; see Figure 5.7. Figure 5.8 shows the relationship between applied current and firing rate for the model, with and without adaptation.

Noise is added to the system using the same method described in section 5.4.2, namely incorporating two independent Poisson processes (one excitatory, one inhibitory) to simulate the influences of neurons not explicitly modelled. The only equation affected is (5.8), which becomes, for an individual uncoupled neuron
Figure 5.7: Voltage and calcium traces of conductance-based model. $I_0 = 5 \mu A$, $\rho = 0.003 \mu M/(\mu A\cdot ms)$.
Figure 5.8: (Top) Firing rate vs. applied current for the conductance-based model. (Bottom) As above, but zoomed in to show the vicinity of the onset of oscillations.
Table 5.2: Parameters in the conductance-based model: values taken from [21].

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>$C$</td>
<td>1 $\mu$F</td>
<td>Membrane capacitance</td>
</tr>
<tr>
<td>$g_L$</td>
<td>0.1 mS</td>
<td>Leak conductance</td>
</tr>
<tr>
<td>$V_L$</td>
<td>-65 mV</td>
<td>Leak reversal potential</td>
</tr>
<tr>
<td>$g_{Na}$</td>
<td>45 mS</td>
<td>Sodium conductance</td>
</tr>
<tr>
<td>$V_{Na}$</td>
<td>55 mV</td>
<td>Sodium reversal potential</td>
</tr>
<tr>
<td>$g_K$</td>
<td>18 mS</td>
<td>Potassium conductance</td>
</tr>
<tr>
<td>$V_K$</td>
<td>-80 mV</td>
<td>Potassium reversal potential</td>
</tr>
<tr>
<td>$g_{Ca}$</td>
<td>1 mS</td>
<td>Calcium conductance</td>
</tr>
<tr>
<td>$V_{Ca}$</td>
<td>120 mV</td>
<td>Calcium reversal potential</td>
</tr>
<tr>
<td>$\rho$</td>
<td>0.003 $\mu$M/(µA·ms)</td>
<td>Calcium influx rate</td>
</tr>
<tr>
<td>$\tau_{Ca}$</td>
<td>80 ms</td>
<td>Calcium decay time constant</td>
</tr>
<tr>
<td>$K_D$</td>
<td>30 $\mu$M</td>
<td>Calcium equilibrium constant</td>
</tr>
<tr>
<td>$\phi_h$</td>
<td>4</td>
<td>$h$ rate scaling</td>
</tr>
<tr>
<td>$\phi_n$</td>
<td>4</td>
<td>$n$ rate scaling</td>
</tr>
<tr>
<td>$g_{AHP}$</td>
<td>5 mS</td>
<td>After hyperpolarization conductance</td>
</tr>
</tbody>
</table>

\[
C \frac{dV}{dt} = I_L + I_{Na} + I_K + I_{Ca} + I_{AHP} + I_0 + \Delta V^+ \eta^+(t) - \Delta V^- \eta^-(t),
\]

(5.21)

where $\eta^+(t)$ and $\eta^-(t)$ are Poisson processes with rates $\lambda^+$ and $\lambda^-$. respectively. The effect of the additional terms in (5.21) is, as before, to increment or decrement voltage instantaneously by $\Delta V^+$ or $\Delta V^-$. This discontinuous change reasonably reproduces the effect of incoming synaptic inputs where the synaptic time constants are small. Simulations were run in which the Poisson processes drove a synaptic coupling current rather than influencing the voltage directly, and the effect was the same. Since the above form, which is computationally more convenient, has been used, taking. I have taken $\Delta V^+ = \Delta V^- = \Delta V$ and $\lambda^+ = \lambda^- = \lambda$: balancing the excitatory and inhibitory noise in this way means that the average firing rate is still given by the curve shown in Figure 5.8. With the addition of this Poisson noise, firing in the neurons is no longer completely regular; see Figure 5.9.

5.5.2 Noise-shaping results

Figure 5.10 illustrates that the noise-shaping effect is still seen in a network conductance-based neurons coupled in all-to-all inhibition (the spectra shown are for the no-adaptation case, in which $\rho = 0$ and thus $[\text{Ca}^{2+}](t) = 0$). It is interesting to note, however, a significant change in the frequency range to which the low-frequency noise power is shifted. In the integrate-and-fire model, the power from the low-frequency noise was shifted to the range near the network frequency (see Figures 5.5 and 5.6). Here, however, the noise power is "piled up" at frequency
Figure 5.9: Firing in the conductance-based neuron with Poisson noise. Parameters: $I_o = 5 \, \mu A$; $\lambda = 1 \, \text{kHz}$; $\Delta V = 1 \, \text{mV}$. 
Figure 5.10: Noise-shaping in a network of fifty conductance-based neurons. no spike-frequency adaptation ($\rho = 0$). (Dashed line) Uncoupled network: $K = 0$; $I_o = 0.26 \mu A$; $F = 1.001$ kHz. The vertical dotted lines indicate the range of individual neuron frequencies, from 17.9 Hz to 22.6 Hz. (Solid line) Coupled network: $K = 12$; $\tau_s = 0.5$ ms; $I_o = 5.2015 \mu A$; $F = 0.9993$ kHz. The vertical dashed line indicates the fastest individual neuron frequency; the range is from 0.12 Hz (off the scale) to 56 Hz. Parameters (common to both cases): $\alpha_i \in [1, 1.25]$; $\lambda = 1$ kHz; $\Delta V = 1$ mV; $\rho = 0$.

below the average network frequency, but still higher than the individual neuron frequencies. As yet, no theoretical explanation for this difference exists.

5.5.3 Effect of adaptation

The improvement in dynamic range and signal-to-noise ratio offered by adding spike-frequency adaptation to the integrate-and-fire model disappears when we move to the conductance-based model of this section; the results are not shown, but the spectrum with adaptation is essentially
CHAPTER 5. NOISE-SHAPING IN POPULATIONS OF COUPLED NEURONS

indistinguishable from the nonadapting spectrum shown in Figure 5.10. It is unclear why this should be the case, but

However, there is still an advantage to be gained from the presence of adaptation. In a heterogeneous network, the individual neurons display a range of baseline firing rates: each neuron fires at a slightly different rate in response to the same level of current input, represented in the model by the range of values of the \( \alpha_t \) in equation (5.8). When the network is coupled, Figure 5.10, and the fastest-firing neurons tend to suppress their slower neighbours; note the wider range of individual firing rates in the coupled network shown in Figure 5.10, as compared to the range of individual rates for the uncoupled network. The coupling can create a situation in which the slowest neurons in the network are effectively silenced, either not spiking at all or spiking so rarely that they take no real part in representing the input signal. Spike-frequency adaptation reduces the tendency for the neurons with high intrinsic rates to dominate the slowest ones (since a fast neuron will adapt and lower its firing rate, giving slower neurons a chance to escape from the inhibition and fire). The signal-processing advantage of this lies in the fact that greater signal-to-noise ratios are attained by increasing \( N \), the number of neurons in the network; if many neurons are completely suppressed, the effective network size is reduced, limiting the attainable signal-to-noise ratio.

Figure 5.11 shows two raster plots, in which horizontal lines represent individual neurons, with the spiking times indicated by dots. The upper plot shows a run without adaptation, while the lower one incorporates the adaptation dynamics. Both runs have been adjusted to have the same average network frequency, but in the no-adaptation case approximately 35 of the 100 neurons play effectively no role in the network's activity, while with adaptation all 100 neurons are at least somewhat active.

The effect grows more pronounced as we increase \( N \), or as we increase the coupling strength \( K \) at a given \( N \). This silencing of the slower neurons implies that a heterogeneous network cannot use its full capacity, and in effect the network contains only \( N_{\text{eff}} \leq N \) neurons, where \( N_{\text{eff}} \) is calculated by setting a threshold firing rate below which a neuron will be counted as silent. Figure 5.12 shows the beneficial effect of adaptation on \( N_{\text{eff}} \), using a threshold of 0.1 Hz as the cutoff for considering a neuron to be active.

The reduction of \( N_{\text{eff}} \) with \( N \) places an upper limit on how many neurons can effectively participate in signal-processing in a network. Since the SNR increases with increasing \( N \) in a coupled network [36], a reduced \( N_{\text{eff}} \) places a limit on the attainable signal-to-noise ratio.
No spike-frequency adaptation

With spike-frequency adaptation

Figure 5.11: Raster plots, comparing coupled networks with and without adaptation; the input current has been adjusted to give the same average individual neuron frequency in each case. The heterogeneity factor $\alpha_i \in [1, 1.25]$ in both cases. (Top) Coupled network, no adaptation ($\rho = 0$): $I_0 = 10.25 \mu A$. Range of individual neuron frequencies: 0 to 82.8 Hz. with an average of 19.99 Hz. At a threshold of 0.1 Hz, $N_{eff} = 65$. (Bottom) Coupled network, with adaptation (parameters as in Table 5.2): $I_0 = 11.2 \mu A$. Range of individual neuron frequencies: 0.7 to 45.1 Hz. with an average of 20.01 Hz. At a threshold of 0.1 Hz, $N_{eff} = 100 = N$. Parameters (common to both cases): $N = 100$; $K = 12$; $\tau_s = 0.5$ ms; $\alpha_i \in [1, 1.25]$; $\lambda = 1$ kHz; $\Delta V = 1$ mV.
Figure 5.12: Effective number of neurons in network ($N_{eff}$) vs. $N$. A neuron is counted towards $N_{eff}$ if it fires at a rate of at least 0.1 Hz. In all cases, the value of $I_o$ has been adjusted to yield an average individual neuron frequency of $20 \pm 0.1$ Hz. (Solid line, circles) No adaptation ($\rho = 0$). (Dashed line, squares) With spike-frequency adaptation (parameters as in Table 5.2).
Chapter 6

Effect of adaptation on neural variability

6.1 Local abbreviations

The following table lists the abbreviations used for convenience in this chapter: as discussed in section 1.7. they are "local" in the sense that they apply only within this chapter.

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Definition</th>
<th>Section</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\bar{\tau}$</td>
<td>$[1 + k \tau c]^{-1}$</td>
<td>6.3</td>
</tr>
<tr>
<td>$\bar{I}$</td>
<td>$I_0 - k \tau c$</td>
<td>↓</td>
</tr>
<tr>
<td>$v_\infty$</td>
<td>$\bar{\tau} \bar{I}$</td>
<td></td>
</tr>
<tr>
<td>$Z_1$</td>
<td>$-(\theta - v_\infty)/\bar{\tau}(B - A)$</td>
<td>6.4.1</td>
</tr>
<tr>
<td>$Z_2$</td>
<td>$1/\bar{\tau}$</td>
<td>↓</td>
</tr>
<tr>
<td>$Z_3$</td>
<td>$(v_\infty - v_0)/\bar{\tau}(D - C)$</td>
<td>6.5.1</td>
</tr>
<tr>
<td>$Z_4$</td>
<td>$-1/\bar{\tau}$</td>
<td>↓</td>
</tr>
</tbody>
</table>

6.2 Introduction and background

If biologically inspired robots are to use networks of spiking artificial neurons as their primary internal control mechanism, we will need to understand the properties of such neurons in considerable detail. Here, we will examine the effect of noise on the variability of a simple spiking neuron model, and consider how this variability changes under the influence of spike-frequency adaptation.

Recordings of spike trains from actual neurons do not, typically, spike at regular intervals; rather, there is a high degree of variability in the interspike intervals (ISIs) [31, 39, 119, 120, 121,
122. There are thought to be two main sources of this variability [31, 115, 125, 126]: intrinsic noise, internal to each neuron and arising from factors such as fluctuations in the opening and closing in finite populations of ion channels; and synaptic noise, caused by the influence on each individual neuron of a large and generally unmonitored population of other neurons, whose incoming spikes cause the neuron's state to vary in apparently random ways.

This chapter addresses a simple observation arising from numerical simulations of the integrate-and-fire (IF) neurons used in chapters 4 and 5. There are two particularly simple ways to introduce variability into the firing times of IF neurons: random voltage reset [36, 109, 115, 116], in which the membrane voltage is reset to a random value after each spike; and random threshold reset [33, 127, 128], in which the firing threshold for the next spike is reset to a random value each time the neuron generates a spike. The simulations point out two interesting facts:

- random voltage reset and random threshold reset have opposite effects as the firing rate decreases: with voltage reset, the variability drops rapidly as the firing rate approaches zero; with threshold reset, the variability increases rapidly.

- adding spike-frequency adaptation alters the variability, even at high firing rates: with voltage reset, adapting IF neurons are less variable than the equivalent tonic (nonadapting) neuron; with threshold reset, adapting neurons are more variable than their tonic equivalents.

Figure 6.1 shows the numerical results described above, using a standard quantity called the coefficient of variability (CV) to summarize the degree of variability of the IF neurons under various conditions. The CV is simply the ratio of the standard deviation to the mean, and is often used in experimental and theoretical work [129].

It is quite straightforward to calculate the probability density (and thus the CV) associated with the interspike interval distribution in the tonic IF case. A closed-form solution for the coefficient of variability in the presence of spike-frequency adaptation will be derived, subject to one approximation. Where this approximation breaks down will be discussed, and finally I will summarize the underlying cause of the difference in variability with and without spike-frequency adaptation.

6.2.1 Notation for probability calculations

Standard terms from probability theory have been used: to avoid ambiguity, these will be reviewed briefly, here. The probability of an event will be denoted Pr\{event\}. A random variable $X$ has a probability density function (pdf) $f_X(x)$ such that the probability of $X$ taking on a value in the set $A$ is $\int_A f_X(x)dx$. For a scalar random variable, as all variables will be in this chapter,
Figure 6.1: Effect of adaptation on IF neuron variability, numerical results. The coefficient of variability (CV) is used to summarize the level of variability; CV is defined as the ratio of the standard deviation to the mean, $CV = \sigma/\mu$. Squares show the results for tonic neurons (no spike-frequency adaptation), while circles indicate the results when adaptation is present ($k_r = 1.2, k_I = 0.6, \beta = 0.2, \tau_e = 2.5$; see section 4.3.2 for a description of these parameters). (Top) Random voltage reset: after each spike, $v$ is reset to a random value, uniformly chosen from the range $[-.3, .3]$; the firing threshold is $\theta = 1$. The horizontal axis shows the average firing rate, $\langle f \rangle$. (Bottom) Random threshold reset: after each spike, the firing threshold $\theta$ is reset to a random value, uniformly chosen from the range $[.7, 1.3]$; the voltage is reset to $v_o = 0$. 
Pr\{a \leq X \leq b\} = \int_a^b f_X(x)dx. Another representation of a random variable's distribution is the cumulative distribution function (cdf) \( F_X(x) = \Pr\{X \leq x\} \). The pdf and cdf are related by

\[
f_X(x) = \frac{dF_X(x)}{dx}.
\] (6.1)

The mean of a random variable is given by

\[
E[X] = \mu_X = \int_{-\infty}^{\infty} x f_X(x)dx.
\] (6.2)

and in general the \( n \)-th moment of \( X \) is given by

\[
E[X^n] = \int_{-\infty}^{\infty} x^n f_X(x)dx.
\] (6.3)

The variance of \( X \) is given by

\[
\] (6.4)

Finally, the standard deviation of \( X \) is given by

\[
\sigma_X = \sqrt{\text{Var}[X]}.
\] (6.5)

and the coefficient of variability (CV) is given by \( \text{CV} = \sigma_X / E[X] = \sigma_X / \mu_X \).

### 6.3 Neuron model

Once again, the integrate-and-fire model described in [108], which includes spike-frequency adaptation, has been used. (This model was also used in chapters 4 and 5.) The nondimensionalized form of the equations is

\[
\dot{v} = -\frac{v}{\tau} + \bar{I} - \delta(v - \theta)
\] (6.6)

\[
\dot{c} = \beta\delta(v - \theta) - c/\tau_c,
\] (6.7)

where \( \tau(c) = [1 + k_Dec]^{-1} \), \( \bar{I}(t,c) = I_0 - k_IC \), and \( \theta \) is the spiking threshold. See section 4.3.2 for information on the parameters. The tonic (nonadapting) version of the equations is obtained simply by eliminating equation (6.7) and using \( c = 0 \) in equation (6.6).

For a fixed value of \( c \),

\[
v(t) = v_0 e^{-t/\tau} + \bar{I}(1 - e^{-t/\tau}),
\] (6.8)
where \( v(0) = v_0 \) is the voltage reset value. This solution is exact in the tonic case, but only an approximation when the neuron display spike-frequency adaptation, since \( c \) is not constant in that case. Setting \( v_\infty = \tau \bar{I} \), we see that \( v(t) \to v_\infty \) as \( t \to \infty \); provided that \( v_\infty > \theta \), the neuron will eventually cross the spiking threshold and be reset to \( v_0 \).

6.4 Random voltage reset

6.4.1 Calculation of CV

In random voltage reset, the voltage \( v \) is reset stochastically rather than to a constant value \( v_0 \). To determine the resulting variability in the interspike intervals, we must define a random variable giving the distribution of the ISIs, then find the coefficient of variability (CV) of this variable.

First, note that for a given reset voltage \( v_0 \), we can solve equation (6.8) for the time, \( t^* \), at which \( v = \theta \):

\[
\theta = v_\infty + e^{-t^*/\tau} (v_0 - v_\infty).
\]

which yields (for fixed \( v_\infty \) and \( \theta \))

\[
t^*(v_0) = \tau \ln \left( \frac{v_0 - v_\infty}{\theta - v_\infty} \right).
\]

where \( v_\infty = \tau \bar{I} \), as before.

If \( v_0 \) is replaced with a random variable \( V_0 \) with some known distribution \( f_{V_0}(v_0) \), then the interspike interval also becomes a random variable. Call this random variable \( T \): its distribution \( f_T(t) \) is determined by the distribution of \( V_0 \). The variability of the interspike intervals is thus determined by the variability of the random variable \( T \).

Before proceeding, we need to find the inverse of equation (6.10), which gives the voltage reset value required to produce a given ISI:

\[
V_0^{-1}(t) = \theta + v_\infty (1 - e^{t/\tau}).
\]

The cdf for \( T \) is

\[
F_T(t) = \Pr\{T \leq t\} = \Pr\{0 \leq T \leq t\}.
\]

where the second equation arises simply from the fact that ISIs may not be negative. Since \( V_0^{-1}(t) \) is monotonically decreasing, equation (6.12) may be rewritten as
Figure 6.2: Random voltage reset. Uniform pdf. The shaded area indicates the area corresponding to the integral in equation (6.16); this area gives the probability that the random variable $T$ falls between 0 and $t$, which is the definition of $F_T(t)$.

\[
F_T(t) = \Pr\{V_o^{-1}(t) \leq V_o \leq V_o^{-1}(0)\} = \Pr\{V_o^{-1}(t) \leq \theta\} = \int_{V_o^{-1}(t)}^{\theta} f_{V_o}(v_o)dv_o. \tag{6.13}
\]

Using the known distribution $f_{V_o}(v_o)$, (6.13) can be evaluated, and the pdf $f_T(t)$ may then be found by taking $f_T(t) = dF_T(t)/dt$. The details of solving (6.13) depend, of course, on the form of random reset distribution. Possible cases include a uniform distribution [36] and a Gaussian distribution [105]; here, I will present only the former.

Assume that the voltage is reset with uniform probability into the voltage range $[A, B]$, with $B \leq \theta$; this gives the following probability distribution function for $V_o$ (see Figure 6.2):

\[
f_{V_o}(v_o) = \begin{cases} 
\frac{1}{B-A}, & A \leq v_o \leq B \\
0, & \text{otherwise} 
\end{cases} \tag{6.14}
\]

The minimum possible ISI occurs when $V_o = B$ (highest voltage = shortest time), while the maximum ISI occurs when $V_o = A$ (lowest voltage = longest time). Define these limiting times as $T_1 = t^*(B)$ and $T_2 = t^*(A)$, where $t^*(v_o)$ is given by equation (6.10). For ISIs with $t < T_1$ or $t > T_2$, the pdf $f_{V_o}(t) = 0$. For $T_1 \leq t \leq T_2$, we have $A \leq V_o(t) \leq B$, and equation (6.13) may be applied as follows:

\[
F_T(t) = \int_{V_o^{-1}(t)}^{\theta} f_{V_o}(v_o)dv_o
\]
\[
= \int_{V_o^{-1}(t)}^{B} f_{V_o}(v_o)dv_o \tag{6.16}
\]
The complete expression for the cdf for $T$ is then

$$F_T(t) = \begin{cases} 0, & t < T_1 \\ \frac{1}{B - A}[(B - v_\infty) - (\theta - v_\infty)e^{t/\tau}], & T_1 \leq t \leq T_2 \\ 1, & t > T_2 \end{cases}$$

Taking the derivative with respect to $t$, we find the pdf,

$$f_T(t) = \begin{cases} \frac{B - \theta}{(B - A)\tau}e^{t/\tau}, & T_1 \leq t \leq T_2 \\ 0, & \text{otherwise} \end{cases}$$

To evaluate the coefficient of variability (CV), we will require the mean and standard deviation of the interspike interval distribution given by equation (6.21). Considering distributions of the form $f_T(t) = Xe^{Yt}$ and carrying out the appropriate integrations, we find

$$E[T] = \mu_T = \frac{X}{Y^2} \left[ e^{YT_2}(YT_2 - 1) - e^{YT_1}(YT_1 - 1) \right]$$

and

$$E[T^2] = \frac{X}{Y} \left[ e^{YT_2}[T_2^2 - \frac{2}{Y^2}(YT_2 - 1)] - e^{YT_1}[T_1^2 - \frac{2}{Y^2}(YT_1 - 1)] \right].$$

Setting

$$Z_1 \equiv -(\theta - v_\infty)/\tau(B - A),$$

$$Z_2 \equiv 1/\tau,$$

and using $X = Z_1, Y = Z_2$ in equations (6.22-6.23), the coefficient of variability may be found: $E[T]$ and $E[T^2]$ combine to give $\text{Var}[T] = E[T^2] - (E[T])^2$, which in turn gives $\sigma_T = \sqrt{\text{Var}(T)}$, and $CV = \sigma_T/\mu_T$.

### 6.4.2 Approximations for $c$ dynamics

In the case of no adaptation, $\tau = 1$ and $I = I_o$, and equations (6.22-6.25) may be applied directly. To apply these equations for the case with spike-frequency adaptation, the dynamics of the adaptation variable $c$ on $\tau$ and $I$ must be taken into account. Using the same approximation
as in section 4.4.1, we replace \( \dot{c} = \beta \delta (v - \theta) - c/\tau_c \) with \( \dot{c} \simeq \beta f - c/\tau_c \), where \( f \) is the firing rate. As before, this implies that \( c \) will rise to a steady-state value \( c_{ss} = \beta \tau_{ad} \left( I_o - \frac{1}{2} \right) \), where \( \tau_{ad} = \left[ \beta \left( k_I + \frac{k_e}{2} \right) + \frac{1}{\tau_c} \right]^{-1} \). Then \( \bar{\tau} = \left[ 1 + k_r c_{ss} \right]^{-1} \) and \( \bar{I} = I_o - k_I c_{ss} \), and the formulae derived in the previous section may be applied as before. This approximation assumes that the firing rate is fast relative to the decay rate \( \tau_c \); for slow rates (small values of the input current \( I_o \)), the approximation breaks down.

Figure 6.3 shows a comparison between numerical runs and the theoretical predictions, for the random voltage reset case. Numerical runs without adaptation are not shown, but they have been performed, and show a very close match between the theory and the numerical results.
6.5 Random threshold reset

6.5.1 Calculation of CV

If the firing threshold \( \theta \) is reset, and we hold \( v_\infty \) and \( v_o \) fixed, equation (6.10) becomes

\[
\tau^*(\theta) = \bar{\tau} \ln \left( \frac{v_o - v_\infty}{\theta - v_\infty} \right),
\]

exactly as before except that now the interspike interval is a function of \( \theta \) rather than of \( v_o \). Finding the inverse of this function gives us the voltage threshold required to give a desired ISI, \( \tau \):

\[
\theta^{-1}(t) = v_\infty + (v_o - v_\infty)e^{-t/\bar{\tau}} \tag{6.27}
\]

Let \( \Theta \) be a random variable whose value is the random threshold. The pdf of \( \Theta \), \( f_\Theta(\theta) \), determines the cdf of the random variable \( T \) (which gives the interspike intervals), as follows:

\[
F_T(t) = \Pr\{T \leq t\} = \Pr\{0 \leq T \leq t\} = \Pr\{\theta^{-1}(0) \leq \Theta \leq \theta^{-1}(t)\} = \Pr\{v_o \leq \Theta \leq \theta^{-1}(t)\} = \int_{v_o}^{\theta^{-1}(t)} f_\Theta(\theta)d\theta \tag{6.28}
\]

Once again, there are two possible cases for the distribution of the random threshold: uniform and Gaussian. and once more I will present only the uniform case, in which the threshold is reset uniformly into the range \([C, D]\), which gives the following pdf for \( \Theta \):

\[
f_\Theta(\theta) = \begin{cases} \frac{1}{D-C}, & C \leq \theta \leq D \\ 0, & \text{otherwise} \end{cases} \tag{6.33}
\]

with \( D \geq C, C \geq v_o \), and \( D \leq v_\infty \); see Figure 6.4. Substituting (6.33) into (6.32) yields, for \( C \leq \theta^{-1}(t) \leq D \):

\[
F_T(t) = \int_{v_o}^{\theta^{-1}(t)} f_\Theta(\theta)d\theta \tag{6.34}
\]

\[
= \int_{v_o}^{\theta^{-1}(t)} \frac{1}{D-C} d\theta \tag{6.35}
\]
Figure 6.4: Random threshold reset, uniform pdf. The shaded area indicates the area corresponding to the integral in equation (6.35); this area gives the probability that the random variable $T$ falls between 0 and $t$, which is the definition of $F_T(t)$.

$$F_T(t) = 1 - \frac{1}{D-C} \left[ \theta^{-1}(t) - C \right]$$

(6.36)

Define $T_1 = t^*(C)$ and $T_2 = t^*(D)$ as the minimum and maximum possible ISIs, respectively. Then substituting (6.27) into (6.36) produces the complete expression for $F_T(t)$:

$$F_T(t) = \begin{cases} 
0, & t < T_1 \\
\frac{1}{D-C} \left[ (v_\infty - C) - (v_\infty - v_o) e^{-t/\tau} \right], & T_1 \leq t \leq T_2 \\
1, & t > T_2 
\end{cases}$$

(6.37)

Taking the derivative of (6.37) with respect to $t$ gives the pdf of the interspike intervals.

$$f_T(t) = \begin{cases} 
\frac{(v_\infty - v_o) e^{-t/\tau}}{\tau(D-C)}, & T_1 \leq t \leq T_2 \\
0, & \text{otherwise} 
\end{cases}$$

(6.38)

The pdf here has the same form as it had in the uniform random voltage reset case, namely $f_T(t) = X e^{Yt}$. Setting

$$Z_3 \equiv \frac{(v_\infty - v_o)}{\tau(D-C)},$$

(6.39)

$$Z_4 \equiv -1/\tau.$$ 

(6.40)

and using $X = Z_3$, $Y = Z_4$ in equations (6.22) and (6.23), we can find the coefficient of variability just as before.
6.5.2 Approximation of $c$ dynamics

As in section 6.4.2, I will use the steady-state value $c_{ss}$ to find the values of $\hat{\tau}$ and $\hat{I}$, then use (6.39-6.40) in (6.22-6.23) to find the CV.

Figure 6.5 compares the results from numerical simulations with the theoretical prediction obtained by using the $c_{ss}$ approximation. As the plot indicates, the fit is not very accurate. This is because the approximation assumes that $v_\infty \gg \theta$, while in the adapting case $v_\infty$ in fact remains quite small even at high firing rates, as will be discussed in section 6.6.2 (see Figure 6.7). The deviation from theory is greater for the random threshold reset than it was for the random voltage reset, because setting the threshold to values as high as $\theta = 1.3$ brings the neuron into a significantly less linear region, in which there are occasionally very long interspike intervals, making the approximation to the $c$ dynamics less accurate.

6.6 Discussion

6.6.1 Difference between voltage and threshold resets

The reason that random voltage reset and random threshold reset have opposite effects on the coefficient of variability as the firing rate decreases is illustrated in Figure 6.6. Consider the tonic case (no spike-frequency adaptation), so that $\hat{\tau} = 1$, $\hat{I} = I_0$, and $v_\infty = \hat{\tau} \hat{I} = I_0$. For large values of $I_0$, $v_\infty \gg \theta$, and the neuron's dynamics are essentially linear during the approach to threshold (in equation (6.6), the $I_0$ term dominates the leak term, $-v/\hat{\tau}$).

At smaller values of $v_\infty$, the approach to threshold is no longer linear, as the leak term comes to dominate the dynamics. In the random voltage reset case, this reduces the variability because the influence of the initial condition $v_0$ is overwhelmed by the effect of the leak term during the asymptotic approach to $v_\infty$. For $v_\infty = \theta + \epsilon (\epsilon \ll 1)$, the firing time increases to infinity, and trajectories arrive at the threshold at the same instant regardless of their initial condition; thus, the coefficient of variability approaches zero.

In the random threshold reset case, a small value of $v_\infty$ increases the variability, because some trajectories meet the threshold when they are still in the largely linear phase, while others enter the curved asymptotic approach to $v_\infty$. For $v_\infty = D + \epsilon (\epsilon \ll 1)$, the shortest firing times have some small finite value, while the longest times (those for which the random variable $\Theta$ takes on the value $D$) approach infinity; the coefficient of variability thus approaches infinity.

Thus, the reason that the coefficients of variability shown in Figures 6.1 for tonic neurons diverge (upwards or downwards) as the firing rate decreases is because the reduced firing rate is accomplished by reducing $I_0$, which corresponds directly to reducing $v_\infty$ and thus moving the neuron into the regimes discussed in the preceding paragraphs. Note that reducing the firing rate while simultaneously increasing the time constant $\hat{\tau}$ could keep the trajectories linear even
Figure 6.5: Random threshold reset: theory and numerical results. After each spike, the firing threshold $\theta$ is uniformly chosen in the range $[0.7, 1.3]$; the reset voltage is constant at $v_0 = 0$. The neuron exhibits spike-frequency adaptation ($k_r = 1.2$, $k_l = 0.6$, $\beta = 0.2$, $\tau_c = 2.5$). The horizontal axis gives the average steady-state firing rate, $< f >$. (Circles) Results from numerical simulations. (Dashed line) Theoretical prediction for CV, using (6.39-6.40) in (6.22-6.23).
Figure 6.6: Difference between random voltage reset and random threshold reset, for $v_\infty = 1.75$.

(Top) Random voltage reset. The solid and dashed lines represent the extremes of a random reset of $v_o$ into the range $[-.3, .3]$. When each of these traces reaches $\theta = 1$, a spike is generated; the interval between these first-passage times thus represents the range of possible interspike intervals. (Bottom) Random threshold reset. The solid and dashed lines represent the extremes of a random reset of $\theta$ into the range $[.7, 1.3]$. Each of these lines starts from the same reset value of $v_o = 0$. The solid line generates a spike when it reaches $\theta = 1.3$, while the dashed line spikes at $\theta = 0.7$. The interval between these first-passage times represents the range of possible interspike intervals. The range is much wider here than in the upper plot. For a much larger value of $v_\infty$, all voltage traces would be nearly linear, and there would be no difference between the random voltage and random threshold resets.
at low rates, and the effects on the coefficient of variability would be eliminated.

6.6.2 Effect of adaptation

The reason for the increase/decrease in variability for adapting neurons compared with tonic neurons at the same frequency is essentially the same effect discussed in section 6.6.1: reducing $v_\infty$ brings the neuron into a regime wherein the approach to threshold becomes dominated by leak terms, and the trajectories diverge from linearity. Figure 6.7 compares the value of $v_\infty$ at various firing rates for a tonic neuron with those for a neuron with adaptation. While $v_\infty$ increases linearly with the firing rate for the tonic neuron, the adapting neuron asymptotes to a small value of $v_\infty$ even at high rates, due to the decrease in $\tau$ and $I$ caused by increasing $c$. Thus the adapting neuron is in the small-$v_\infty$ regime discussed above, even when it is firing rapidly, and so it experiences the corresponding effects on its CV (a decrease in the random voltage reset case, and an increase in the random threshold reset case).

These effects on neural variability are something to be borne in mind in future modelling work using the integrate-and-fire model: noise introduced by a random reset may have unexpected effects on the variability in the presence of spike-frequency adaptation, depending on the choice of reset method.
Figure 6.7: Variation of $v_\infty$ with firing rate, with and without adaptation; results are from numerical runs with no random reset. Since the value of $v_\infty$ varies slightly during each interspike interval, (as $c$ is incremented, then decays), the value on the horizontal axis represents the average of $v_\infty$ over many intervals. Note that the tonic (no adaptation) case has $v_\infty$ increasing linearly with firing rate, while in the adapting case $v_\infty$ asymptotes to a small value.
Chapter 7

Summary and Conclusions

The work presented in this thesis constitutes part of a much larger effort, namely an attempt to extract principles from animal behaviour which may be applied to produce robots with the sort of behavioural competence displayed by real animals. The challenges involved are formidable, and we are still at an early stage of research: it is not clear which aspects of animal physiology are critical to their efficient operation, and which may be neglected. Progress towards robots rivalling the performance of biological organisms will depend on the efforts of many researchers, from many different fields; no single researcher can hope to carry out work in all of the required directions. My goal has been to find certain aspects of the larger problem on which I could make some progress, and to pursue work in these directions with the hope of contributing something to our current understanding.

Animals are built of muscles, bones, neurons, and so on: these are very different materials than those found in present-day robots, which are made mainly of metal, plastic, motors, microchips, and the like. There will therefore be a limit to how directly we may apply the information we obtain by studying animals to the task of building robots: many details of the operation of an animal's physical body will be neither reproducible in a robot, nor relevant to the robot's operation. (Does the fact that a squirrel comes equipped with a heart mean that our robotic squirrel must have an artificial heart, even if it has no "blood"?) Which aspects of an animal's physical constitution must be emulated to make a biologically inspired robot?

To address this question, we need to abstract the details of animal physiology into mathematical models; this abstraction is what will enable common principles to be implemented in substrates as different as cytoplasm and silicon. Like most other researchers, I have focussed on neurons, the signal-processing cells which are most directly responsible for an animal's behavioural patterns. Each chapter of the thesis has presented a model of some aspect of the dynamical behaviour of networks of neurons.

Chapter 2 described a simple method for adding spike-frequency adaptation (a common
property of real neurons) to existing analog neuron models; the resulting models were called "phasic analog neurons." Adaptation extends the dynamics of networks of analog neurons, and in particular, it introduces the possibility of oscillatory solutions in situations where there would otherwise be only fixed-point solutions. The mathematical analysis presented in the chapter enabled us to characterize the conditions under which oscillations would arise in a simple two-neuron network, and to predict the stability of the oscillatory cycles arising. Since analog neurons models are a popular means of representing neural activities in artificial systems, and especially in robotics, this extended range of dynamical behaviours may be of relevance to other researchers. The mathematical analysis provides guidance in choosing the parameter settings for a two neuron network such that the resulting oscillations have some desired form; this minimizes the amount of trial and error searching required.

In Chapter 3, one application of phasic analog neurons was presented: a network of twelve such neurons was used to generate command signals to be sent to the legs of a six-legged walking robot. Two concepts from biological locomotion were used to guide the design of the network: each leg was driven by a "central pattern generator" producing a baseline rhythm; and the motion of leg was influenced by a "stretch reflex" which resisted motion past a certain distance away from the neutral position. A number of biologically inspired networks based on analog neuron models have been used by other researchers to generate hexapod gaits. The network presented here constitutes a new variation on the usual form of such networks, and one which is unusual in its use of adaptation to generate the oscillatory rhythms.

Chapter 4 considered neural oscillators similar to those discussed in the previous two sections, but from a different perspective: rather than examining two analog neurons, two populations of individually spiking neurons were considered, with coupling between the two populations. Once again, oscillations arose in the presence of spike-frequency adaptation, and mathematical analysis provided predictions of the point of onset and the period of these oscillations. The main point of interest from a theoretical perspective was the ability to make such predictions for a system with a large number of dimensions, by simplifying the dynamics to a two-dimensional form. The analysis was based on earlier work describing the dynamics of neural populations [105], but extends the previous approach by considering the effect of spike-frequency adaptation. From an engineering perspective, oscillators consisting of a large number of individual units could have advantages in terms of robustness: since no single unit is responsible for the collective oscillatory behaviour, failure of one or more units will not destroy the oscillations.

Moving from motor behaviour to sensory processing, Chapter 5 discussed a signal-processing effect called noise shaping, which has recently [36] been shown to arise in networks of coupled model neurons. The chapter presents computational results extending the previous work: the effect is shown not to rely on the specific type of noise introduced into the system; the addition of spike-frequency adaptation is shown to improve the network's ability to detect signals; and the
noise-shaping effect is shown to be present in a more elaborate model than that previous used, suggesting that the phenomenon may arise generically in networks of spiking units, rather than as a consequence of the specific model selected for the initial studies. Once again, the point of interest from an engineering perspective is the illustration that a highly parallel, highly redundant system can carry out effective signal processing. Since such a system would be expected to be very robust, it is of interest to attempt to understand the principles involved, and these computational studies have provided the basis for theoretical work now in progress.

Various methods have been used to inject noise into model neurons, and Chapter 6 presented some simulations and analysis of the effect of spike-frequency adaptation on two popular sources of noise used with the integrate-and-fire model. If researchers are to use integrate-and-fire model with spike-frequency adaptation present, they must bear in mind that it may change the effect of the noise that they inject into the model; when dealing with coupled networks, such a change is not always clearly visible in the network output. By considering individual neurons, the work presented in the chapter clarifies the source of the effect, allowing researchers using such models to account for it.

The task of trying to understand how animals work is one of the most challenging facing modern science, and we have only just begun. The vast complexity of biological systems is attracting more and more engineers, physical scientists, and mathematicians to study them; as the efforts of many researchers accumulate over time, we may one day reach the point where we have a level of understanding which will enable us to build robots able to deal with their environment as well as animals do. I hope that this thesis has provided some small contribution towards that goal.
Bibliography


BIBLIOGRAPHY


