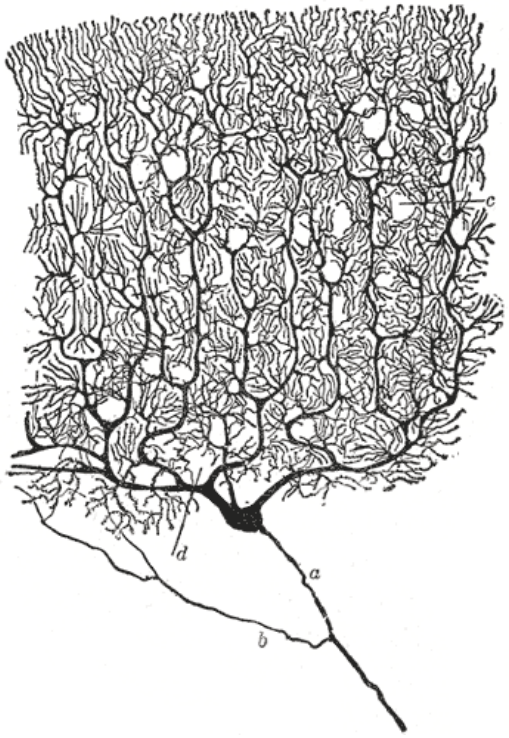

Figure 1: A drawing by Santiago Ramón y Cajal of a Purkinje cell. [Picture taken from `http://en.wikipedia.org/wiki/Golgi's_method`]

Introduction

These notes are about the cerebellum and is partly based on [1]. I offer an error bounty of between 20p and 2 pounds for mistakes. Contact me at `conor.houghton@bristol.ac.uk` or come up after a lecture.

Anatomy of the cerebellum

The cerebellum has a number of striking features; it has a more stereotypical structure than most brain area and this structure is conversed across species. It also has one of the brains largest cells, the Purkinje cell, and its most numerous, the granule cell.

Purkinje cells have a distinctive structure with a huge, highly branched, but flat dendritic arbor, see Fig. 1; this allows an extensive connectivity with each Purkinje cell receiving inputs from around 100,000 other cells. In the cerebellum the Purkinje cell are lined up like pages in a book, with their arbors lying in parallel planes. They receive two excitatory inputs, weak inputs from parallel fibres, axons that run perpendicular to the planes of the Purkinje cell dendritic arbors, and a strong input from a climbing fibre, a single axon which winds around the Purkinje cell and makes multiple contacts with it, see Fig. 2.

Another peculiarity is that the Purkinje cell has different responses to different inputs; in response to multiple weak inputs from the parallel fibers it fires a normal sort of spike, called in this context a *simple spike*; in response to single spike from the climbing fiber is fires a special spike, called a *complex spike*, with a leading spike, a number of small 'spikelets' and a

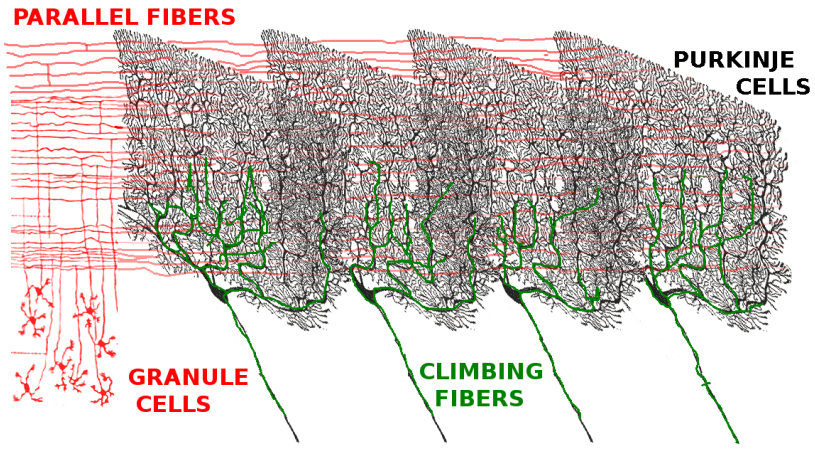

Figure 2: A cartoon of the cerebellar circuitary. A vertical axon rises from each granule cells, splits once and then extends horizontally in two directions making connections with multiple Purkinje cells. Each Purkinje cell has its own climbing fiber which winds up around it.

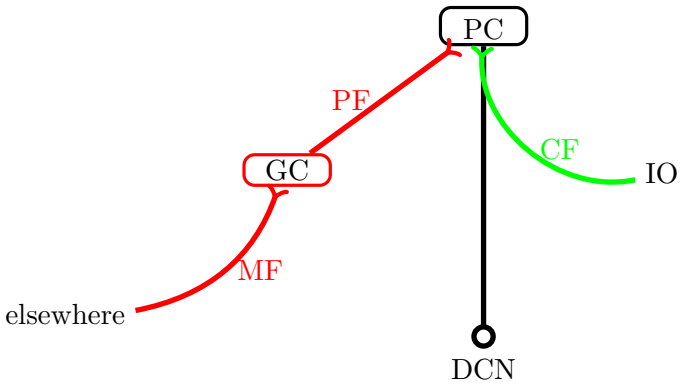

Figure 3: A schematic of the cerebellar circuit. The granule cells (GC) receive input from a diverse range of other parts of the brain along the mossy fibers (MF). Each granule cell will combine input from just three or four mossy fibers and do this in lots of different combinations. The parallel fiber (PF) carries spikes from the GC to the Purkinje cell (PC). The PC also receives input from a climbing fiber (CF) coming from Inferior Olivary Nucleus (IO). In turn it sends an inhibitory signal to the Deep Cerebellar Nucleus (DCN).

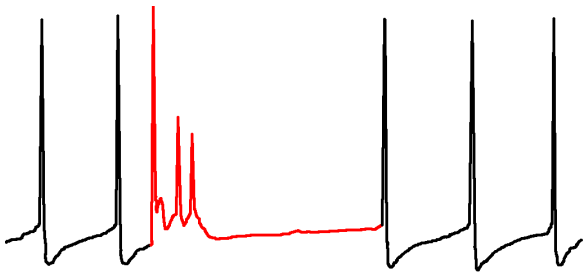

Figure 4: A complex spike. This drawing shows a simple spikes in black and a complex spike in red. The complex spike is followed by a long refractory period during which spiking is not possible. This is a sketch, not an actual recording, but a typical time scale would have this refractory period 50 ms long.

sustained after-period of depolarization; this is illustrated in Fig. 4.

The Marr-Albus model

It is still unclear exactly what the cerebellum does; what is known is that it is important for actions, fine motor control and proprioception; problems with the cerebellum are associated with ataxia, loss of fine motor control, poor motor learning and poor balance. There is a specific gait associated with cerebellar damage, one that exhibits a certain self-consciousness or vigilance is required for movement. According to most ideas about cerebellar function it is required for the calculation of fine motor signals [1], or for predicting the sensory or proprioceptive consequences of motor actions [2].

Whatever exactly it does, it is widely believed, in accordance with the Marr-Albus model [3, 1], that the connections from parallel fibers to Purkinje cells acts as a perceptron. Thus, if y is the output of the Purkinje cell and, in this simple model, taking into account the fact Purkinje cells are inhibitory

$$y = -\sum w_i x_i \tag{1}$$

where the x_is are the activities in the parallel fibers and w_i is the strength of the synapse from the ith climbing fiber to the Purkinje cell. According to the perceptron rule there is a desired output d and the synapses are adjusted according to

$$\Delta w_i = -\eta(d - y)x_i \tag{2}$$

where η is a small learning rate. The idea in the Marr-Albus model is that the climbing fiber carries the error signal $d - y$.

Thus, in a simple example, say $\mathbf{w} = (1, 1, 1, 1)$ initially and the input $\mathbf{x} = (1, 0, 1, 0)$ has the desired output $d = -1$, well

$$y = -\mathbf{w} \cdot \mathbf{x} = -2 \tag{3}$$

so $d - y = 1$ and $\Delta\mathbf{w} = -\eta(1, 0, 1, 0)$, so if $\eta = 0.25$ after learning we would have $\mathbf{w} = (0.75, 1, 0.75, 1)$ so

$$y = -\mathbf{w} \cdot \mathbf{x} = -1.5 \tag{4}$$

and the error has fallen to $d - y = 0.5$. Conversely, imagine $\mathbf{w} = (1, 1, 1, 1)$ but $\mathbf{x} = (1, 1, 0, 0)$ is intended to represent $d = -3$, here

$$y = -\mathbf{w} \cdot \mathbf{x} = -2 \tag{5}$$

so $d - y = -1$ and $\Delta\mathbf{w} = -\eta(-1, -1, 0, 0)$ and after learning with $\eta = 0.25$ we would have $\mathbf{w} = (1.25, 1.25, 1, 1)$. In other words, we need both positive and negative errors with positive errors associated with synaptic depression and negative errors with synaptic potentiation; these have been observed experimentally [4, 5] with climbing fibre activity greater than or less than average corresponding to decreases and increases in synapse strengths.

This can only be part of the description of this network. For example, synapses form the parallel fibers can only be positive, not negative; this can be accounted for by including inhibitory cells in the network. Furthermore, a linear model like that being used here can't learn complicated patterns like the XOR pattern:

x_1	x_2	d
0	0	0
0	1	-1
1	0	-1
1	1	0

Learning a pattern like this requires a network with more than one layer; in fact, this is already provided by the granule cell layer. Each granule cell receives input from between one and seven mossy fibers, there are 3×10^{11} granule cells, roughly 100 to 150 times the number of mossy fibers. Finally there are large inhibitory cells called Golgi cells in the network, these have long time constants, providing delays; this is clearly useful for motor control where different muscles move at different times or different motor consequence unfold at different times during a motion [6, 7].

This leaves lots of things mysterious; how this needs to be changed to account for real spiking neurons, how the error signal adjusts the synapse strengths and why this very special architecture is ideal for this sort of calculation. Another question relates to the error; our brain sees muscles and nerves, our observation sees objects and motion, how are the two reconciled: when you throw a dart you can see you hit the one instead of the triple 20, you can't see which finger muscle was activated too strongly or too weakly. However, with a low learning rate d, or even $d - y$ isn't needed exactly, in fact, just the sign is needed; that is something that could plausibly be deduced in cortex.

References

[1] Albus, JS. (1971) A theory of cerebellar function. Mathematical Biosciences 10: 25–61.

[2] Gao J-H, Parsons LM, Bower JM, Xiong J, Li J and Fox PT (1996) Cerebellum implicated in sensory acquisition and discrimination rather than motor control.

Science 272: 545–7.

[3] Marr D (1969) A theory of cerebellar cortex. Journal of Physiology 202: 437–70.

[4] Ito M, Sakurai M and Tongroach P. (1982) Climbing fibre induced depression of both mossy fibre responsiveness and glutamate sensitivity of cerebellar Purkinje cells. Journal of Physiology, 324: 113–34.

[5] Dean P, Porrill J, Ekerot CF and Jörntell H. (2010). The cerebellar microcircuit as an adaptive filter: experimental and computational evidence. Nature Reviews Neuroscience, 11: 30–43.

[6] Fujita M. (1982) Adaptive filter model of the cerebellum. Biological cybernetics, 45: 195–206.

[7] Shidara M, Kawano K, Gomi H and Kawato M (1993) Inverse-dynamics model eye movement control by Purkinje cells in the cerebellum. Nature, 365: 50–2.