and the rest of the $[0,1)$ interval for the out-of-phase fixed point.

4 Discussion

This brief paper shows how delayed inhibitory interconnections can permit either in-phase and out-of-phase oscillations, depending on the precise connection strengths and delays. We have investigated only the simplest possible system, that of two neurons: a detailed analytical discussion is in (Nischwitz 94). Further work is required to investigate these effects in more complex systems. Multiple stable phase effects in larger systems are described in (Nischwitz et al 92), using varying T, and in (Nischwitz and Glünder 94) for weakly coupled systems. Because the system's synchronization capabilities are limited, such systems with weak inhibitory interconnections may be able to support multiple simultaneous synchronous sets of oscillators, as suggested by (Shastri 89), without falling into a single phase, as found in (Cairns et al 93).

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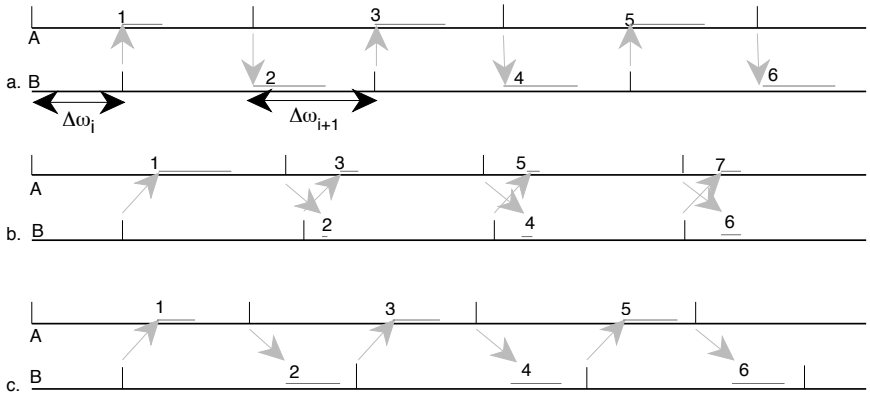

Figure 2: a. Behaviour of 2-unit system with inhibitory connections with 0 delay. The short horizontal lines show the delay caused by the arrival of an inhibitory spike. Each inhibitory spike arrival is numbered. b. Behaviour of a 2-unit system with delay (of $0.2 \times$ period). c. Behaviour of 2 unit system, with the effect of the inhibitory connection halved.

(due to the delay). This causes the next spike at A to be delayed so that its inhibitory effect on B does not occur until after B spikes (2). It therefore has only a small effect. We call this crossover. The effect of the next spike at B on A (3) is also small, since it arrives a little way through A's cycle. Thus both spikes have their effect near the start of the other unit's cycle, making both effects small. This relies on the delay being nonzero, but small relative to the period. If A spikes again before B (as shown) the effect of B on A (3) will be bigger than the previous effect of A on B (2), so that the units become closer in phase. If B spikes again before A, the opposite happens, and the units again become closer in phase. This continues until the units are in phase, at which point the effect of each on the other is identical, and the synchronisation is stable.

Figure 2c shows the effect of a delay, but with a less strong inhibitory connection. In this case, the effect of the first spike at B on A is large (1), but small enough to permit the next spike at A to have its effect at B (2) before B spikes. This spike's inhibitory effect will be larger than the previous effect of B on A, since it arrives nearer the end of the cycle. However, its effect is not so large as to make the inhibitory effect of B's next spike arrive after A spikes (3), and this continues, forcing the units to remain out of phase.

If we assume that the potential is not allowed to become negative, it is possible to derive the basins of attraction of these fixed points (see (Nischwitz 94)). The derivation rests on the strictly decreasing nature of $P(\varepsilon, x)$, and on finding exactly when crossover can occur. For small ε, and large θ/T, so that the initial gradient of -1 lasts for a short time, the basin of attraction of the in-phase fixed point is

$$\omega \in [0, \tau - P(\varepsilon, \omega + \tau)] \cup [1 - (\tau - P(\varepsilon, \omega + \tau)), 1)$$

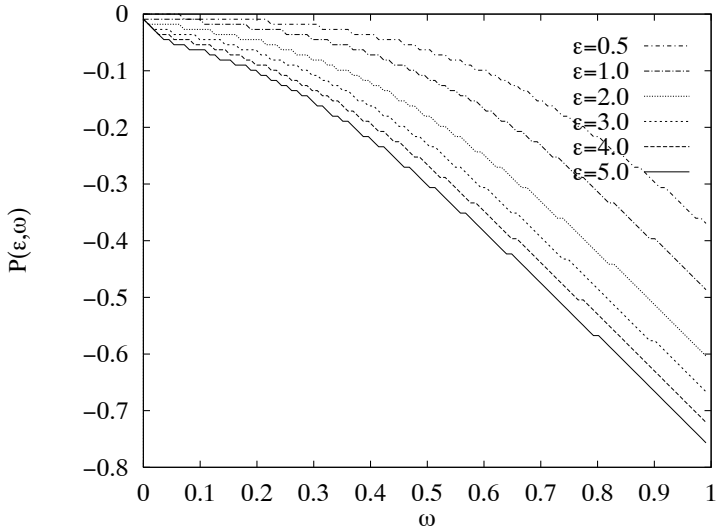

Figure 1: Phase response graph for the neuron, with an inhibitory input of varying strength (ε). ω is the point in the cycle of the arrival of the (inhibitory) input where 0 and 1 are identified with each other, and are the point at which the spike is produced. The Y axis shows the resultant change in phase, $P(\varepsilon, \omega)$. The effect is always to extend the cycle, and the strength of the effect is smallest just after spiking, and largest just before spiking. $T = 20$, $\theta = 19.96$ and $\gamma = 0.95$.

One key to the behaviour of the system is the phase response function (Rinzel and Ermintrout 89), $P(\varepsilon, x)$, the change in phase at one unit caused by an impulse from the other arriving at phase point x. Figure 3 shows this for a single neuron for a number of varying negative coupling strengths, ε. The graph shape depends on whether the unit can have a negative potential: if so then $P(\varepsilon, 0) < 0$, and if not the graph starts with a line of gradient -1 from the origin as shown. $P(\varepsilon, x)$ is strictly decreasing in x on $[0, 1)$, and $P(\varepsilon, x) = P(\varepsilon, x + k)$ for integer k. An informal analysis of the main behaviour is presented: a detailed analysis can be found in (Nischwitz 94) and is the subject of ongoing work.

Figure 2a shows the behaviour of the system with a delay (τ) of 0. If the units start off out of phase, they become antiphase with the period extended due to the inhibition. If B leads A by $\Delta\omega_i$, then the phase difference at A's next spike is

$$\Delta\omega_{i+1} = \Delta\omega_i + P(\varepsilon, \Delta\omega_i) - P(\varepsilon, 1 - \Delta\omega_i - P(\varepsilon, \Delta\omega_i)) \qquad (2)$$

For $\Delta\omega_i < 0.5$, the effect on B is larger than that on A, so that $\Delta\omega_{i+1} > \Delta\omega_i$. As (Mirolla and Strogatz 90) suggest, and is proven in (Nischwitz 94) for units with a refractory period, two fixed points exist, in phase, and antiphase, and the basin of attraction of the antiphase one is all initial $\Delta\omega_i$ except 0.

Figure 2b illustrates one possible result of a delay. The effect of the first spike from B on A (1) is larger, since it arrives nearer the end of the period

Synchronization of Integrate-and-fire Neurons with Delayed Inhibitory Lateral Connections.

Leslie S. Smith†, David E. Cairns†[1], Alfred Nischwitz‡
†CCCN, Department of Computing Science, University of Stirling
Stirling FK9 4LA, Scotland, UK
‡Lehrstuhl für Nachrichtentechnik, Technische Universität München
D–80333 München, Federal Republic of Germany

1 Introduction

Integrate-and-fire (leaky integrator) neurons are both mathematically tractable and have a degree of biological plausibility. Systems of two neurons, interacting via symmetric pulsatile coupling with zero delay and zero absolute refractory period have been studied by (Mirolla and Strogatz 90). For positive coupling, they found two fixed points, an unstable one with the units out of phase, and a stable one with the units in phase. For negative coupling, the stable and unstable fixed points are reversed, if a refractory period is assumed.

We show that for delayed symmetric pulsatile inhibitory coupling, a pair of integrate-and-fire neurons can synchronise. (Glünder and Nischwitz 93) and (Nischwitz et al 92) observed this in simulations of ring-shaped networks of such units. Delayed inhibitory connections have been used by (König and Schillen 91), but using a non-spiking unit, and (Schuster and Wagner 90) use an activation dependent (rather than pulsatile) coupling.

2 The Network

The network examined here consists of two symmetrically connected integrate-and-fire neurons, with equation

$$\frac{dx_i}{dt} = T - \gamma x_i + \varepsilon Y_{|i-1|}(t-\tau) \tag{1}$$

where x_i is the voltage-like state variable of the neuron ($i \in \{0,1\}$), T is the constant tonic input, γ is the dissipation, ε the coupling strength, and τ the coupling delay. On the potential $x_i(t)$ reaching threshold, θ, the output, $Y_i(t)$ becomes instantaneously $\delta(t)$, resulting in a sudden change in potential of the other unit by ε a (short) time τ later. x_i is then reset to 0. The unit will oscillate with period $\frac{\log(T/(T-\theta))}{\gamma}$ for $\theta < T$ and $\varepsilon = 0$.

3 Analysis and Results

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