# Neuronal Dynamics

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## From Single Neurons to Networks and Models of Cognition

Wulfram Gerstner, Werner M. Kistler, Richard Naud and Liam Paninski

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# Preface

This textbook for advanced undergraduate and beginning graduate students provides a systematic introduction into the fields of Neuron Modeling, Neuronal Dynamics, Neural Coding and Neural Networks. It can be used as a text for introductory courses to Computational and Theoretical Neuroscience or as main text for a more focused course on Neural Dynamics and Neural Modeling at the graduate level. The book is also useful resource for Researchers and Students who want to learn how different models of neurons and descriptions of neural activity are related to each other.

All mathematical concepts are introduced the pedestrian way: step by step. All chapters are richly illustrated by figures and worked-out examples. Each chapter closes with a short Summary and a series of mathematical Exercises. An the authors' WEB page Python source code is provided for numerical simulations that illustrate the main ideas and models of the chapter.

The book is organized into four parts with a total of 20 Chapters. Part I provides a general introduction into the foundations of Computational Neuroscience and its mathematical tools. It covers classic material such as the Hodgkin-Huxley model, ion channels and dendrites, or phase plane analysis of two-dimensional systems of differential equations. A special focus is put on the firing threshold for the generation of action potentials, in the Hodgkin-Huxley models, as well as in reduced two-dimensional neuron models such as the Morris-Lecar model.

Part II focuses on Simplified Models for the dynamics of a *single* neuron. It covers nonlinear integrate-and-fire models with and without adaptation, in particular the quadratic and exponential integrate-and-fire model, as well as the Izhikevich-model and Adaptive Exponential Integrate-and-Fire model. The question of noise in the neural dynamics is posed and two classic descriptions of noise are presented. First, stochasticity arising from random spike arrival. This approach leads to a noise term in the differential equation of the voltage, and can be formulated as a Langevin equation. Second, intrinsic stochasticity of neurons leading to an 'escape' across the firing threshold even when then neuron is in the subthreshold regime. This approach leads to the framework of a Generalized Linear Model which is systematically introduced and discussed in applications of neuronal coding and decoding. The relation between the neuron models of Part II and biological data is highlighted and systematic parameter optimization algorithms are presented.

Part III takes the simplified models derived in part II and builds networks out of these. The collective properties of the network dynamics are described in terms of equations for the population activity also called population firing rate. The conditions und which population activity can be described by standard rate model are identified.

Part IV makes the link from dynamics to cognition. The population activity equations

are used for an analysis of famous paradigms of Computational and Cognitive Neuroscience, such as the neural activity during decision making or memory retrieval. In Part IV we also sketch the theory of learning in relation to synaptic plasticity. The book closes with a fascinating application of the principles of neuronal dynamics to help patients suffering from Parkinsons desease.

A small fraction of the text of the present book is based on 'Spiking Neuron Models' (Cambridge Univ. Press) which was first published in 2002 and reprinted several times since then. In the mean time, the field has changed and we felt that a simple update of 'Spiking Neuron Models' for a second edition would not be enough to give credits to the developments that have occurred.

Scientifically, the scope of 'Spiking Neuron Models' was limited in several respects: First, it mainly focused on *linear* integrate-and-fire models, and mentioned their nonlinear counterparts only in passing. In the present book, nonlinear integrate-and-fire models are treated in a full chapter. Second, adaptation was neglected in the treatment 10 years ago - mainly because population equations for adaptive neurons were not yet available. In the present book, adaptive integrate-and-fire models are covered at length in a separate chapter and the population activity equations for adaptive neurons are derived. Third, while the Spike Response Model with escape noise has always contained all the features of a Generalized Linear Model (GLM), by the year 2002 the theory of GLMs had not yet found its way into the field of neuroscience and was therefore simply absent from the old book. Given the phenomenal rise of GLMs in neuroscience, the theory of GLM for fitting neuronal data features at a prominent location in the present book. Finally, during teaching we always felt the need to show famous applications of the principles of neuronal dynamics, such as retrieval of contents from associative memories or decision dynamics and the neuroscience of free will. The present book covers these topics.

On a more general level, we felt that it would be useful to have a book that is, from the beginning, designed as a textbook rather than a monograph. Therefore, the present book makes the link to experimental data more visible, has more explanatory text, and, last not least, provides a series of exercises that have already been tested in the classroom over several years.

We hope that this book will be useful for students and researchers alike

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**Advice to the Reader:**

Each chapter starts with a specific question and gives first intuitive answers in the first section. As the chapter proceeds the material gets more advanced, and the presentation becomes more technical. For a first reading of the book, it is possible to read only the first section, or first two sections, of each Chapter and just glance at the subsequent sections.

More specific advice depends on the background. For example, readers who are new to the field of computational neuroscience are advised to spend enough time with the classic material of Part I, before they move on to part II and IV. The expert reader may skip part I completely and start directly with part II.

In part III, the main ideas are exposed in Chapters 12 and 15 which present the foundations for the rate models in part IV. The more technical chapters and sections of part III can be skipped at a first reading, but are necessary for a thorough understanding of the current developments in the field of computational neuroscience.

Part IV contains applications of neuronal dynamics to questions of cognition and can be read in any arbitrary order.

**Acknowledgements**. We would like to thank our students, visitors, exchange students, and postdocs who carefully read and commented each on at least two chapters, some even many more: Dane Corneil, Andrea De Antoni, Mortiz Deger, Mohammad Faraji, Felipe Gerhard, Laureline Logiaco, Skander Mensi, Alexandre Payeur, Christian Pozzorini, Kerstin Preuschoff, Tilo Schwalger, Alex Seeholzer, Hesam Setareh, Carlos Stein, Tim Vogels, Friedemann Zenke, Lorric Ziegler.

The writing of the text was a joint work of the four authors. Werner Kistler and Wulfram Gerstner were the authors of 'Spiking Neuron Models' from which several sections survived. Liam Paninski was mainly involved in Chapters 9-11 of the present book and gave valuable input to other chapers of Part II. Richard Naud contributed to writing chapters 1-11 and 14 with a leading role in some of these, made valuable comments and suggestions for all other chapters, and was responsible for all the figures. Wulfram Gerstner wrote the first drafts of part III and IV and contributed text to all other chapters.

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# Contents

# Part I

# Foundations of Neuronal Dynamics

# Chapter 1

# Introduction: Neurons and Mathematics

The primary aim of this chapter is to introduce several elementary notions of neuroscience, in particular the concepts of action potentials, postsynaptic potentials, firing thresholds, refractoriness, and adaptation. Based on these notions a preliminary model of neuronal dynamics is built and this simple model (the leaky integrate-and-fire model) will be used as a starting point and reference for the generalized integrate-and-fire models, which are the main topic of the book, to be discussed in Parts II and III. Since the mathematics used for the simple model is essentially that of a one-dimensional linear differential equation, we take this first chapter as an opportunity to introduce some of the mathematical notation that will be used throughout the rest of the book.

Due to the limitations of space we cannot – and do not want to – give a comprehensive introduction into such a complex field as neurobiology. The presentation of the biological background in this chapter is therefore highly selective and focuses on those aspects needed to appreciate the biological background of the theoretical work presented in this book. For an in-depth discussion of neurobiology we refer the reader to the literature mentioned at the end of this chapter.

After the review of neuronal properties in Section 1.1 and 1.2 we will turn, in Section 1.3, to our first mathematical neuron model. The last two sections are devoted to a discussion of the strengths and limitations of simplified models.

## 1.1 Elements of Neuronal Systems

Over the past hundred years, biological research has accumulated an enormous amount of detailed knowledge about the structure and function of the brain. The elementary processing units in the central nervous system are neurons, which are connected to each other in an intricate pattern. A tiny portion of such a network of neurons is sketched in Fig. 1.1, which shows a drawing by Ramón y Cajal, one of the pioneers of neuroscience around 1900. We can distinguish several neurons with triangular or circular cell bodies and long wire-like extensions. This picture can only give a glimpse of the network of neurons in the cortex. In reality, cortical neurons and their connections are packed into a dense network with more than $10^4$ cell bodies and several kilometers of 'wires' per cubic

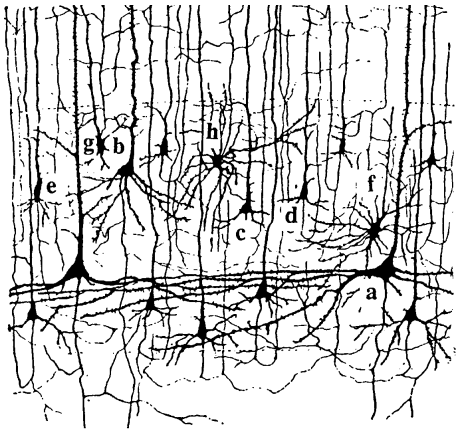


**Fig.** 1.1: This reproduction of a drawing of Ramón y Cajal shows a few neurons in the mammalian cortex that he observed under the microscope. Only a small portion of the neurons contained in the sample of cortical tissue have been made visible by the staining procedure; the density of neurons is in reality much higher. Cell b is a typical example of a pyramidal cell with a triangularly shaped cell body. Dendrites, which leave the cell laterally and upwards, can be recognized by their rough surface. The axons are recognizable as thin, smooth lines which extend downwards with a few branches to the left and right. From Ramòn y Cajal (1909).

millimeter. Across areas of the brain the wiring pattern may look different. In all areas, however, neurons of different sizes and shapes form the basic elements.

Still, the cortex does not consist exclusively of neurons. Beside the various types of neuron, there is a large number of 'supporter' cells, so-called glia cells, that are required for energy supply and structural stabilization of brain tissue. Since glia cells are not directly involved in information processing, we will not discuss them any further. We will also neglect a few rare subtypes of neuron, such as non-spiking neurons in the mammalian retina. Throughout this book we concentrate on spiking neurons only.

### 1.1.1 The Ideal Spiking Neuron

A typical neuron can be divided into three functionally distinct parts, called dendrites, soma, and axon; see Fig. 1.2. Roughly speaking, the dendrites play the role of the 'input device' that collects signals from other neurons and transmits them to the soma. The soma is the 'central processing unit' that performs an important non-linear processing step: If the total input arriving at the soma exceeds a certain threshold, then an output signal is generated. The output signal is taken over by the 'output device', the axon, which delivers the signal to other neurons.

The junction between two neurons is called a synapse. Let us suppose that a neuron sends a signal across a synapse. It is common to refer to the sending neuron as the presynaptic cell and to the receiving neuron as the postsynaptic cell. A single neuron in vertebrate cortex often connects to more than $10^4$ postsynaptic neurons. Many of its axonal branches end in the direct neighborhood of the neuron, but the axon can also stretch over several centimeters so as to reach neurons in other areas of the brain.

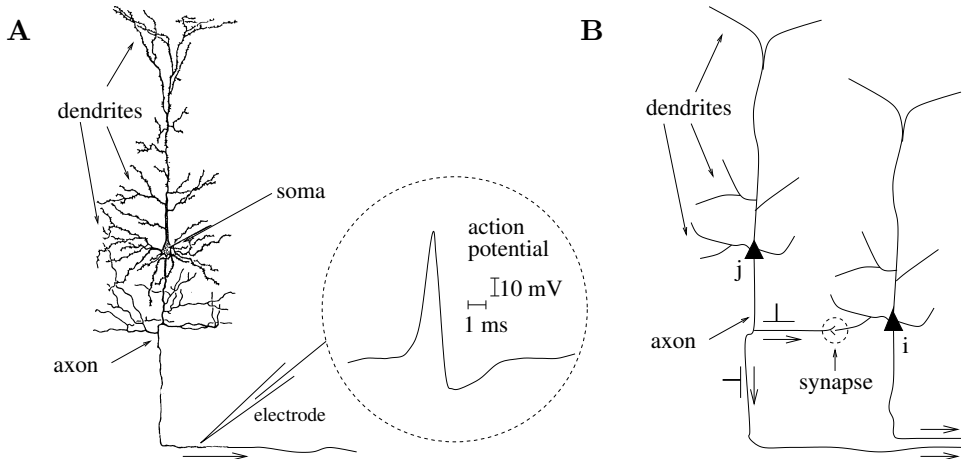


**Fig.** 1.2: **A**. Single neuron in a drawing by Ramón y Cajal. Dendrite, soma, and axon can be clearly distinguished. The inset shows an example of a neuronal action potential (schematic). The action potential is a short voltage pulse of 1-2 ms duration and an amplitude of about 100 mV. **B**. Signal transmission from a presynaptic neuron $j$ to a postsynaptic neuron $i$. The synapse is marked by the dashed circle. The axons at the lower right end lead to other neurons. (Schematic figure.)

### 1.1.2 Spike Trains

The neuronal signals consist of short electrical pulses and can be observed by placing a fine electrode either on the soma or close to the soma or axon of a neuron; see Fig. 1.2. The pulses, so-called action potentials or spikes, have an amplitude of about 100 mV and typically a duration of 1-2 ms. The form of the pulse does not change as the action potential propagates along the axon. A chain of action potentials emitted by a single neuron is called a spike train – a sequence of stereotyped events which occur at regular or irregular intervals; see Fig. 1.3. Since isolated spikes of a given neuron look alike, the form of the action potential does not carry any information. Rather, it is the number and the timing of spikes which matter. The action potential is the elementary unit of signal transmission.

Action potentials in a spike train are usually well separated. Even with very strong input, it is impossible to excite a second spike during or immediately after a first one. The minimal distance between two spikes defines the absolute refractory period of the neuron. The absolute refractory period is followed by a phase of relative refractoriness where it is difficult, but not impossible to excite an action potential.

### 1.1.3 Synapses

The site where the axon of a presynaptic neuron makes contact with the dendrite (or soma) of a postsynaptic cell is the synapse. The most common type of synapse in the vertebrate brain is a chemical synapse. At a chemical synapse, the axon terminal comes very close to the postsynaptic neuron, leaving only a tiny gap between pre- and postsynaptic cell membrane. This is called the synaptic cleft. When an action potential arrives at a synapse, it triggers a complex chain of bio-chemical processing steps that lead to

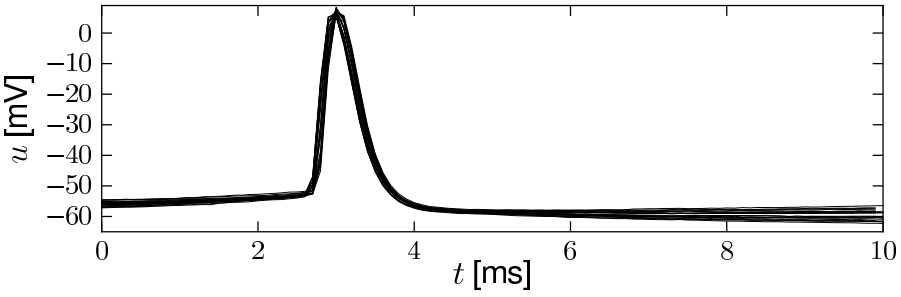


**Fig.** 1.3: Action potentials are stereotypical events. Membrane potential recordings aligned on the time of maximum voltage show little variability of the action potential shape. Data is a courtesy of Maria Toledo-Rodriguez and Henry Markram (Toledo-Rodriguez et al., 2004).

a release of neurotransmitter from the presynaptic terminal into the synaptic cleft. As soon as transmitter molecules have reached the postsynaptic side, they will be detected by specialized receptors in the postsynaptic cell membrane and lead (either directly or via a biochemical signaling chain) to an opening of specific channels causing ions from the extracellular fluid to flow into the cell. The ion influx, in turn, changes the membrane potential at the postsynaptic site so that, in the end, the chemical signal is translated into an electrical response. The voltage response of the postsynaptic neuron to a presynaptic spike is called the postsynaptic potential.

Apart from chemical synapses neurons can also be coupled by electrical synapses, sometimes called gap junctions. Specialized membrane proteins make a direct electrical connection between the two neurons. Not much is known about the functional aspects of gap junctions, but they are thought to be involved in the synchronization of neurons.

### 1.1.4 Neurons are part of a big system

Neurons are embedded in a network of billions of other neurons and glial cell that make up the brain tissue. The brain is organized in different regions and areas. The cortex can be thought of as a thin but extended sheet of neurons, folded over other brain structures. Some cortical areas are mainly involved in processing sensory input, other areas are involved in working memory or motor control.

Neurons in sensory cortices can be experimentally characterized by the stimuli to which they exhibit a strong response. For example, neurons in the primary visual cortex respond to dots of lights only within a small region of the visual space. The limited zone where a neuron is sensitive to stimuli is called the neuron's receptive field (Fig. 1.4).

The receptive field of so-called simple cells in visual cortex is not homogeneous, but has typically two of three elongated subfields. When a light dot falls into one of the positive subfields, the neuron increases its activity, i.e., it emits more spikes than in the absence of a stimulus. Whenever a light dot falls into a negative subfield, it decreases the activity compared to its spontaneous activity in the presence of a gray screen. A spot of light is in fact not the best stimulus. The neuron responds maximally to a moving light bar with an orientation aligned with the elongation of the positive subfield (Hubel and Wiesel, 1968).

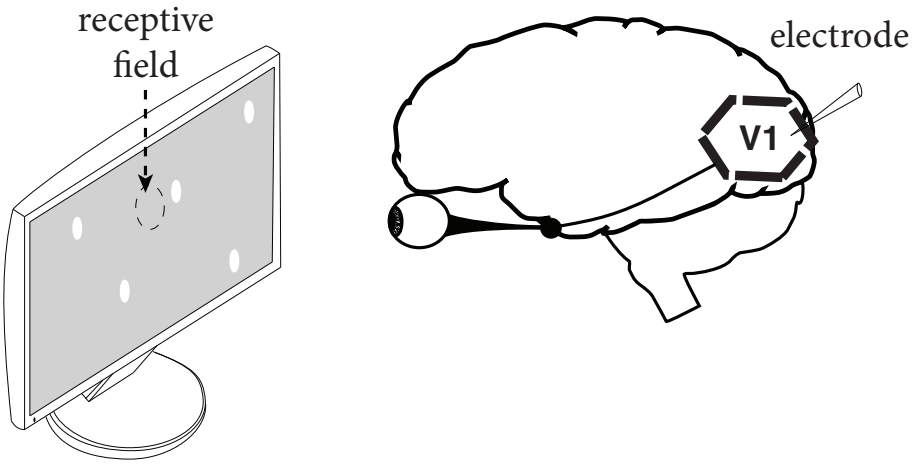


**Fig.** 1.4: Receptive fields in visual cortex. An electrode probes the activity of a neuron while light dots are presented on a gray screen. The neuron responds whenever the stimulus falls into its receptive field, schematically indicated as an oval.

A large body of the neuroscience literature consists in determining the receptive fields of neurons in sensory cortices. While neurons in visual cortex respond to appropriate visual stimuli, neurons in auditory cortex or somatosensory cortex respond to auditory or tactile stimuli. The concept of receptive field becomes less well defined if one moves away from sensory cortex. For example, in inferotemporal cortex, neurons respond to objects independently of their size and location; in working memory tasks, frontal cortex neurons are active during periods where no stimulus is present at all. In Parts II, III, and IV of this book we touch on aspects of receptive fields and memory of neuronal networks embedded in a big system. For the moment, we return to a simple, idealized neuron.

## 1.2 Elements of Neuronal Dynamics

The effect of a spike on the postsynaptic neuron can be recorded with an intracellular electrode which measures the potential difference $u(t)$ between the interior of the cell and its surroundings. This potential difference is called the membrane potential. Without any input, the neuron is at rest corresponding to a constant membrane potential $u_{\rm rest}$. After the arrival of a spike, the potential changes and finally decays back to the resting potential, cf. Fig. 1.5A. If the change is positive, the synapse is said to be excitatory. If the change is negative, the synapse is inhibitory.

At rest, the cell membrane has already a strongly negative polarization of about – 65 mV. An input at an excitatory synapse reduces the negative polarization of the membrane and is therefore called depolarizing. An input that increases the negative polarization of the membrane even further is called hyperpolarizing.

### 1.2.1 Postsynaptic Potentials

Let us formalize the above observation. We study the time course $u_i(t)$ of the membrane potential of neuron $i$. Before the input spike has arrived, we have $u_i(t) = u_{\rm rest}$. At $t = 0$

the presynaptic neuron $j$ fires its spike. For $t > 0$, we see at the electrode a response of neuron $i$

$$u_i(t) - u_{\rm rest} =: \epsilon_{ij}(t)\,. \tag{1.1}$$

The right-hand side of Eq. (1.1) defines the postsynaptic potential (PSP). If the voltage difference $u_i(t) - u_{\rm rest}$ is positive (negative) we have an excitatory (inhibitory) postsynaptic potential or short EPSP (IPSP). In Fig. 1.5A we have sketched the EPSP caused by the arrival of a spike from neuron $j$ at an excitatory synapse of neuron $i$.

### 1.2.2 Firing Threshold and Action Potential

Consider two presynaptic neurons $j = 1, 2$, which both send spikes to the postsynaptic neuron $i$. Neuron $j = 1$ fires spikes at $t_1^{(1)}, t_1^{(2)}, \ldots$, similarly neuron $j = 2$ fires at $t_2^{(1)}, t_2^{(2)}, \ldots$. Each spike evokes a postsynaptic potential $\epsilon_{i1}$ or $\epsilon_{i2}$, respectively. As long as there are only few input spikes, the total change of the potential is approximately the sum of the individual PSPs,

$$u_i(t) = \sum_j \sum_f \epsilon_{ij}(t - t_j^{(f)}) + u_{\rm rest}\,, \tag{1.2}$$

i.e., the membrane potential responds linearly to input spikes; see Fig. 1.5B.

On the other hand, linearity breaks down if too many input spikes arrive during a short interval. As soon as the membrane potential reaches a critical value $\vartheta$, its trajectory shows a behavior that is quite different from a simple summation of PSPs: The membrane potential exhibits a pulse-like excursion with an amplitude of about 100 mV. This short voltage pulse will propagate along the axon of neuron $i$ to the synapses with other neurons. After the pulse the membrane potential does not directly return to the resting potential, but passes, for many neuron types, through a phase of hyperpolarization below the resting value. This hyperpolarization is called 'spike-afterpotential'.

Single EPSPs have amplitudes in the range of one millivolt. The critical value for spike initiation is about 20 to 30 mV above the resting potential. In most neurons, four spikes – as shown schematically in Fig. 1.5C – are thus not sufficient to trigger an action potential. Instead, about 20-50 presynaptic spikes have to arrive within a short time window to trigger a postsynaptic action potential.

## 1.3 Integrate-And-Fire Models

We have seen in the previous section that, to a first and rough approximation, neuronal dynamics can be conceived as a summation process (sometimes also called 'integration' process) combined with a mechanism that triggers action potentials above some critical voltage. Indeed in experiments firing times are often defined as the moment when the membrane potential reaches some threshold value from below. In order to build a phenomenological model of neuronal dynamics, we describe the critical voltage for spike initiation by a formal threshold $\vartheta$. If the voltage $u_i(t)$ (that contains the summed effect of all inputs) reaches $\vartheta$ from below, we say that neuron $i$ fires a spike. The moment of threshold crossing defines the firing time $t_i^{(f)}$.

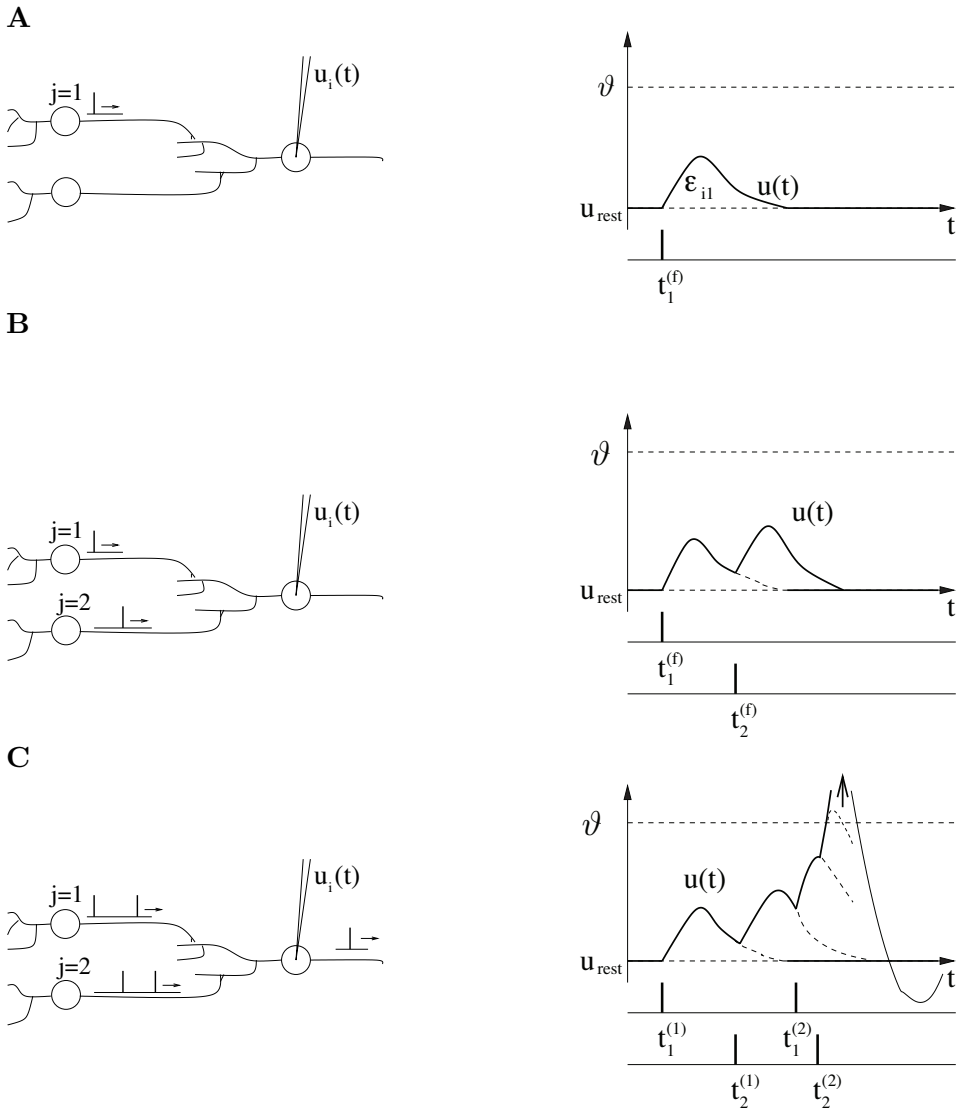


**Fig.** 1.5: A postsynaptic neuron $i$ receives input from two presynaptic neurons $j = 1, 2$. **A**. Each presynaptic spike evokes an excitatory postsynaptic potential (EPSP) that can be measured with an electrode as a potential difference $u_i(t) - u_{\rm rest}$. The time course of the EPSP caused by the spike of neuron $j = 1$ is $\epsilon_{i1}(t - t_1^{(f)})$. **B**. An input spike from a second presynaptic neuron $j = 2$ that arrives shortly after the spike from neuron $j = 1$, causes a second postsynaptic potential that adds to the first one. **C**. If $u_i(t)$ reaches the threshold $\vartheta$, an action potential is triggered. As a consequence, the membrane potential starts a large positive pulse-like excursion (arrow). On the voltage scale of the graph, the peak of the pulse is out of bounds. After the pulse the voltage returns to a value below the resting potential $u_{\rm rest}$.

The model makes use of the fact that neuronal action potentials of a given neuron always have roughly the same form. If the shape of an action potential is always the same, then the shape cannot be used to transmit information: rather information is contained in the presence or absence of a spike. Therefore action potentials are reduced to 'events' that happen at a precise moment in time.

Neuron models where action potentials are described as events are called 'Integrate-and-Fire' models. No attempt is made to describe the shape of an action potential. Integrate-and-fire models have two separate components that are both necessary to define their dynamics: first, an equation that describes the evolution of the membrane potential $u_i(t)$; and second, a mechanism to generate spikes.

In the following we introduce the simplest model in the class of integrate-and-fire models using the following two ingredients: (i) a *linear* differential equation to describe the evolution of the membrane potential; (ii) a threshold for spike firing. This model is called the 'Leaky Integrate-and-Fire' Model. Generalized integrate-and-fire models that will be discussed in Part II of the book can be seen as variations of this basic model.

### 1.3.1 Integration of Inputs

The variable $u_i$ describes the momentary value of the membrane potential of neuron $i$. In the absence of any input, the potential is at its resting value $u_{\rm rest}$. If an experimentalist injects a current $I(t)$ into the neuron, or if the neuron receives synaptic input from other neurons, the potential $u_i$ will be deflected from its resting value.

In order to arrive at an equation that links the momentary voltage $u_i(t) - u_{\rm rest}$ to the input current $I(t)$, we use elementary laws from the theory of electricity. A neuron is surrounded by a cell membrane, which is a rather good insulator. If a short current pulse $I(t)$ is injected into the neuron, the additional electrical charge $q = \int I(t')dt'$ has to go somewhere: it will charge the cell membrane (Fig. 1.6A). The cell membrane therefore acts like a capacitor of capacity $C$. Because the insulator is not perfect, the charge will, over time, slowly leak through the cell membrane. The cell membrane can therefore be characterized by a finite leak resistance $R$.

The basic electrical circuit representing a leaky integrate-and-fire model consists of a capacitor $C$ in parallel with a resistor $R$ driven by a current $I(t)$; see Fig. 1.6. If the driving current $I(t)$ vanishes, the voltage across the capacitor is given by the battery voltage $u_{\rm rest}$. For a biological explanation of the battery we refer the reader to the next chapter. Here we have simply inserted the battery 'by hand' into the circuit so as to account for the resting potential of the cell (Fig. 1.6A).

In order to analyze the circuit, we use the law of current conservation and split the driving current into two components,

$$I(t) = I_R + I_C \tag{1.3}$$

The first component is the resistive current $I_R$ which passes through the linear resistor $R$. It can be calculated from Ohm's law as $I_R = u_R/R$ where $u_R = u - u_{\rm rest}$ is the voltage across the resistor. The second component $I_C$ charges the capacitor $C$. From the definition of the capacity as $C = q/u$ (where $q$ is the charge and $u$ the voltage), we find a

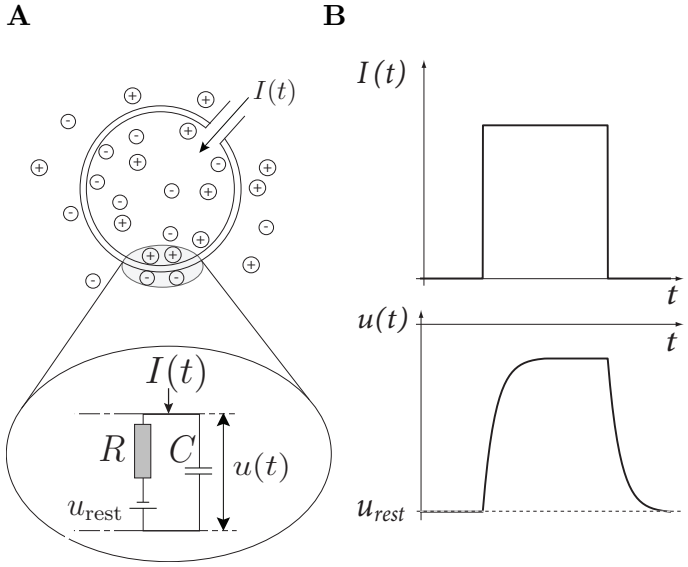


**Fig.** 1.6: Electrical properties of neurons: the passive membrane. **A**. A neuron, which is enclosed by the cell membrane (big circle), receives a (positive) input current $I(t)$ which increases the electrical charge inside the cell. The cell membrane acts like a capacitor in parallel with a resistor which is in line with a battery of potential $u_{\rm rest}$ (zoomed inset). **B**. The cell membrane reacts to a step current (top) with a smooth voltage trace (bottom).

capacitive current $I_C = {\rm d}q/{\rm d}t = C\,{\rm d}u/{\rm d}t$. Thus

$$I(t) = \frac{u(t) - u_{\rm rest}}{R} + C\,\frac{{\rm d}u}{{\rm d}t}\,. \tag{1.4}$$

We multiply Eq. (1.4) by $R$ and introduce the time constant $\tau_m = R\,C$ of the 'leaky integrator'. This yields the standard form

$$\tau_m\,\frac{{\rm d}u}{{\rm d}t} = -[u(t) - u_{\rm rest}] + R\,I(t)\,. \tag{1.5}$$

We refer to $u$ as the membrane potential and to $\tau_m$ as the membrane time constant of the neuron.

From the mathematical point of view, Eq. (1.5) is a linear differential equation. From the point of view of an electrical engineer, it is the equation of a leaky integrator or $RC$-circuit where resistor $R$ and capacitor $C$ are arranged in parallel. From the point of view of the neuroscientist, Eq. (1.5) is called the equation of a passive membrane.

What is the solution of Eq. (1.5)? We suppose that, for whatever reason, at time $t = 0$ the membrane potential takes a value $u_{\rm rest} + \Delta u$. For $t > 0$ the input vanishes $I(t) = 0$. Intuitively we expect that, if we wait long enough, the membrane potential relaxes to its resting value $u_{\rm rest}$. Indeed, the solution of the differential equation with initial condition $u(t_0) = u_{\rm rest} + \Delta u$ is

$$u(t) - u_{\rm rest} = \Delta u\,\exp\left(-\frac{t - t_0}{\tau_m}\right) \quad \text{for } t > t_0\,. \tag{1.6}$$

Thus, in the absence of input, the membrane potential decays exponentially to its resting value. The membrane time constant $\tau_m = RC$ is the characteristic time of the decay. For a typical neuron it is in the range of 10ms, and hence rather long compared to the duration of a spike which is on the order of 1ms.

The validity of the solution (1.6) can be checked by taking the derivative on both sides of the equation. Since it is the solution in the absence of input, it is sometimes called the 'free' solution.

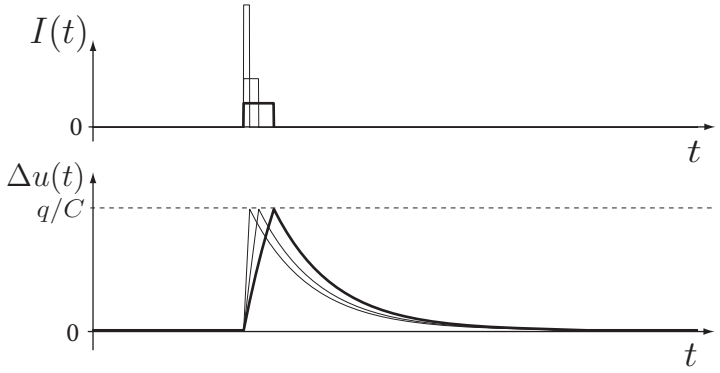


**Fig.** 1.7: Short pulses and total charged delivered on the passive membrane. The amplitude of the voltage response (bottom) of a leaky integrator driven by a short current pulse $I(t)$ (top) depends only on the total charge $q = \int I(t)dt$, but not on the height of the current pulse.

### 1.3.2 Pulse Input

Before we continue with the definition of the integrate-and-fire model and its variants, let us study the dynamics of the passive membrane defined by Eq. (1.5) in a simple example. Suppose that the passive membrane is stimulated by a constant input current $I(t) = I_0$ which starts at $t = 0$ and ends at time $t = \Delta$. For the sake of simplicity we assume that the membrane potential at time $t = 0$ is at its resting value $u(0) = u_{\rm rest}$.

As a first step, let us calculate the time course of the membrane potential. The trajectory of the membrane potential can be found by integrating (1.5) with the initial condition $u(0) = u_{\rm rest}$. The solution for $0 < t < \Delta$ is

$$u(t) = u_{\rm rest} + R\,I_0 \left[1 - \exp\left(-\frac{t}{\tau_m}\right)\right] . \tag{1.7}$$

If the input current never stopped, the membrane potential (1.7) would approach for $t \to \infty$ the asymptotic value $u(\infty) = u_{\rm rest} + R\,I_0$. We can understand this result by looking at the electrical diagram of the $RC$-circuit in Fig. 1.6. Once a steady state is reached, the charge on the capacitor no longer changes. All input current must then flow through the resistor. The steady-state voltage at the resistor is therefore $RI_0$ so that the total membrane voltage is $u_{\rm rest} + RI_0$.

**Example: Short pulses and the Dirac $\delta$ function**

For short pulses the steady state value is never reached. At the end of the pulse, the value of the membrane potential is given according to Eq. (1.7) by $u(\Delta) = u_{\rm rest} + R\,I_0 \left[1 - \exp\left(-\frac{\Delta}{\tau_m}\right)\right]$. For pulse durations $\Delta \ll \tau_m$ (where $\ll$ means much smaller than) we can expand the exponential term into a Taylor series: $\exp(x) = 1 + x + x^2/2 \ldots$. To first order in $x = -\frac{\Delta}{\tau_m}$ we find

$$u(\Delta) = u_{\rm rest} + R\,I_0 \frac{\Delta}{\tau_m} \quad \text{for } \Delta \ll \tau_m. \tag{1.8}$$

Thus, the voltage deflection depends linearly on the amplitude and the duration of the pulse (Fig. 1.7, thick line).

We now make the duration $\Delta$ of the pulse shorter and shorter while increasing the amplitude of the current pulse to a value $I_0 = q/\Delta$, so that the integral $\int I(t)\mathrm{d}t = q$ remains constant. In other words, the total charge $q$ delivered by the current pulse is always the same. Interestingly, the voltage deflection at the end of the pulse calculated from Eq. (1.8) remains unaltered, however short we make the pulse. Indeed, from Eq. (1.8) we find $u(\Delta) - u_{\rm rest} = q\,R/\tau_m = q/C$ where we have used $\tau_m = RC$. Thus we can consider the limit

of an infinitely short pulse

$$I(t) = q\,\delta(t) = \lim_{\Delta\to 0} \frac{q}{\Delta} \quad \text{for } 0 < t < \Delta \quad \text{and 0 otherwise.} \tag{1.9}$$

$\delta(t)$ is called the Dirac $\delta$-function. It is defined by $\delta(x) = 0$ for $x \neq 0$ and $\int_{-\infty}^{\infty} \delta(x)\mathrm{d}x = 1$.

Obviously, the Dirac $\delta$-function is a mathematical abstraction since it is practically impossible to inject a current with an infinitely short and infinitely strong current pulse into a neuron. Whenever we encounter a $\delta$-function, we should remember that, as a stand-alone object, it looks strange, but becomes meaningful as soon as we integrate over it. Indeed the input current defined in Eq. (1.9) needs to be inserted into the differential equation (1.5) and integrated. The mathematical abstraction of the Dirac $\delta$ function suddenly makes a lot of sense, because the voltage change induced by a short current pulse is always the same, whenever the duration of the pulse $\Delta$ is much shorter than the time constant $\tau_m$. Thus, the exact duration of the pulse is irrelevant, as long as it is short enough.

With the help of the $\delta$-function, we no longer have to worry about the time course of the membrane potential *during* the application of the current pulse: the membrane potential simply jumps at time $t = 0$ by an amount $q/C$. Thus, it is as if we added instantaneously a charge $q$ onto the capacitor of the $RC$ circuit.

What happens for times $t > \Delta$? The membrane potential evolves from its new initial value $u_{\rm rest} + q/C$ in the absence of any further input. Thus we can use the 'free' solution from Eq. (1.6) with $t_0 = \Delta$ and $\Delta u = q/C$.

We can summarize the considerations of this subsection by the following statement. The solution of the linear differential equation with pulse input

$$\tau_m \frac{\mathrm{d}u}{\mathrm{d}t} = -[u(t) - u_{\rm rest}] + R\,q\,\delta(t)\,. \tag{1.10}$$

is $u(t) = u_{\rm rest}$ for $t \leq 0$ and given by

$$u(t) - u_{\rm rest} = q\,\frac{R}{\tau_m}\,\exp\left(-\frac{t}{\tau_m}\right) \quad \text{for } t > 0 \tag{1.11}$$

The right-hand side of the equation is called the impulse-response function or Green's function of the linear differential equation.

### 1.3.3 The Threshold for Spike Firing

Throughout this book, the term 'firing time' refers to the moment when a given neuron emits an action potential $t^{(f)}$. The firing time $t^{(f)}$ in the leaky integrate-and-fire model is defined by a threshold criterion

$$t^{(f)}: \quad u(t^{(f)}) = \vartheta\,. \tag{1.12}$$

The form of the spike is not described explicitly. Rather, the firing time is noted and immediately after $t^{(f)}$ the potential is reset to a new value $u_r < \vartheta$,

$$\lim_{\delta\to 0;\delta>0} u(t^{(f)} + \delta) = u_r\,. \tag{1.13}$$

For $t > t^{(f)}$ the dynamics is again given by (1.5) until the next threshold crossing occurs. The combination of leaky integration (1.5) and reset (1.13) defines the leaky integrate-and-fire model (Stein, 1967b). The voltage trajectory of a leaky integrate-and-fire model driven by a constant current $I_0$ is shown in Fig. 1.9.

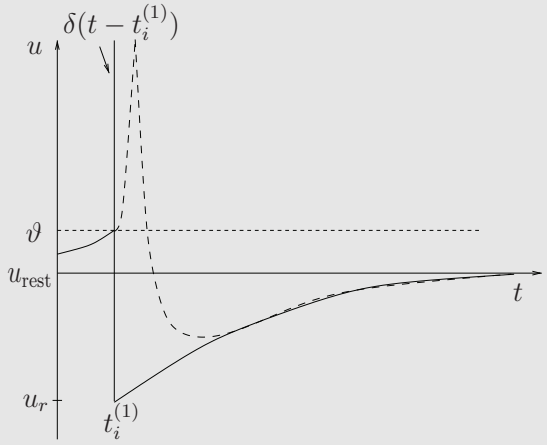


**Fig.** 1.8: In formal models of spiking neurons the shape of an action potential (dashed line) is usually replaced by a $\delta$ pulse (vertical line). The negative overshoot (spike-afterpotential) after the pulse is replaced by a 'reset' of the membrane potential to the value $u_r$. The pulse is triggered by the threshold crossing at $t_i^{(1)}$.

For the firing times of neuron $i$ we write $t_i^{(f)}$ where $f = 1, 2, \ldots$ is the label of the spike. Formally, we may denote the spike train of a neuron $i$ as the sequence of firing times

$$S_i(t) = \sum_f \delta(t - t_i^{(f)}) \tag{1.14}$$

where $\delta(x)$ is the Dirac $\delta$ function introduced before, with $\delta(x) = 0$ for $x \neq 0$ and $\int_{-\infty}^{\infty} \delta(x) \mathrm{d}x = 1$. Spikes are thus reduced to points in time (Fig. 1.8). We remind the reader that the $\delta$-function is a mathematical object that needs to be inserted into an integral in order to give meaningful results.

### 1.3.4 Time-dependent Input (*)[1]

We study a leaky integrate-and-fire model which is driven by an arbitrary time-dependent input current $I(t)$; cf. Fig. 1.9B. The firing threshold has a value $\vartheta$ and after firing the potential is reset to a value $u_r < \vartheta$.

In the absence of a threshold, the linear differential equation (1.5) has a solution

$$u(t) = u_{\rm rest} + \frac{R}{\tau_m} \int_0^{\infty} \exp\left(-\frac{s}{\tau_m}\right) I(t-s)\, \mathrm{d}s\,. \tag{1.15}$$

where $I(t)$ is an arbitrary input current and $\tau_m = RC$ is the membrane time constant. We assume here that the input current is defined for a long time back into the past: $t \to -\infty$ so that we do not have to worry about the initial condition. A sinusoidal current $I(t) = I_0 \sin(\omega\, t)$ or a step current pulse, $I(t) = I_0 \Theta(t)$ where $\Theta$ denotes the Heaviside step function with $\Theta(t) = 0$ for $t \leq 0$ and $\Theta(t) = 1$ for $t > 0$, are two examples of a

[1] Sections marked by an asterisk are mathematically more advanced and can be omitted during a first reading of the book.

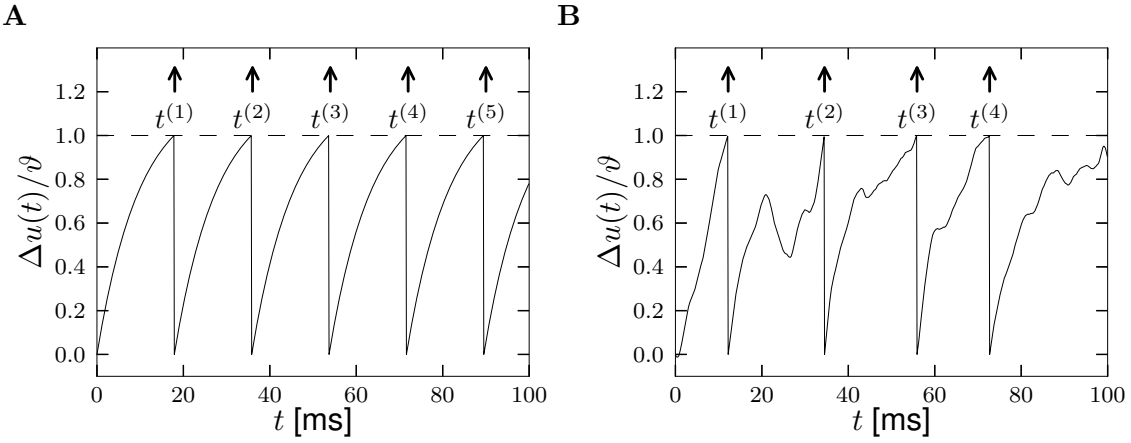


**Fig.** 1.9: Integrate-and-fire model. **A**. Time course of the membrane potential of an integrate-and-fire neuron driven by constant input current $I_0 = 1.5$. The voltage $\Delta u(t) = u - u_{\rm rest}$ is normalized by the value of the threshold $\vartheta$. Units of input current are chosen so that $I_0 = 1$ corresponds to a trajectory that reaches the threshold for $t \to \infty$. After a spike, the potential is reset to $u_r = u_{\rm rest}$. **B**. Voltage response to a time-dependent input current.

time-dependent current, but the solution, Eq. (1.15), is also valid for every other time-dependent input current.

So far our leaky integrator does not have a threshold. What happens to the solution Eq. (1.15), if we add a threshold $\vartheta$? Each time the membrane potential hits the threshold, the variable $u$ is reset from $\vartheta$ to $u_r$. In the electrical circuit diagram, the reset of the potential corresponds to removing a charge $q_r = C\,(\vartheta - u_r)$ from the capacitor (Fig. 1.6) or, equivalently, adding a negative charge $-q_r$ onto the capacitor. Therefore, the reset corresponds to a short current pulse $I_r = -q_r\,\delta(t - t^{(f)})$ at the moment of the firing $t^{(f)}$. Indeed, it is not unusual to say that a neuron 'discharges' instead of 'fires'. Since the reset happens each time the neuron fires, the reset current is

$$I_r = -q_r \sum_f \delta(t - t^{(f)}) = -C\,(\vartheta - u_r) S(t)\,, \tag{1.16}$$

where $S(t)$ denotes the spike train, defined in Eq. (1.14).

The short current pulse corresponding to the 'discharging' is treated mathematically just like any other time-dependent input current. The total current $I(t) + I_r(t)$, consisting of the stimulating current and the reset current, is inserted into the solution (1.15) to give the final result

$$u(t) = u_{\rm rest} + \sum_f (u_r - \vartheta)\,\exp\left(-\frac{t - t^{(f)}}{\tau_m}\right) + \frac{R}{\tau_m}\int_0^\infty \exp\left(-\frac{s}{\tau_m}\right) I(t-s)\,{\rm d}s\,, \tag{1.17}$$

where the firing times $t^{(f)}$ are defined by the threshold condition

$$t^{(f)} = \{t | u(t) = \vartheta\}\ . \tag{1.18}$$

Note that with our definition of the Dirac $\delta$-function in Eq. (1.9), the discharging reset follows immediately after the threshold crossing, so that the natural sequence of events – first firing, then reset – is respected.

Eq. (1.17) looks rather complicated. It has, however, a simple explanation. In Sect. 1.3.2 we have seen that a short input pulse at time $t'$ causes at time $t$ a response of the membrane proportional to $\exp\left(-\frac{t-t'}{\tau_m}\right)$, sometimes called the impulse response function or Green's function; cf. Eq. (1.11). The second term on the right-hand side of Eq. (1.17) is the effect of the discharging current pulses at the moment of the reset.

In order to interpret the last term on the right-hand side, we think of a stimulating current $I(t)$ as consisting of a rapid sequence of discrete and short current pulses. In discrete time, there would be a different current pulse in each time step. Because of the linearity of the differential equation, the effect of all these short current pulses can be added. When we return from discrete time to continuous time, the sum of the impulse response functions turns into the integral on the right-hand side of Eq. (1.17).

### 1.3.5 Linear Differential Equation vs. Linear Filter: Two Equivalent Pictures (*)

The leaky integrate-and-fire model is defined by the differential equation (1.5), i.e.,

$$\tau_m \frac{\mathrm{d}u}{\mathrm{d}t} = -[u(t) - u_{\rm rest}] + R\,I(t)\,, \tag{1.19}$$

combined with the reset condition

$$\lim_{\delta\to 0;\delta>0} u(t^{(f)}+\delta) = u_r\,, \tag{1.20}$$

where $t^{(f)}$ are the firing times

$$t^{(f)} = \{t|u(t)=\vartheta\}\ . \tag{1.21}$$

As we have seen in the previous subsection, the linear equation can be integrated and yields the solution (1.17). It is convenient to rewrite the solution in the form

$$u(t) = \int_0^\infty \eta(s)S(t-s)ds + \int_0^\infty \kappa(s)I(t-s)\,\mathrm{d}s\,. \tag{1.22}$$

where we have introduced filters $\eta(s) = (u_r-\vartheta)\exp\left(-\frac{s}{\tau_m}\right)$ and $\kappa(s) = \frac{1}{C}\exp\left(-\frac{s}{\tau_m}\right)$. Interestingly, Eq. (1.22) is much more general than the leaky integrate-and-fire model, because the filters do not need to be exponentials but could have any arbitrary shape. The filter $\eta$ describes the reset of the membrane potential and, more generally, accounts for neuronal refractoriness. The filter $\kappa$ summarizes the linear electrical properties of the membrane. Eq. (1.22) in combination with the threshold condition (1.21) is the basis of the Spike Response Model and Generalized Linear Models, which will be discussed in Part II.

### 1.3.6 Periodic drive and Fourier transform (*)

Formally, the complex Fourier transform of a real-valued function $f(t)$ with argument $t$ on the real line is

$$\hat{f}(\omega) = \int_{-\infty}^{\infty} f(t)\, e^{-i\omega t} \mathrm{d}t = |\hat{f}(\omega)|\, e^{i\phi_f(\omega)} \tag{1.23}$$

where $|\hat{f}(\omega)|$ and $\phi_f(\omega)$ are called amplitude and phase of the Fourier transform at frequency $\omega$. The mathematical condition for a well-defined Fourier transform is that the function $f$ be Lebesgue integrable with integral $\int_{-\infty}^{\infty} |f(t)| \mathrm{d}t < \infty$. If $f$ is a function of time, then $\hat{f}(\omega)$ is a function of frequency. An inverse Fourier transform leads back from frequency-space to the original space, i.e., time.

For a linear system, the above definition gives rise to several convenient rules for Fourier-transformed equations. For example, let us consider the system

$$u(t) = \int_{-\infty}^{\infty} \kappa(s)\, I(t-s)\, \mathrm{d}s\,, \tag{1.24}$$

where $I(t)$ is a real-valued input (e.g., a current), $u(t)$ the real-valued system output (e.g., a voltage) and $\kappa$ a linear response filter, or kernel, with $\kappa(s) = 0$ for $s < 0$ because of causality. The convolution on the right-hand side of Eq. (1.24) turns after Fourier transformation into a simple multiplication, as shown by the following steps of calculation:

$$\begin{aligned} \hat{u}(\omega) &= \int_{-\infty}^{\infty} \left[ \int_{-\infty}^{\infty} \kappa(s)\, I(t-s)\, \mathrm{d}s \right] e^{-i\omega t} \mathrm{d}t \\ &= \int_{-\infty}^{\infty} \int_{-\infty}^{\infty} \kappa(s) e^{-i\omega s}\, I(t-s)\, e^{-i\omega(t-s)} \mathrm{d}s \mathrm{d}t \\ &= \hat{\kappa}(\omega)\, \hat{I}(\omega) \end{aligned} \tag{1.25}$$

where we introduced in the last step the variable $t' = t - s$ and used the definition (1.23) of the Fourier transform.

Similarly, the derivative $du/dt$ of a function $u(t)$ can be Fourier-transformed using the product rule of integration. The Fourier transform of the derivative of $u(t)$ is $i\omega\hat{u}(\omega)$.

While introduced here as a purely mathematical operation, it is often convenient to visualize the Fourier transform in the context of a physical system driven by a periodic input. Consider the linear system of Eq. (1.24) with an input

$$I(t) = I_0\, e^{i\omega t}\,. \tag{1.26}$$

A short comment on the notation. If the input is a current, it should be real-valued, as opposed to a complex number. We therefore take $I_0$ as a real and positive number and focus on the *real part* of the complex equation (1.26) as our physical input. When we perform a calculation with complex numbers, we therefore implicitly assume that, at the very end, we only take the real part of solution. However, the calculation with complex numbers turns out to be convenient for the steps in between.

Inserting the periodic drive, Eq. (1.26), into Eq. (1.24) yields

$$u(t) = \int_{-\infty}^{\infty} \kappa(s)\, I_0 e^{i\omega(t-s)}\, \mathrm{d}s = \left[ \int_{-\infty}^{\infty} \kappa(s)\, e^{-i\omega s}\, \mathrm{d}s \right] I_0 e^{i\omega t}\,. \tag{1.27}$$

Hence, if the input is periodic at frequency $\omega$ the output is so, too. The term in square brackets is the Fourier transform of the linear filter. We write $u(t) = u_0\, e^{i\phi_\kappa(\omega)}\, e^{i\omega t}$. The ratio between the amplitude of the output and that of the input is

$$\frac{u_0}{I_0} = |\hat{\kappa}(\omega)|\,. \tag{1.28}$$

The phase $\phi_\kappa(\omega)$ of the Fourier-transformed linear filter $\kappa$ corresponds to phase shift between input and output or, to say it differently, a delay $\Delta = \phi_\kappa/\omega = \phi_\kappa\, T/2\pi$ where $T$ is the period of the oscillation. Fourier transforms will play a role in the discussion of signal processing properties of connected networks of neurons in Part III of the book.

**Example: Periodic drive of a passive membrane**

We consider the differential equation of the passive membrane defined in Eq. (1.5) and choose voltage units such that $u_{\rm rest} = 0$, i.e.,

$$\tau_m \frac{\mathrm{d}u}{\mathrm{d}t} = -u(t) + R\,I(t)\,. \tag{1.29}$$

The solution, given by Eq. (1.15), corresponds to the convolution of the input $I(t)$ with a causal linear filter $\kappa(s) = (1/C)\, e^{(-s/\tau_m)}$ for $s > 0$. In order to determine the response amplitude $u_0$ to a periodic drive $I(t) = I_0\, e^{i\omega t}$ we need to calculate the Fourier transform of $\kappa$:

$$|\hat{\kappa}(\omega)| \;=\; \left|\frac{1}{C}\int_0^\infty e^{\frac{-t}{\tau_m}}\, e^{-i\omega t}\,\mathrm{d}t\right| = \frac{1}{C}\left|\frac{\tau_m}{1+i\omega\tau_m}\right|\,. \tag{1.30}$$

For $\omega\tau_m \gg 1$ the right-hand side is proportional to $\omega^{-1}$. Therefore the amplitude of the response to a periodic input decreases at high frequencies.

## 1.4 Limitations of the Leaky Integrate-and-Fire Model

The leaky integrate-and-fire model presented in Section 1.3 is highly simplified and neglects many aspects of neuronal dynamics. In particular, input, which may arise from presynaptic neurons or from current injection, is integrated linearly, independently of the state of the postsynaptic neuron:

$$\tau_m \frac{\mathrm{d}u}{\mathrm{d}t} = -[u(t) - u_{\rm rest}] + R\,I(t) \tag{1.31}$$

where $I(t)$ is the input current. Furthermore, after each output spike the membrane potential is reset,

$$\text{if } u(t) = \vartheta \text{ then } \lim_{\delta\to 0;\delta>0} u(t+\delta) = u_r \tag{1.32}$$

so that no memory of previous spikes is kept. Let us list the major limitations of the simplified model discussed so far. All of these limitations will be addressed in the extension of the leaky integrate-and-fire model presented in Part II of the book.

### 1.4.1 Adaptation, Bursting, and Inhibitory Rebound

To study neuronal dynamics experimentally, neurons can be isolated and stimulated by current injection through an intracellular electrode. In a standard experimental protocol

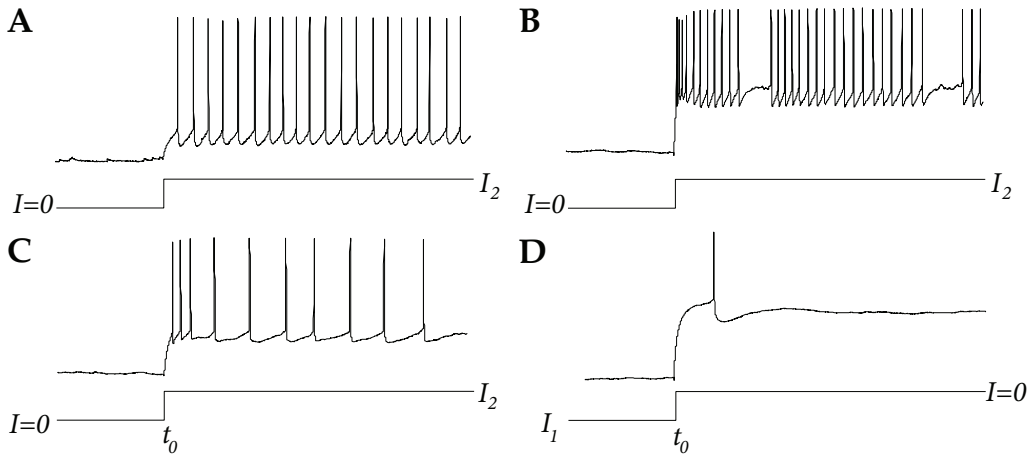


**Fig.** 1.10: Response to a current step. In **A** - **C**, the current is switched on at $t = t_0$ to a value $I_2 > 0$. Fast-spiking neurons (**A**) have short interspike intervals without adaptation while regular-spiking neurons (**C**) exhibit adaptation, visible as an increase in the duration of interspike intervals. An example of a stuttering neuron is shown in **B**. Many neurons emit an inhibitory rebound spike (**D**) after an inhibitory current $I_1 < 0$ is switched off. Data is a courtesy of Henry Markram and Maria Toledo-Rodriguez (Markram et al., 2004; Toledo-Rodriguez et al., 2004).

we could, for example, impose a stimulating current that is switched at time $t_0$ from a value $I_1$ to a new value $I_2$. Let us suppose that $I_1 = 0$ so that the neuron is quiescent for $t < t_0$. If the current $I_2$ is sufficiently large, it will evoke spikes for $t > t_0$. Most neurons will respond to the current step with a spike train where intervals between spikes increase successively until a steady state of periodic firing is reached; cf. Fig. 1.10C. Neurons that show this type of adaptation are called regularly-firing neurons (Connors and Gutnick, 1990). Adaptation is a slow process that builds up over several spikes. Since the standard leaky integrate-and-fire model resets the voltage after each spike to the same value and restarts the integration process, no memory is kept beyond the most recent spike. Therefore, the leaky integrate-and-fire neuron cannot capture adaptation. Detailed neuron models, which will be discussed in Chapter 2, explicitly describe the slow processes that lead to adaptation. To mimic these processes in integrate-and-fire neurons, we need to add up the contributions to refractoriness of several spikes back in the past. As we will see in Chapter 6, this can be done in the 'filter' framework of Eq. (1.22) by using a filter $\eta$ for refractoriness with a time constant much slower than that of the membrane potential. Or by combining the differential equation of the leaky integrate-and-fire model with a second differential equation describing the evolution of a slow variable; cf. Chapter 6.

A second class of neurons are fast-spiking neurons. These neurons show no adaptation (cf. Fig. 1.10A) and can therefore be well approximated by non-adapting integrate-and-fire models. Many inhibitory neurons are fast-spiking neurons. Apart from regular-spiking and fast-spiking neurons, there are also bursting and stuttering neurons which form a separate group (Connors and Gutnick, 1990). These neurons respond to constant stimulation by a sequence of spikes that is periodically (bursting) or aperiodically (stuttering) interrupted by rather long intervals; cf. Fig. 1.10B. Again, a neuron model that has no memory

beyond the most recent spike cannot describe bursting, but the framework in Eq. (1.22) with arbitrary 'filters' is general enough to account for bursting as well.

Another frequently observed behavior is post-inhibitory rebound. Consider a step current with $I_1 < 0$ and $I_2 = 0$, i.e., an inhibitory input that is switched off at time $t_0$; cf. Fig. 1.10D. Many neurons respond to such a change with one or more 'rebound spikes': Even the release of inhibition can trigger action potentials. We will return to inhibitory rebound in Chapter 3.

### 1.4.2 Shunting Inhibition and Reversal Potential

In the previous paragraph we focused on an isolated neuron stimulated by an applied current. In reality, neurons are embedded into a large network and receive input from many other neurons. Suppose a spike from a presynaptic neuron $j$ is sent at time $t_j^{(f)}$ towards the synapse of a postsynaptic neuron $i$. When we introduced in Fig. 1.5 the postsynaptic potential that is generated after the arrival of the spike at the synapse, its shape and amplitude did not depend on the state of the postsynaptic neuron $i$. This is of course a simplification and reality is somewhat more complicated. In Chapter 3 we will discuss detailed neuron models that describe synaptic input as a change of the membrane conductance. Here we simply summarize the major phenomena.

In Fig. 1.11 we have sketched schematically an experiment where the neuron is driven by a constant current $I_0$. We assume that $I_0$ is too weak to evoke firing so that, after some relaxation time, the membrane potential settles at a constant value $u_0$. At $t = t^{(f)}$ one of the presynaptic neurons emits a spike so that shortly afterwards the action potential arrives at the synapse and provides additional stimulation of the postsynaptic neuron. More precisely, the spike generates a current pulse at the postsynaptic neuron (postsynaptic current, PSC) with amplitude

$$\text{PSC} \propto \quad [u_0 - E_{\text{syn}}] \tag{1.33}$$

where $u_0$ is the membrane potential and $E_{\text{syn}}$ is the 'reversal potential' of the synapse. Since the amplitude of the current input depends on $u_0$, the response of the postsynaptic potential does so as well. Reversal potentials are systematically introduced in Chapter 2; models of synaptic input are discussed in Chapter 3.1.

**Example: Shunting inhibition**

The dependence of the postsynaptic response upon the momentary state of the neuron is most pronounced for inhibitory synapses. The reversal potential of inhibitory synapses $E_{\text{syn}}$ is below, but usually close to the resting potential. Input spikes thus have hardly any effect on the membrane potential if the neuron is at rest; cf. Fig. 1.11A. However, if the membrane is depolarized, the very same input spikes evoke a larger inhibitory postsynaptic potential. If the membrane is already hyperpolarized, the input spike can even produce a depolarizing effect. There is an intermediate value $u_0 = E_{\text{syn}}$ – the reversal potential – where the response to inhibitory input 'reverses' from hyperpolarizing to depolarizing.

Though inhibitory input usually has only a small impact on the membrane potential, the local conductivity of the cell membrane can be significantly increased. Inhibitory synapses are often located on the soma or on the shaft of the dendritic tree. Due to their strategic position, a few inhibitory input spikes can 'shunt' the whole input that is gathered by the dendritic tree from hundreds of excitatory synapses. This phenomenon is called 'shunting inhibition'.

The reversal potential for excitatory synapses is usually significantly above the resting

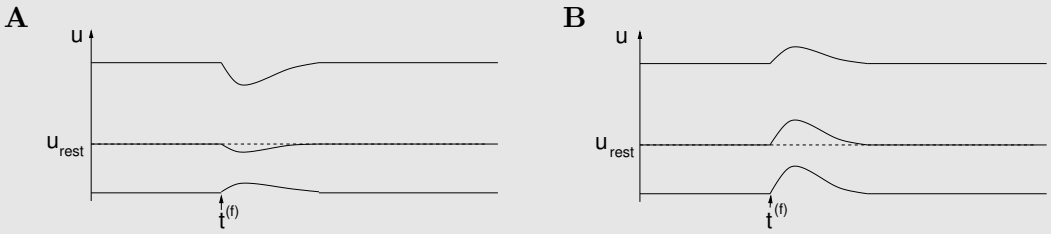


**Fig.** 1.11: The shape of postsynaptic potentials depends on the momentary level of depolarization. **A**. A presynaptic spike that arrives at time $t^{(f)}$ at an inhibitory synapse has hardly any effect on the membrane potential when the neuron is at rest, but a large effect if the membrane potential $u$ is above the resting potential. If the membrane is hyperpolarized below the reversal potential of the inhibitory synapse, the response to the presynaptic input changes sign. **B**. A spike at an excitatory synapse evokes a postsynaptic potential with an amplitude that depends only slightly on the momentary voltage $u$. For large depolarizations the amplitude saturates and becomes smaller. (Schematic figure.)

potential. If the membrane is depolarized $u_0 \gg u_{\rm rest}$ the amplitude of an excitatory postsynaptic potential is reduced, but the effect is not as pronounced as for inhibition. For very high levels of depolarization a saturation of the EPSPs can be observed; cf. 1.11B.

### 1.4.3 Conductance Changes after a Spike

The shape of the postsynaptic potentials does not only depend on the level of depolarization but, more generally, on the internal state of the neuron, e.g., on the timing relative to previous action potentials.

Suppose that an action potential has occurred at time $t_i^{(f)}$ and that a presynaptic spike arrives at a time $t_j^{(f)} > t_i^{(f)}$ at the synapse $j$. The form of the postsynaptic potential depends now on the time $t_j^{(f)} - t_i^{(f)}$; cf. Fig. 1.12. If the presynaptic spike arrives during or shortly after a postsynaptic action potential, it has little effect because some of the ion channels that were involved in firing the action potential are still open. If the input spike arrives much later, it generates a postsynaptic potential of the usual size. We will return to this effect in Chapter 2.

### 1.4.4 Spatial Structure

The form of postsynaptic potentials also depends on the location of the synapse on the dendritic tree. Synapses that are located far away from the soma are expected to evoke a smaller postsynaptic response at the soma than a synapse that is located directly on the soma; cf. Chapter 3. If several inputs occur on the same dendritic branch within a few milliseconds, the first input will cause local changes of the membrane potential that influence the amplitude of the response to the input spikes that arrive slightly later. This may lead to saturation or, in the case of so-called 'active' currents, to an enhancement of the response. Such nonlinear interactions between different presynaptic spikes are neglected in the leaky integrate-and-fire model. Whereas a purely linear dendrite can be incorporated in the 'filter' description of the model, as we will see in Chapter 6, nonlinear interactions cannot. Small regions on the dendrite where a strong nonlinear boosting of synpatic currents occurs are sometimes called dendritic 'hot spots'. The boosting can lead to dendritic spikes which, in contrast to normal somatic action potentials last for tens of

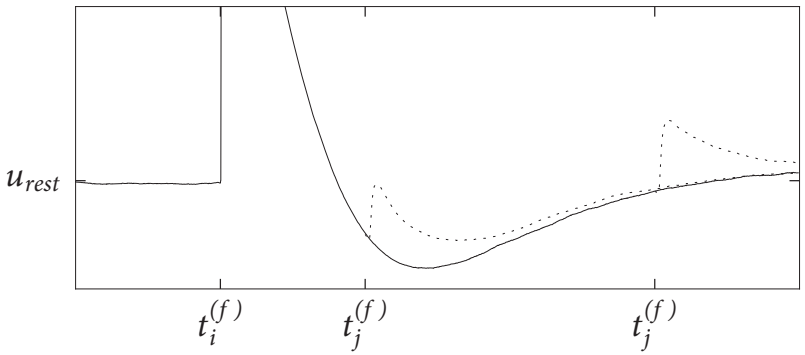


**Fig.** 1.12: The shape of postsynaptic potentials (dashed lines) depends on the time $t-t_i^{(f)}$ that has passed since the last output spike of neuron $i$. The postsynaptic spike has been triggered at time $t_i^{(f)}$. A presynaptic spike that arrives at time $t_j^{(f)}$ shortly after the spike of the postsynaptic neuron has a smaller effect than a spike that arrives much later. Data is a courtesy of Thomas Berger (Berger et al., 2009).

milliseconds (Larkum and Nevian, 2008).

## 1.5 What Can We Expect from Integrate-And-Fire Models?

The leaky integrate-and-fire model is an extremely simplified neuron model. As we have seen in the previous section, it neglects many features that neuroscientists have observed when they study neurons in the living brain or in slices of brain tissue. Therefore the question arises: what should we expect from such a model? Clearly we cannot expect it to explain the complete biochemistry and biophysics of neurons. Nor do we expect it to account for highly nonlinear interactions that are caused by active currents in some 'hot spots' on the dendritic tree. However, the integrate-and-fire model is surprisingly accurate when it comes to generating spikes, i.e., precisely timed events in time. Thus, it could potentially be a valid model of spike generation in neurons, or more precisely, in the soma.

It is reasonable to require from a model of spike generation that it should be able to predict the moments in time when a real neuron spikes. Let us look at the following schematic set-up (Fig. 1.13). An experimentalist injects a time-dependent input-current $I(t)$ into the soma of a cortical neuron using a first electrode. With an independent second electrode he or she measures the *voltage* at the soma of the neuron. Not surprisingly, the voltage trajectory contains from time to time sharp electrical pulses. These are the action potentials or spikes.

A befriended mathematical neuroscientist now takes the time course $I(t)$ of the input current that was used by the experimentalist together with the time course of the membrane potential of the neuron and adjusts the parameters of a leaky integrate-and-fire model so that the model generates, for the very same input current, spikes at roughly the same moments in time as the real neuron. This needs some parameter tuning, but seems feasible. The relevant and much more difficult question, however, is whether the neuron model can now be used to predict the firing times of the real neuron for a novel time-dependent input current that was not used during parameter optimization (Fig. 1.13).

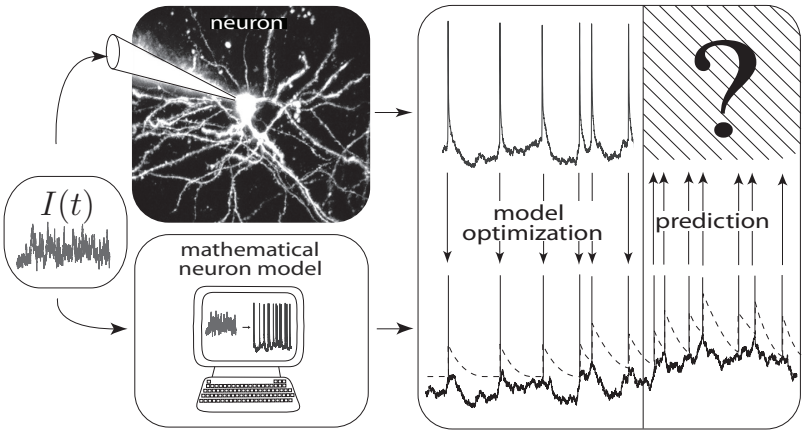


**Fig.** 1.13: The challenge of spike time prediction. A current $I(t)$ is experimentally injected into the soma of a real neuron *in vitro* through an electrode. The response of the neuron is recorded and half of the response is made available for model optimization while part of the response remains hidden. The challenge is then to use the input $I(t)$ to predict the spike times of the hidden response with a mathematical neuron model.

As discussed above, neurons not only show refractoriness after each spike but also exhibit adaptation which builds up over hundreds of milliseconds. A simple leaky integrate-and-fire model does not perform well at predicting the spike times of a real neuron. However, if adaptation (and refractoriness) is added to the neuron model, the prediction works surprisingly well. A straightforward way to add adaptation is to make the firing threshold of the neuron model dynamic: after each spike the threshold $\vartheta$ is increased by an amount $\theta$, while during a quiescent period the threshold approaches its stationary value $\vartheta_0$. We can use the Dirac $\delta$-function to express this idea

$$\tau_{\rm adapt}\frac{\rm d}{{\rm d}t}\vartheta(t) = -[\vartheta(t)-\vartheta_0] + \theta\sum_f \delta(t-t^{(f)}) \qquad (1.34)$$

where $\tau_{\rm adapt}$ is the time constant of adaptation (a few hundred milliseconds) and $t^{(f)} = t^{(1)}, t^{(2)}, t^{(3)}...$ are the firing times of the neuron.

The predictions of an integrate-and-fire model with adaptive threshold agree nicely with the voltage trajectory of a real neuron, as can be seen from Fig. 1.14. The problem of how to construct practical, yet powerful, generalizations of the simple leaky integrate-and-fire model is the main topic of Part II of the book. Another question arising from this is how to quantify the performance of such neuron models (see Chapters 11).

Once we have identified good candidate neuron models, we will ask in Part III, whether we can construct big populations of neurons with these models, and whether we can use them to understand the dynamic and computational principles as well as potential neural codes used by populations of neurons. Indeed, as we will see, it is possible to make the transition from plausible single-neuron models to large and structured populations. This does not mean that we understand the full brain, but understanding the principles of large

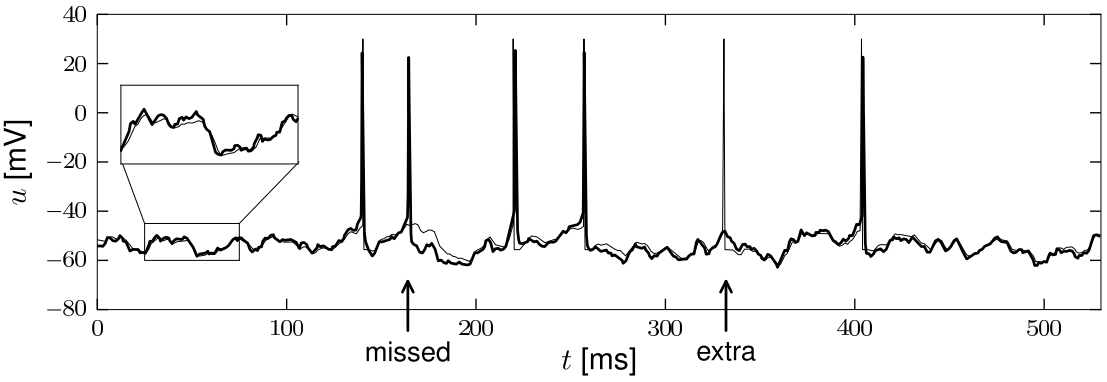


**Fig.** 1.14: Comparing a generalized integrate-and-fire model with experimental traces. A voltage trace (thick black trace) recorded in a real neuron driven by a fluctuating current is superposed on the voltage trace generated by a generalized integrate and fire model (thin line) driven by the same current. The subthreshold voltage fluctuations are accurately predicted (inset) and the spike timings are well predicted on average, apart from a few additional or missed spikes (arrows).

populations of neurons from well-tested simplified neuron models is a first and important step in this direction.

## 1.6 Summary

The neuronal signal consists of short voltage pulses called action potentials or spikes. These pulses travel along the axon and are distributed to several postsynaptic neurons where they evoke postsynaptic potentials. If a postsynaptic neuron receives a sufficient number of spikes from several presynaptic neurons within a short time window, its membrane potential may reach a critical value and an action potential is triggered. We say that the neuron has 'fired' a spike. This spike is the neuronal output signal which is, in turn, transmitted to other neurons.

A particularly simple model of a spiking neuron is the leaky integrate-and-fire model. First, a linear differential equation describes how input currents are integrated and transformed into a membrane voltage $u(t)$. Here the input can be the input current injected by an experimentalist into an isolated neuron or synaptic input currents caused by spikes arriving from other neurons in a large and highly connected network. Second, the model neuron generates an output spike if the membrane voltage reaches the threshold $\vartheta$. Finally, after spike firing, the integration of the linear differential equation resumes from a reset value $u_r$.

The simple leaky integrate-and-fire model does not account for long-lasting refractoriness or adaptation. However, if the voltage dynamics of the leaky integrate-and-fire model is enhanced by mechanisms of adaptation, then it can be a powerful tool to accurately predict spike times of cortical neurons. Such generalized integrate-and-fire models are the main topic of Part II of this book.

## Literature

An elementary, non-technical introduction to neurons and synapses can be found in the book by Thompson (1993). At an intermediate level is the introductory textbook of Purves et al. (Purves et al., 2008) while the "Principles of Neural Science" by Kandel et al. (2000b) can be considered as a standard textbook on neuroscience covering a wealth of experimental results.

The use of mathematics to explain neuronal activity has a long tradition in theoretical neuroscience, over one hundred years. Phenomenological spiking neuron models similar to the leaky integrate-and-fire model have been proposed by Lapique in 1907 who wanted to predict the first spike after stimulus onset (so that his model did not yet have the reset of the membrane potential after firing), and have been developed further in different variants by others (Lapicque, 1907; Hill, 1936; McCulloch and Pitts, 1943; Stein, 1965; Geisler and Goldberg, 1966; Weiss, 1966; Stein, 1967b). For the 'filter' description of integrate-and-fire models see for example Gerstner et al. (1996) and Pillow et al. (1998). The elegance and simplicity of integrate-and-fire models makes them a widely used tool to describe principles of neural information processing in neural networks of a broad range of sizes.

A different line of mathematical neuron models are biophysical models, first developed by Hodgkin and Huxley (Hodgkin and Huxley, 1952); these biophysical models are the topic of the next chapter.

## Exercises

1. **Synaptic current pulse**. *Synaptic inputs can be approximated by an exponential current* $I(t) = q\,\frac{1}{\tau_s}\exp[-\frac{t-t^{(f)}}{\tau_s}]$ *for* $t > t^{(f)}$ *where* $t^{(f)}$ *is the moment when the spike arrives at the synapse.*

   *(a) Use Eq. (1.5) to calculate the response of a passive membrane with time constant* $\tau_m$ *to an input spike arriving at time* $t^{(f)}$.

   *(b) In the solution resulting from (a), take the limit* $\tau_s \to \tau_m$ *and show that in this limit the response is proportional to* $\propto [t - t^{(f)}]\,\exp[-\frac{t-t^{(f)}}{\tau_s}]$. *A function of this form is sometimes called an* $\alpha$*-function.*

   *(c) In the solution resulting from (a), take the limit* $\tau_s \to 0$. *Can you relate your result to the discussion of the Dirac-*$\delta$ *function?*

2. **Time-dependent solution**. *Show that Eq. (1.15) is a solution of the differential equation Eq. (1.5) for time-dependent input* $I(t)$. *To do so, start by changing the variable in the integral from* $s$ *to* $t' = t - s$. *Then take the derivative of Eq. (1.15) and compare the terms to those on both sides of the differential equation.*

3. **Chain of linear equations**. *Suppose that arrival of a spike at time* $t^{(f)}$ *releases neurotransmitter into the synaptic cleft. The amount of available neurotransmitter at time* $t$ *is* $\tau_x\,\frac{dx}{dt} = -x + \delta(t - t^{(f)})$. *The neurotransmitter binds to the postsynaptic membrane and opens channels that enable a synaptic current* $\tau_s\,\frac{dI}{dt} = -I + I_0\,x(t)$. *Finally, the current charges the postsynaptic membrane according to* $\tau_m\,\frac{du}{dt} = -u + R\,I(t)$. *Write the voltage response to a single current pulse as an integral.*

# Part II

# Generalized Integrate-and-Fire Neurons

# Part III

# Networks of Neurons and Population Activity

# Part IV

# Dynamics of Cognition

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# Index