Signal anticipation and delay in excitable media: group delay of the FitzHugh-Nagumo model

Akke Mats Houben

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Abstract

An expression for the group delay of the FitzHugh-Nagumo model in response to low amplitude input is obtained by linearisation of the cubic term of the voltage equation around its stable fixed-point. It is found that a negative group delay exists for low frequencies, indicating that the evolution of slowly fluctuating signals are anticipated by the voltage dynamics.

The effects of the group delay for different types of signals are shown numerically for the non-linearised FitzHugh-Nagumo model, and some observations on the signal aspects that are anticipated are stated.

1 Introduction

A neuronal spike is a fast transient event, lasting typically around 1 ms, but transmission of spikes between pairs of neurons can take between 1 and 100 ms [1, 9, 40, 39], and the typical timescale of sub-threshold changes in the membrane potential of several neuron models, as fitted to experimental data, is usually set from 10 to 100 ms [10]. Thus we can conclude that there must exists a non-negligible delay between a neuron receiving a stimulation and the resultant action potential arriving at another neuron.

This delay seems at odds with the well-known observations that behavioural or electrophysiological responses (which both seem to involve large numbers of neurons) generally occur in the order of just a few hundred milliseconds [e.g. 12, 13]. It is thus not surprising that rapid signal propagation between neurons and within neuronal networks has received a lot of attention [e.g. 11, 14, 29, 30, 51, 53, 54]. It has been proposed that dynamical systems with certain properties can anticipate their input signals. Some (excitable) dynamical systems, such as neurons [e.g. 8, 32] and neural systems [25], have been shown to anticipate aspects of their inputs through so-called anticipated synchronisation [40], which for excitable systems appears through canard solutions to the system [20]. Alternatively, model neurons with specific properties [47], as well as some electrical systems [e.g. 24, 26, 28] and certain active media [e.g. 4, 3, 7, 13, 34, 36, 37, 48, 50], are able to anticipate the transmission of aspects of specific signals through the existence of a negative group delay or unexpectedly high group velocities.

In this article, I show that the dynamics of a widely used model for excitable media, the FitzHugh-Nagumo model [13, 19, 27], near its stable fixed point $\bar{p}(\bar{v}, \bar{w})$, possesses a frequency band with a negative group delay. Meaning that the dynamics of the model anticipate ‘sub-threshold’ inputs of signals with certain characteristics within this frequency band. A chain of these models then seemingly respond to these types of signals before the first in the chain is good and well stimulated.

In what follows, first an illustration of negative group delay is given (section 2), and an expression for the group delay of the FitzHugh-Nagumo model for low-amplitude input is obtained (section 3). Following (section 4) the effects of the group delay are demonstrated for several types of signals both numerically and conceptually.
2 Negative group delay

Group delay $\tau(\omega)$ is defined as the additive inverse of the derivative of the phase-response $\Delta \tilde{H}(\omega)$ of a system:

$$\tau(\omega) := -\frac{d}{d\omega} \Delta \tilde{H}(\omega),$$

and determines the time-delay of the amplitude envelope of inputs at each frequency $\omega$.

To understand the effect of a group delay, consider a signal $f(t) = g(t)e^{i\omega t}$, consisting of a sinusoid with frequency $\omega_c$, modulated by $g$, relative to the input, low-frequency envelope $g(t)$, being passed through a filter $\tilde{H}(\omega)$.

For simplification assume the filter has a flat unit amplitude response $|\tilde{H}(\omega)| := 1$. The spectrum of $f(t)$ passed through the filter is

$$\tilde{f}(\omega) \tilde{H}(\omega) = \tilde{g}(\omega - \omega_c)e^{i\phi(\omega)}.$$

Since the modulation of the envelope is much slower than that of the sinusoidal signal, the spectrum of $G(\omega)$ is, relative to the signal, narrow around $\omega = 0$. So, the output signal will contain a narrow band of frequencies, around $\omega = \omega_c$. Approximating $\phi(\omega)$ by its Taylor series at $\omega_c$ up to first order, and substituting $\Omega = \omega - \omega_c$ gives for the inverse Fourier transform

$$(f * H)(t) = e^{i(\omega_c t + \phi(\omega_c))} \int_{-\infty}^{\infty} \tilde{g}(\Omega)e^{i\phi'(\omega_c)\Omega}e^{i\Omega t} d\Omega,$$

leading to, by the definition of group delay,

$$(f * H)(t) = g(t) e^{i(\omega_c t + \phi(\omega_c))}.$$  

Thus the envelope $g(t)$ of a signal at frequency $\omega_c$ is shifted by an amount of $\tau(\omega_c)$. Systems where $\tau(\omega)$ is negative for a range of $\omega$ are said to contain a negative group delay, and the output of these systems will anticipate the envelope of signals within this band.

2.1 Group delay for filtered signals

For a broad-band signal $f(t)$ filtered by a narrow-band filter $h_{\omega_c}(t)$ with a spectrum centered at $\omega_c$, we can show that the group-delay instead shifts the complete signal $(h_{\omega_c} * f)(t)$. Passing this signal through the same filter $H(t)$ as before gives

$$(h_{\omega_c} * f) * H)(t) = \int (h_{\omega_c} * f)(\omega)e^{i\phi(\omega)}e^{i\omega t} d\omega.$$

Assuming that $h_{\omega_c}(\omega)$ decays rapidly at both sides of $\omega_c$, we can use the same linear approximation as before, leading to

$$e^{i[\omega_c t + \phi(\omega_c)]} \int (h_{\omega_c} * f)(\Omega + \omega_c)e^{i\phi'(\omega_c)\Omega}e^{i\Omega t} d\Omega,$$

which shows that this results in a time-shift and scaling of the filtered signal:

$$(h_{\omega_c} * f) * H)(t) = e^{i\phi(\omega_c)}(h_{\omega_c} * f)(t - \tau(\omega_c)).$$

So for signals filtered narrowly around a frequency $\omega_c$ it is the complete signal that is shifted by an amount $\tau(\omega_c)$.

3 ... in the FitzHugh-Nagumo model

The FitzHugh-Nagumo model [13, 27] describes the dynamics of the membrane potential $v$ of a neuron alongside a recovery variable $w$:

$$\begin{align*}
\frac{dv}{dt} &= v - \frac{v^3}{3} - w + I \\
\frac{dw}{dt} &= a(v + b - cw),
\end{align*}$$

with three parameters: $a$, determining the timescale of the dynamics of $w$; an offset $b$; and $c$ which influences the slope of the $w$-nullcline and decay rate of $w$.

The fixed point $\bar{v}(\bar{w})$ of (1) can be easily found by solving the cubic equation $\bar{v}^3 + (1/c-1)\bar{v}+b/c = I$ and plugging the resulting value for $\bar{v}$ into the equation $\bar{w} = (\bar{v}+b)/c$. For low-amplitude input $I$, if the fixed-point is stable we can approximate the cubic term $v^3$ by its Taylor series at $\bar{v}$, allowing the equation for the voltage evolution of $\bar{v}$ to be approximated by that
of an approximated membrane potential \( x = \bar{v} + \epsilon \), with governing equation

\[
\frac{dx}{dt} = x - \frac{\bar{v}^3}{3} - \bar{v}^2(x - \bar{v}) - w + I + O(\epsilon^2)
\]

which is a linear first order differential equation. It is then straightforward to obtain the spectrum of the dynamics of \( x \):

\[
\tilde{x}(\omega) = \frac{\tilde{I}(\omega) + DC(\omega)}{\bar{v}^2 - 1 + i\bar{v} + \frac{a}{ac+i\omega}},
\]

with direct term \( DC = |ab/(ac + i\bar{v}) + (1 - 1/3)\bar{v}^3|\delta(\omega) \). If we ignore the DC term, dividing \( \bar{x} \) by \( \tilde{I} \) leads to the transfer function \( \tilde{H}(\omega) \) of \( v \) close to \( \bar{v} \).

Figures 1a and 1b show, respectively, the magnitude- and phase-transfer functions of the linearised variable (solid lines) and the spectrum of the original non-linear equation obtained numerically (dashed lines) for different parameters.

The phase-spectrum is the argument of the transfer-function

\[
\angle \tilde{H}(\omega) = \tan^{-1} \left( \frac{a\omega/(a^2\epsilon^2 + \omega^2) - \omega}{\bar{v}^2 - 1 + a^2\epsilon/(a^2\epsilon^2 + \omega^2)} \right),
\]

whose additive inverse differentiated with respect to \( \omega \) gives the group delay

\[
\tau(\omega) = -\frac{d}{d\omega} \tan^{-1} \left( \frac{a\omega/(a^2\epsilon^2 + \omega^2) - \omega}{\bar{v}^2 - 1 + a^2\epsilon/(a^2\epsilon^2 + \omega^2)} \right)
= -\frac{a^3\epsilon - 2a((\bar{v}^2 - 1) + ac)\omega}{(a^2\epsilon^2 + \omega^2)^2} + \frac{a(\bar{v}^2 - 1) - a^2\epsilon}{a^2\epsilon^2 + \omega^2} - (\bar{v}^2 - 1)
\left[ \frac{\omega a/(a^2\epsilon^2 + \omega^2) - \omega}{\omega} \right] + ((\bar{v}^2 - 1) + a^2\epsilon/(a^2\epsilon^2 + \omega^2)) \right)^2
\]

In order for (4) to be negative we need

\[
A\omega^4 + B\omega^2 + C < 0,
\]

where

\[
A := (\bar{v}^2 - 1),
B := (\bar{v}^2 - 1)(2a^2\epsilon^2 + a) + 3a^2\epsilon,
C := (\bar{v}^2 - 1)(a^4\epsilon^2 - a^2\epsilon^2) + a^4\epsilon^2 - \bar{v}^2\epsilon.
\]

The l.h.s. of (5) has a real positive root only if

\[
B^2 - 4AC \geq 0
\]

given by

\[
\omega_0 = \sqrt{\frac{\sqrt{B^2 - 4AC} - B}{2A}}.
\]

Using the slope of (6) at this zero-crossing

\[
\frac{d}{d\omega} A\omega^4 + B\omega^2 + C \bigg|_{\omega = \omega_0} = 4A\omega_0^3 + 2B\omega_0,
\]

noting that \( 0 < \omega_0^2 < a \) and that for stable fixed-points \( v^2 - 1 + ac > 0 \), we obtain the result that (2) has a frequency band with negative group delay for \( \omega < \omega_0 \).

The group delay is maximally negative for \( \omega = 0 \) and increases monotonically to \( \omega = \omega_0 \), meaning that the envelope of signals with frequencies \( 0 < \omega < \omega_0 \) are transmitted with a negative delay. For high frequencies (4) tends to the limit

\[
\lim_{\omega \to \infty} \tau(\omega) = 0^+,
\]

thus high frequencies are transmitted without significant delay or anticipation. In fact, for the canonical case in which the timescales of \( v \) and \( w \) are sufficiently separated, \( a \ll 1 \), the group delay approaches this limit fast for \( \omega > 1 \). The group delay is then maximal for \( \omega_0 < \omega < 1 \). Figure 2 shows the group delay function \( \tau(\omega) \) for some different parameters.
4 Anticipation of signals

Group delay affects the envelope of band-limited signals, so the types of signals for which the group delay of the membrane potential will have an effect are the modulations of ‘constant’ band-limited signals. The linear approximation of $\phi(\omega)$ at $\omega = \omega_c$ during the illustration of the group delay implies, as stated before, that the frequency spectrum of the modulation needs to have a low-pass characteristic, so that the spectrum of the modulation is narrow around $\omega = 0$.

In the following the anticipation of different types of signals by the voltage variable of the non-linearised model neuron are shown numerically. In order to differentiate between the effects of the phase delay $-\phi(\omega)/\omega$ and the group delay the simulations are carried out with a chain of 17 model neurons, thus leading to a total expected group delay of $17\tau(\omega)$. In each run the first in the chain of neurons receives an input $I_0(t)$, and the subsequent neurons receive an input directly from the membrane potential of each previous neuron

$$I_i(t) = \eta[v_{i-1}(t) - v_0], \text{ for } i = 1, 2, 3, \ldots$$

with coupling strength $\eta$. For each simulation all neurons have parameter values $a = 0.08$, $b = 0.7$ and $c = 0.8$, leading to a negative group delay for frequencies below $\omega_0/(2\pi) = \nu_0 \approx 15.14$Hz (c.f. fig.2), and $\eta = 0.95|H(\omega)|^{-1}$.

Since $\tau(\omega)$ is not flat for $\omega < 1$, considerable frequency smearing is expected even for narrow-band signals in this range. Therefore, in the following numerical examples it is not expected that the magnitude of the observed time-shift corresponds absolutely to the theoretical prediction but, as will be shown, the time-shifts correspond qualitatively to the shape of $\tau(\omega)$ and in most cases agree well on the magnitude of the time-shift as well.

4.1 Wave pulses

The classical way to show the effects of group delay is to use wave pulses: sinusoidal signals of different frequencies modulated by a windowing function. These signals would correspond to short oscillatory bursts, which could indicate or establish transient coordination of the activity of otherwise independent elements [21, 38, 49], which is proposed to underly processes in the brain [e.g. 6, 16, 45]. In a more general sense the signals of this type are amplitude modulated signals, in which the amplitude of a (high frequency) carrier signal is modulated by a lower frequency signal which is to be transmitted.

The windowing function used in the following is a Gaussian pulse

$$g(t) = e^{-\alpha t^2}$$

of width $\sigma$, which has a spectrum of the form

$$\tilde{g}(\omega) = \sqrt{\frac{\pi}{\alpha}} e^{-\frac{\omega^2}{2\alpha}},$$

which is centered at $\omega = 0$ and whose magnitude $|\tilde{g}(\omega)|$ has a fast roll off depending on $\alpha$. Thus for wide enough Gaussian pulses, leading to small values for $\alpha$, the linear approximation of the phase-spectrum should hold.
The top panels of Fig.3 show the input wave pulses with carrier frequencies of \( \nu_c = \nu_0/2 \approx 7.57 \text{Hz} \) (left-panel) and \( \nu_c = 2\nu_0 \approx 30.28 \text{Hz} \) (right-panel), falling, respectively, into the negative group delay band and a band near the maximal group delay of the membrane potential (c.f. Fig.2). The middle panels show the membrane potential of the last neuron in a chain of 17 cascaded model neurons, in which the first neuron received the wave pulse as in the first panel

\[
I_0(t) = e^{-\alpha(t-t_0)^2} A \sin(\omega_c t)
\]

at time \( t = t_0 \) with carrier frequency \( \omega_c \) and amplitude \( A = 1 \times 10^{-2} \).

The bottom panels of Fig.3 show the amplitude envelope of the input signal (solid lines) and that of the last neuron (dashed lines), measured as the output of a strong lowpass filter, for the two different signals.

In the membrane potential of the last neuron, a clear shift forward in time of the signal envelope is visible for signals in the negative group delay band (bottom left panel), in agreement with the group delay predicted by (4), whereas the envelope of the signal in the positive group delay band is delayed (bottom right panel).

The time-shift versus pulse-carrier frequency is shown by Fig.4 which shows the time lag \( \tau(\omega_c) \) for Gaussian wave pulses. Numerical (connected dots) alongside theoretical (solid black line) group delay pulse with carrier frequency \( \omega_c \) (x-axis), with the membrane potential of the last of the chain of 17 neurons. It is visible that the numerical results (connected dots) agree well with the expected group delay \( \tau(\omega_c) \) (solid black line).

4.2 Filtered noise

Pure sinusoidal signals, even though they allow simple analyses, are rare. More commonly, biological and physical signals are considered to be noisy [e.g. 3, 12, 22, 33, 44]. In the following it is shown, as anticipated, that the membrane potential of the model neuron also anticipates the fluctuations of band-limited white noise input.

An ideal zero-mean Gaussian white noise \( \xi(t) \) with variance \( \sigma^2 \) has a flat, wide-band, power spectral density \( |\xi(\omega)|^2 = \int \sigma^2 \delta(t) e^{-\omega^2 t^2} \, dt = \sigma^2 \). In the following \( \xi(t) \) is a normal Gaussian noise, thus \( \sigma^2 = 1 \). This signal will be filtered by a Gaussian filter centered at a frequency \( \omega_c \) and a narrow band-width determined by \( 0 < \alpha \ll 1 \). Thus the first neuron receives an
input with spectrum
\[
\hat{I}_0(\omega) = A_0 e^{-\frac{(\omega - \omega_0)^2}{4\alpha}} \xi(\omega).
\]
Such narrow band filtering of sub-threshold inputs can arise due to signal transmission with heterogeneous transmission delays between neurons [18].

The left column of figure 5 shows the results of the chain of model neurons being driven by a band-limited noise with center-frequency \( \nu_c = \nu_0/2 \approx 7.57 \text{ Hz} \), and \( \alpha = 1 \times 10^{-4} \) falling within the negative group delay band. The top panels show a section of one input signal (top most panel) together with the membrane potential of the last neuron in the chain (second to top panel) in response to that signal. The bottom most panel shows the envelope of the input (solid line) and that of the membrane potential of the last neuron (dashed line). The right panel shows the same, but for an input with \( \nu_c = 2\nu_0 \approx 30.28 \text{ Hz} \).

Figure 5: Anticipation and delay of band-limited noise pulses with pass-band within a negative and positive group delay band, respectively

Figure 6: Peak correlation time versus \( \omega_c \) for filtered noise input. Numerical results (connected symbols) for different filtering bandwidths, alongside theoretical group delay (solid black line)

4.3 Modulated & filtered noise

In the foregoing section the noise considered was stationary, however it is likely that in biological and physical settings the noise sources evolve over time. In addition, modulations of stationary noise sources can serve as a form of signalling through a medium. Such as mean- and variance-modulation as proposed 'communication channels' for neurons and neural networks [e.g. 17, 23].

Considering a noise signal subjected to slow mean- and variance-modulation, \( g_\mu(t) \) and \( g_\sigma(t) \),
\[
f(t) = g_\mu(t) + g_\sigma(t) \xi(t),
\]
with either the result \( f(t) \) or the input noise \( \xi(t) \) being passed through a band-pass filter as before. Clearly in each case the noise signal will be affected by the group delay, if the pass-band falls within a frequency range exhibiting a group delay. It is also apparent that in both cases the mean-modulation \( g_\mu(t) \) will not be affected by the group delay, and thus will be passed without delay or advance.
The variance-modulation however will be affected, but only in case of the noise signal being band-limited before the variance modulation, which is easily understood from the fact that the power of $g_\sigma(t)$ is focussed outside of the pass band of the band-limited filter and thus will only serve as an amplitude modulation to the higher frequency noise signal.

5 Conclusion

In this paper it is shown that the dynamics of the voltage variable of the FitzHugh-Nagumo model possesses a negative group delay for slowly fluctuating inputs, by finding an expression for the group delay of a linear approximation of the governing equation for the voltage variable at the fixed point of the model system.

The effects of the group delay are demonstrated numerically for different types of signals and signal modulations and it is shown that a chain of unidirectionally coupled model neurons anticipates certain aspects of inputs if the input consists of a carrier signal with a frequency falling within the negative group delay band.

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