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Jansen-Rit Model

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Synonyms

Jansen model; Jansen-Zetterberg model

Definition

The Jansen-Rit model is a neural population model of a local cortical circuit. It contains three interconnected neural populations: one for the pyramidal projection neurons and two for excitatory and inhibitory interneurons forming feedback loops. Each neural population is described by a second-order differential operator transforming the mean incoming spike rate to the mean membrane potential and a nonlinear function transforming the mean membrane potential to a mean output spike rate.

Detailed Description

The Jansen-Rit model is a neural population model comprising three interconnected neural populations (see Fig. 1). It has been first described by Jansen and colleagues (Jansen et al. 1993; Jansen and Rit 1995) and goes back to earlier work by Lopez da Silva and colleagues (1974, 1976).

Mathematical Description

The state variables of the model comprise the mean membrane potentials $V(t)$ and the mean firing rates $m(t)$ of the populations. For each population a rate-to-potential operator (RPO) describes the dynamics of the synapses and dendritic trees, while the potential-to-rate operator (PRO) describes the output nonlinearity at the axonal hillocks. The RPO can be described as an alpha function, which is a Green's function of a second-order differential equation, where τ is a time constant summarizing passive dendritic cable delays and neurotransmitter kinetics in the synapse, H is the synaptic gain, and c is the connection strength:

$$\left(\frac{\partial^2}{\partial t^2} + \frac{2}{\tau}\frac{\partial}{\partial t} + \frac{1}{\tau^2}\right) V(t) = \frac{Hc}{\tau} m(t). \tag{1}$$

This is equivalent to convolving the incoming spike rate with the alpha function, which has been shown to adequately describe the postsynaptic potential in response to a single spike, e.g., by Freeman (1975). The PRO is a sigmoid function, where V_0 is the firing threshold, r is the slope

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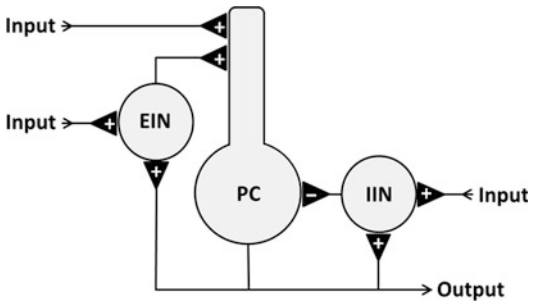

Fig. 1 Schematic drawing of the Jansen-Rit model of a cortical area. Pyramidal cells (*PC*) send axons to other areas (*output*). Collaterals contact excitatory and inhibitory interneurons (*EIN*, *IIN*), which in turn form feedback loops onto the PC. Excitatory synapses from other brain areas (*input*) contact all three neural populations. *Black triangles* symbolize excitatory (marked '+') or inhibitory (marked '−') synaptic contacts

reflecting the variance of firing thresholds within the population, and e_0 is the firing rate at threshold:

$$m(t) = \frac{2e_0}{1 + e^{r(V_0 - V(t))}}. \tag{2}$$

This function can be seen as an approximation of the superposition of many Heaviside functions belonging to individual neurons with normally distributed firing thresholds (Liley et al. 2002).

Full Set of Equation

As shown in Fig. 1, the Jansen-Rit model comprises seven synaptic contacts, each of which can be described by Eqs. 1 and 2. In the original parameterization of Jansen et al. (1993), the PRO parameters are kept constant for all populations ($e_0 = 2.5\ \mathrm{s}^{-1}$; $r = 558.7\ \mathrm{V}^{-1}$; $V_0 = 6$ mV), and the remaining parameters are only distinguished between excitatory and inhibitory synapses ($\tau_e = 10$ ms; $\tau_i = 20$ ms; $H_e = 3.25$ mV; $H_i = -22$ mV). Equations for synapses with the same postsynaptic and the same type of presynaptic (excitatory or inhibitory) populations can be combined, which reduces the number of equations to 4. The following definitions normalize the variables and combine some of the parameters in a convenient way (Spiegler 2011):

Time: $\kappa = t/\tau_c$ with τ_c being a conveniently chosen reference time constant.
Time constants: $\beta_e = \tau_c/\tau_e$ and $\beta_i = \tau_c/\tau_i$.
Potential: $x(\kappa) = r \cdot V(\kappa\tau_c)$.
Firing rate: $z(\kappa) = m(\kappa\tau_c)/(2e_0)$.
Coupling a→b: $\alpha_{ba} = 2e_0 r c_{ba} H_{e,i}\ \tau_{e,i}$ with c_{ba} being the connectivity from population a to population b. Population indices a and b can be 1 for EINs, 2 for IINs, 3 for PCs, and T for external input.
Sigmoid: $\gamma = \exp(rV_0)$.

With these definitions, the differential operator according to Eq. 1 can be reformulated for excitatory and inhibitory synapses as follows:

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$$L_{e,i} = \left(\frac{\partial^2}{\partial \kappa^2} + 2\beta_{e,i}\frac{\partial}{\partial \kappa} + \beta_{e,i}^2\right). \tag{3}$$

The PRO according to Eq. 2 is described by the following sigmoid function:

$$S(x(\kappa)) = \frac{1}{1+\gamma e^{-x(\kappa)}}. \tag{4}$$

The state variables are now x_{1e}, x_{2e}, x_{3e} (normalized excitatory postsynaptic potentials for all three populations), and x_{3i} (normalized inhibitory postsynaptic potential in the PCs). The normalized external input firing rates are z_{1T}, z_{2T}, z_{3T}. The state equations can now be written as:

$$L_e x_{1e}(\kappa) = \beta_e^2(\alpha_{13}S(x_{3e}(\kappa) + x_{3i}(\kappa)) + \alpha_{1T}z_{1T}(\kappa)), \tag{5}$$

$$L_e x_{2e}(\kappa) = \beta_e^2(\alpha_{23}S(x_{3e}(\kappa) + x_{3i}(\kappa)) + \alpha_{2T}z_{2T}(\kappa)), \tag{6}$$

$$L_e x_{3e}(\kappa) = \beta_e^2(\alpha_{31}S(x_{1e}(\kappa)) + \alpha_{3T}z_{3T}(\kappa)), \tag{7}$$

$$L_i x_{3i}(\kappa) = \beta_i^2(\alpha_{32}S(x_{2e}(\kappa))). \tag{8}$$

Dynamic Properties

In spite of its apparent simplicity, the Jansen-Rit model is capable of producing a surprisingly rich repertoire of dynamic behaviors, which populate the parameter space in a very complex way. When the neural populations are exposed to constant external input, thus keeping the system in its equilibrium state, the model can produce constant output, harmonic oscillations, and large-amplitude nonharmonic oscillations. The transitions between these phases are characterized by subcritical and supercritical Andronov-Hopf bifurcations, Shil'nikov's homoclinic bifurcations, and further global bifurcations. Bistability occurs in large parts of the parameter space. While Grimbert and Faugeras (2006) investigated the dynamics of the original parameter configuration proposed by Jansen and Rit, Spiegler and colleagues (2010) systematically investigated the large parts of the parameter space of the model. When the input to the neural populations is oscillatory, the system also produces complex nonharmonic oscillations as well as quasiperiodic and chaotic behavior, depending in a complex fashion on the amplitude and frequency of the input (Spiegler et al. 2011).

Applications

The Jansen-Rit model has been demonstrated to explain a broad range of phenomena in electric brain activity, including epilepsy-like activity (Wendling et al 2000; Touboul et al. 2011), narrowband brain oscillations (David and Friston 2003), event-related activity (David et al. 2006), and steady-state responses (Moran et al. 2009). Within the framework of dynamic causal modeling (David et al. 2006), networks of Jansen-Rit models were used to account for the outcomes of neuroscientific experiments (e.g., Garrido et al. 2009).

Advantages and Limitations

The Jansen-Rit model is a very parsimonious model of cortical mean-field activity, which retains a considerable degree of biological realism, as it accounts for the major functional roles of neurons:

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PCs project to distant areas and, due to their asymmetric appearance and aligned arrangement, form the major source of extracranially detectable electric brain signals; interneurons form local positive and negative feedback loops. The description of the populations by RPO and PRO is also in principal agreement with cell physiological observations. On the other hand, the circuit represents a significant simplification: in reality many more distinguishable populations are interconnected in a complex fashion. Therefore, it is difficult to map the parameters of the model directly onto the physical properties of neural populations.

Cross-References

- ▶ Amari Model
- ▶ Bifurcations, Neural Population Models and
- ▶ Chaos, Neural Population Models and
- ▶ Down Under Neural Population Models
- ▶ Dynamic Causal Modelling with Neural Population Models
- ▶ Epilepsy, Neural Population Models of
- ▶ Neural Population Models
- ▶ Wilson-Cowan Model

References

David O, Friston KJ (2003) A neural mass model for MEG/EEG: coupling and neuronal dynamics. Neuroimage 20:1743–1755

David O, Kiebel SJ, Harrison LM, Mattout J, Kilner JM et al (2006) Dynamic causal modeling of evoked responses in EEG and MEG. Neuroimage 30:1255–1272

Freeman WJ (1975) Mass action in the nervous system: examination of the neurophysiological basis of adaptive behavior through the EEG. Academic, New York

Garrido MI, Kilner JM, Kiebel SJ, Friston KJ (2009) Dynamic causal modeling of the response to frequency deviants. J Neurophysiol 101:2620–2631

Grimbert F, Faugeras O (2006) Bifurcation analysis of Jansen's neural mass model. Neural Comput 18:3052–3068

Jansen BH, Rit VG (1995) Electroencephalogram and visual evoked potential generation in a mathematical model of coupled columns. Biol Cybern 73:357–366

Jansen BH, Zouridakis G, Brandt ME (1993) A neurophysiologically-based mathematical model of flash visual evoked potentials. Biol Cybern 68:275–283

Liley DTJ, Cadusch PJ, Dafilis MP (2002) A spatially continuous mean field theory of electrocortical activity. Netw Comput Neural Syst 13:67–113

Lopes da Silva FH, Hoeks A, Smits H, Zetterberg LH (1974) Model of brain rhythmic activity. The alpha-rhythm of the thalamus. Kybernetik 15:27–37

Lopes da Silva FH, van Rotterdam A, Barts P, van Heusden E, Burr W (1976) Models of neuronal populations: the basic mechanisms of rhythmicity. Prog Brain Res 45:281–308

Moran RJ, Stephan KE, Seidenbecher T, Pape HC, Dolan RJ, Friston KJ (2009) Dynamic causal models of steady-state responses. Neuroimage 44(3):796–811

Spiegler A (2011) Dynamics of biologically informed neural mass models of the brain. PhD Thesis, TU Ilmenau, Germany

DOI 10.1007/978-1-4614-7320-6_65-1

Spiegler A, Kiebel SJ, Atay FM, Knösche TR (2010) Bifurcation analysis of neural mass models: impact of extrinsic inputs and dendritic time constants. Neuroimage 52:1041–1058

Spiegler A, Knösche TR, Schwab K, Haueisen J, Atay FM (2011) Modeling brain resonance phenomena using a neural mass model. PLoS Comput Biol 7:e1002298

Touboul J, Wendling F, Chauvel P, Faugeras O (2011) Neural mass activity, bifurcations, and epilepsy. Neural Comput 23:3232–3286

Wendling F, Bellanger JJ, Bartolomei F, Chauvel P (2000) Relevance of nonlinear lumped-parameter models in the analysis of depth-EEG epileptic signals. Biol Cybern 83:367–378