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The Emergence of Consciousness when REM goes low: Model Constructs and Implications

Introduction

During World War II, in Dublin, Erwin Schrödinger held a series of lectures with the theme title: *What is Life?* Some of the ideas he presented were seminal in the search for the physical/chemical substrate that is evolutionary memory. Schrödinger used his aperiodic crystal model to point out other features that characterize living systems. Another one of these, Schrödinger was the first to point out, is living systems' ability act negentropically (Schrödinger, 1943). Death, therefore, is when the system conforms to the 2nd Law of Thermodynamics in its classical formulation (Boltzmann, 1867).

In 1993, a symposium was held in Dublin to commemorate Schrödinger's series of lectures. One of the invited speakers was Stephen Jay Gould who, with his notorious disdain for physicists (and those who approach biological problems with a mathematical toolkit) began his talk with an embedded quote:

The obvious may be devilishly difficult to define — as best exemplified by Louis Armstrong's famous retort to a naively passionate fan's request for a definition of jazz: "Man, if ya gotta ask you'll never know."

Gould's implication was clear: As a biologist, you need not ask *What is Life?* — you simply *know* what life is!

If discussants in the Vienna Conferences on Consciousness were to address the question "What is Consciousness?", could the Armstrong quote then be transferred verbatim?

It is certainly true that you are alive if you ask yourself: "*What is Life?*".

It is certainly true that you are conscious when you ask yourself: "*What is Consciousness?*".

Note that in the second statement “if” has been replaced by “when” — you can — and do — lose consciousness and regain it many times throughout your life.

While we are alive (no pun intended!), we exist in three phases:

- the state of nonREM sleep
- the state of REM sleep (or: unconscious wakefulness)
- the state of consciousness (or: conscious wakefulness)

During our life, we move in and out of these three states, albeit with a pattern that I will use as a starting point for my deliberations concerning consciousness, or more specifically, the state of conscious wakefulness. During what we call “sleep” in everyday speech we alternate between nonREM and REM sleep. Only after a few bouts of REM sleep do we wake up — i.e., we arrive at conscious wakefulness. I address the topic of consciousness in the context of “waking up”: the transition from unconscious wakefulness to conscious wakefulness.

The McCarley-Hobson model is based on physiological observations of the activity of two populations of neurons in the *pons*: the aminergic population enables nonREM sleep, the cholinergic population, situated in the laterodorsal and pedunculo-pontine tegmented nuclei overcomes nonREM sleep and enables REM “sleep”. The two populations are antagonistic and behave analogous to a predator-prey model. The REM-generating neurons decrease activity as nonREM neurons increase theirs. nonREM neurons thus behave like predators, living off the population of ‘REM neurons’. When the nonREM neuronal activity increases above a certain level, we fall into nonREM sleep. As the nonREM neurons proliferate, they over-activate the system and the number of REM-active neurons collapses. The nonREM cells then have “nothing to feed on” and their numbers decrease rapidly, resulting in a resurgence in a REM-active neuron population — we are then in the state of unconscious wakefulness. In the McCarley-Hobson model, the REM neurons thus behave like prey on which the ‘nonREM neurons’ feed.

This description is intriguing and apparently convincing. As many other convincing descriptive theories in biology, their validity is drawn into question whenever one attempts to quantify them. Better yet: if these descriptions are to survive scrutiny for their consistency and their agreement with data, serious modifications may be required, or they may be discarded altogether. One thrust of this presentation is to show that it may be that becoming conscious is necessitated by the incompleteness of the classical McCarley-Hobson model.

The classical McCarley-Hobson Model of nonREM-REM sleep

The classic predator-prey model of Lotka-Volterra assumes that prey, in the absence of predators, have an exponential growth:

$$\frac{dR}{dt} = r_0 R \quad r_0 > 0 \quad R(t) = R(0)e^{r_0 t} .$$

In this model, the predators die out exponentially if they cannot feed on prey:

$$\frac{dn}{dt} = -\delta_0 n \quad \delta_0 > 0 \quad n(t) = n(0)e^{-\delta_0 t} .$$

The notation I have chosen makes it convenient to identify the population $n(t)$ with the nonREM (or REM-off) neurons, and the population $R(t)$ as the REM-promoting (REM-on) neurons. The population $n(t)$ consists of aminergic neurons (at least in a simplified description) and the population $R(t)$ consists of cholinergic neurons.

The model must be a system of coupled differential equations, or — biologically — two populations that interact. In the classical McCarley-Hobson model, there are inhibitory effects: during sleep (here nonREM-sleep and REM-sleep) the $n(t)$ are suppressing the REM-promoting neurons, much like predators increase their numbers by consuming prey and thereby reducing the population size of prey animals. This increase/decrease mechanism leads to interaction terms. In their most simple form, the set of differential equations then becomes

$$\begin{aligned} \frac{dR}{dt} &= r_0 R - b R n \\ \frac{dn}{dt} &= -\delta_0 n + d n R \end{aligned}$$

with all constants positive.

We can simplify the equations somewhat, so as to recognize their population features.

$$\begin{aligned} \frac{1}{R} \frac{dR}{dt} &= r_0 - b n \quad \Rightarrow \quad \frac{d}{dt} \ln R = r_0 - b n \\ \frac{1}{n} \frac{dn}{dt} &= -\delta_0 + d R \quad \Rightarrow \quad \frac{d}{dt} \ln n = -\delta_0 + d R \end{aligned}$$

In the classical McCarley-Hobson model, therefore, the logarithm of each populations is linearly coupled to the other population.

This set of equations has solutions that have the graphical form shown in Fig. 1.

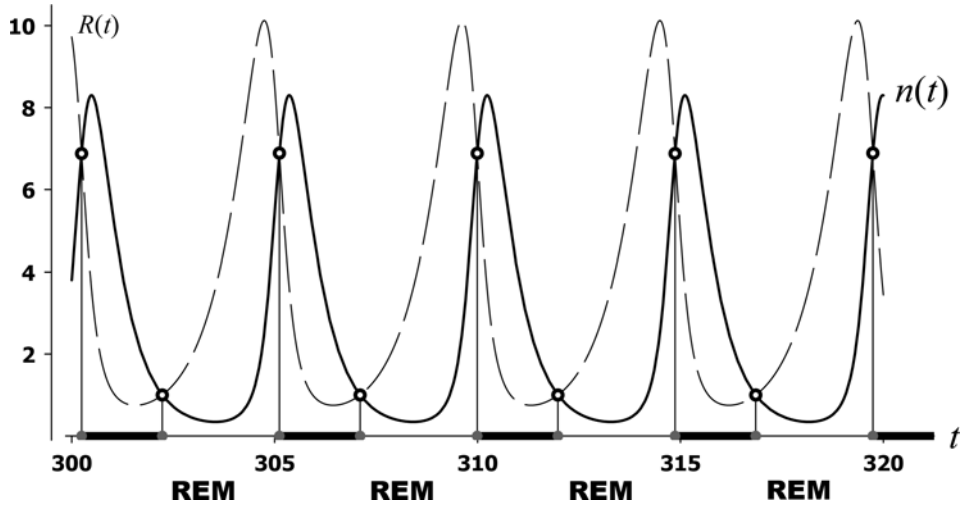


Figure 1 The solutions to the classical Lotka-Volterra system of differential equations. If we assume that REM sleep occurs when the cholinergic neurons are more populous than the aminergic ones ($R(t) > n(t)$), then the phases of REM-sleep can be determined from the graph.

There are problems with the classical McCarley-Hobson model. First, note that the bouts of REM sleep and nonREM sleep alternate periodically. Experimentally, this is not so. As will be shown below, the variability of REM bouts need not be random; rather, we can present models in which the variable length of the REM phase is due to modifications of the Lotka-Volterra differential equations, not adding a random sequence to the solution of the classical McCarley-Hobson model.

The second problem is much more severe and therefore much more difficult to rectify in a model. Because the functions $n(t)$ and $R(t)$ remain periodic, the classical REM/nonREM McCarley-Hobson model prevents the individual from awakening — consciousness does not exist in the classical McCarley-Hobson model!

A modified McCarley-Hobson Model

We adopt a somewhat artificial modification approach, similar to the one introduced by McCarley and Steriade. We assume that the coefficients that are responsible for the exponential growth/decay are functions of time, viz.

$$\frac{dR}{dt} = r(t)R - bRn$$

$$\frac{dn}{dt} = -\delta(t)n + d n R.$$

Here, we investigate solutions when $r(t)$ and $\delta(t)$ are

$$r(t) = r_0 \sin \omega t + c_0 \quad \delta(t) = \delta_0 \cos \omega t + c_0.$$

This modified McCarley-Hobson model is a six-parameter model (r_0 , δ_0 , b , d , c_0 , and ω). The advantage of this ‘simple’ modification is primarily for the investigator: he/she can trace the effects on the solutions $n(t)$ and $R(t)$.

Figures 2–5 show solutions for a suitable choice of parameter values.

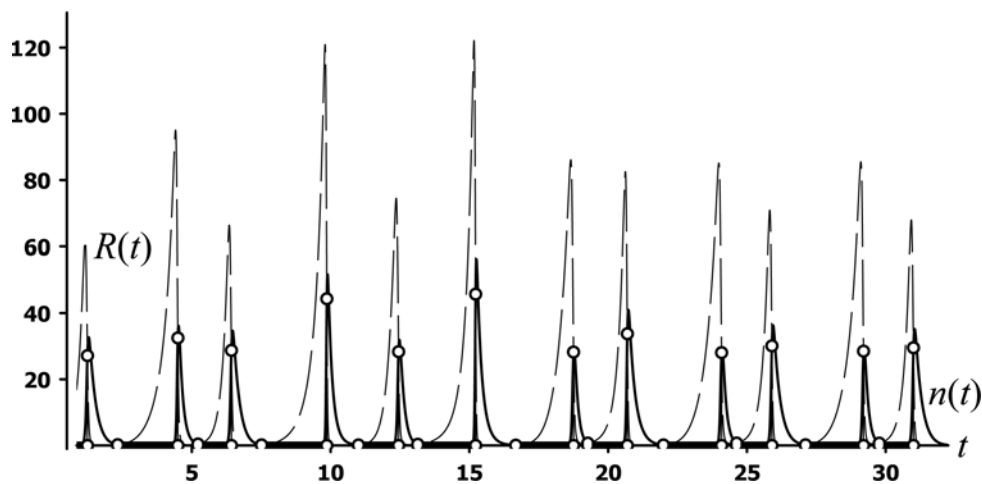


Figure 2 The solutions to the modified Lotka-Volterra system of differential equations if the growth and decay constants are periodic functions of time. If we assume that REM sleep occurs when the cholinergic neurons are more populous/active than the aminergic ones ($R(t) > n(t)$), then the phases of REM sleep can be determined from the graph. The thick lines on the abscissa indicate the intervals of REM sleep. Observe that these phases are no longer periodic, nor are their durations constant. The system of differential equations is nonetheless deterministic and there is no randomness in the variation of the duration of the REM sleep phases.

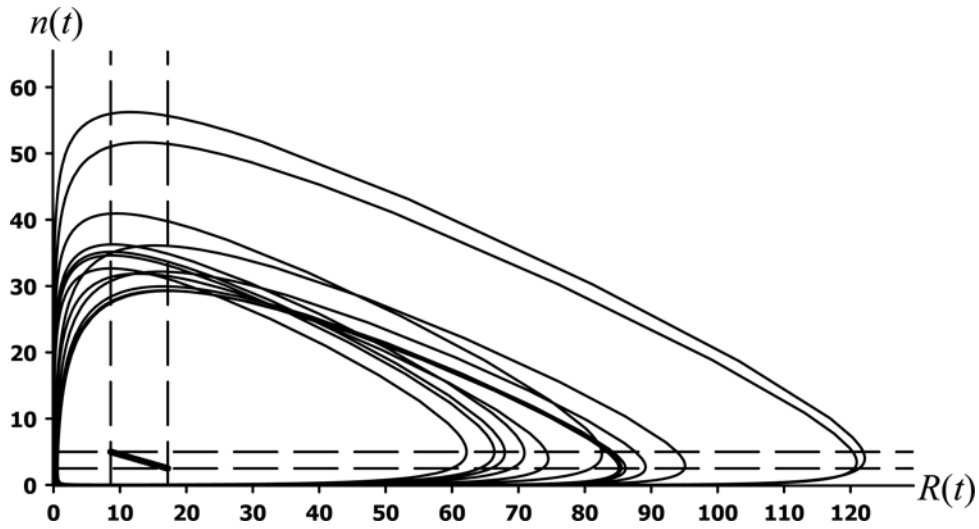


Figure 3 The solutions $R(t)$ and $n(t)$ to the modified Lotka-Volterra system of differential equations if the growth and decay constants are periodic functions of time. The dashed lines indicate the limits of the values for $r(t)$ and $\delta(t)$ (Fig. 3 for the solutions shown here). Because $r(t)$ and $\delta(t)$ remain in phase (as they have the same angular frequency), the values vary along the thick sloped line segment. Observe that there is no limit cycle for the parametric curve $\{n(t), R(t)\}$.

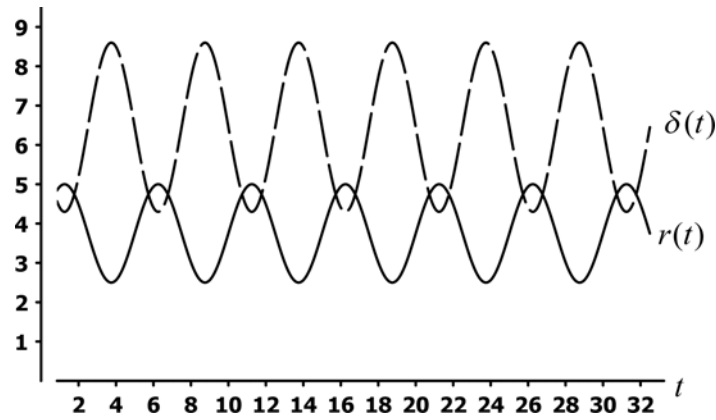


Figure 3 The time variation of the functions $r(t)$ and $\delta(t)$ used for the solutions $R(t)$ and $n(t)$ shown in Fig. 2. Despite their periodicity, both solutions $R(t)$ and $n(t)$ of the modified Hobson model exhibit stochastic behavior.

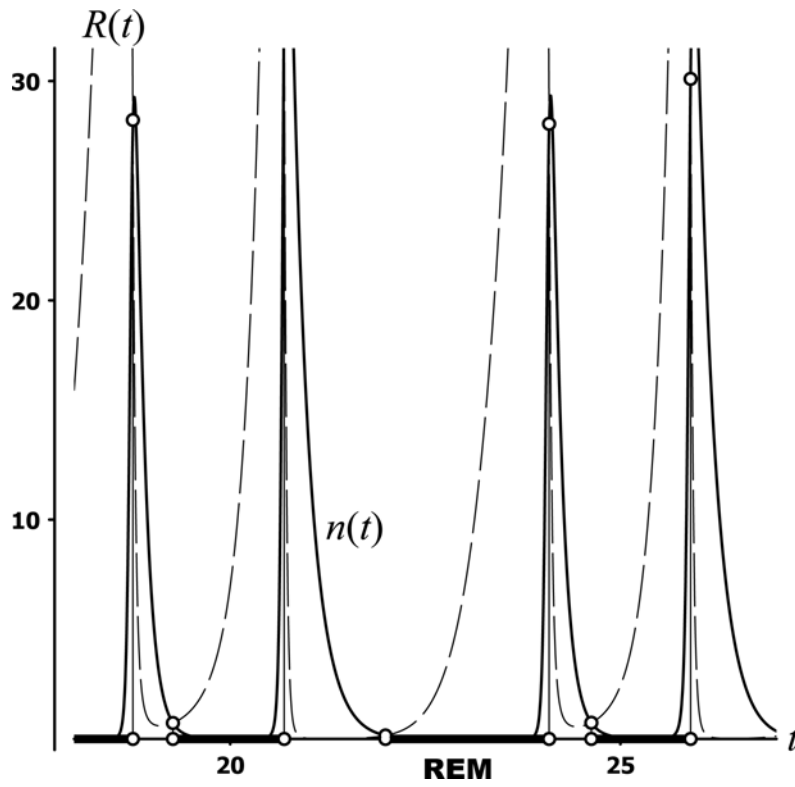


Figure 4 An enlarged detail of Fig. 2 specifying one way of modeling the REM phase. In the model considered here, REM occurs $\forall t$ while $R(t) > n(t)$. Marking the points T_k defined by $R(T_k) = n(T_k)$ on the abscissa predicts the duration of the REM and nonREM phases during unconscious life.

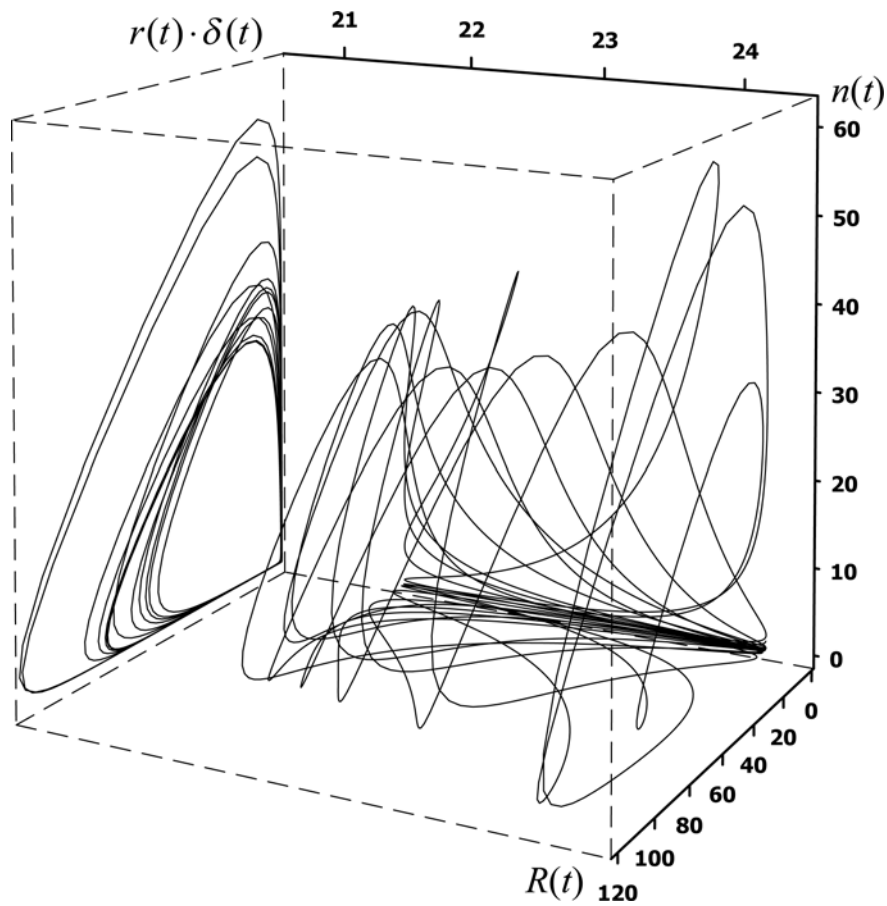


Figure 5 A graph of one possible modified McCarley-Hobson model that incorporates conscious wakefulness. The 3rd axis $C(t) = r(t) \cdot \delta(t)$ models the level of consciousness. An animated simulation shows that the system (represented by a point on the curve, for details see Fig. 6) remains parallel to the C axis near the points in $R(t)$ - $n(t)$ phase space where $R(t) \approx 0$ and $n(t) \approx 0$ — as is to be expected from psychological observations.

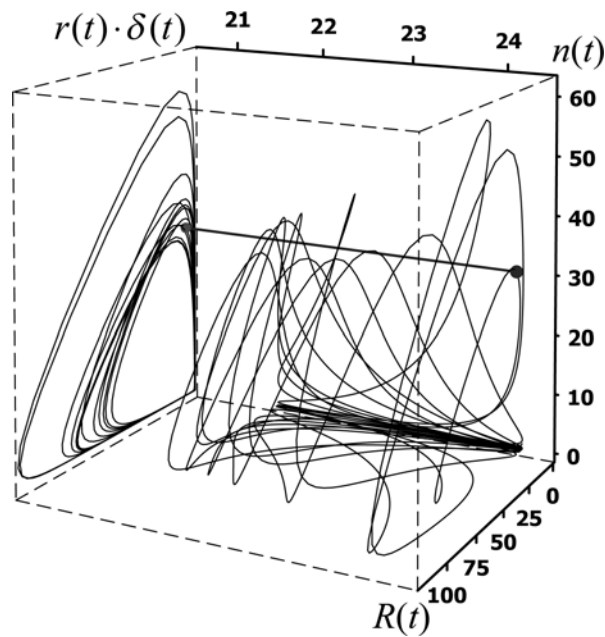


Figure 6 A moment in time of the system shown in Fig. 5. The point graphed represents the system at this moment. The modified McCarley-Hobson model of REM-nonREM sleep is a projection of the modified model curve onto the $R(t)$ - $n(t)$ plane.

The animated display of this graph shows the following behavior in the $R(t)$ - $n(t)$ plane: the system slowly moves along the R -axis (taking its time to REM-sleep), with very small values of $n(t)$. It then ‘shoots’ to the n -axis, and slowly returning along it towards the origin; during this time, we are in nonREM sleep.

This motion in the $R(t)$ - $n(t)$ plane is a projection. Two observations in the n - R - C plane are particularly noteworthy. When the system is close to $R(t) \approx 0$ and $n(t) \approx 0$, then it moves rapidly away from or towards the origin along the C -axis. If both $R(t) \approx 0$ and $n(t) \approx 0$, we are awake! Curiously, the increase in C while we are awake justifies identifying C -activity with consciousness. When the system moves parallel to the R -axis (not in projection, but rather perpendicular to the n - C plane), it occasionally moves to large C values. We interpret this as emergent consciousness during REM wakefulness; in other words: the model hints at the possibility of emergent consciousness during dreaming.

Waking up: models of the emergence of consciousness

In the modified McCarley-Hobson model presented in the previous section, consciousness was modeled artificially (i.e, without any biological/neurological consideration), namely $C(t) = r(t) \cdot \delta(t)$ was *ad hoc*. A proper, ambitious model

of consciousness must incorporate the neuronal activity that leads to its emergence when the individual leaves unconscious wakefulness. By implication, a cat would then also emerge into consciousness when it awakens. Modeling consciousness with a generalized Hobson model that models the neurological behavior of all three processes must be a system of three differential equations.

True, the modified McCarley-Hobson model of the previous section can also be written as a system of three differential equations, where the differential equation for $C(t)$

$$\frac{d}{dt}C = -r(t)\delta_0\omega\sin\omega t + \delta(t)r_0\omega\cos\omega t$$

has a solution

$$C(t) = (r_0\sin\omega t + c_0) \cdot (\delta_0\cos\omega t + c_0).$$

Good models of consciousness emergence must have a system of differential equations that model the underlying neurological processes.

$$\begin{aligned}\frac{d}{dt}R &= r(R)R - f(n, R) \\ \frac{d}{dt}n &= -\delta(n)n + c f(n, R) \\ \frac{d}{dt}C &= \mu(C)C - g(n, R, C)nRC\end{aligned}$$

Furthermore, we can revisit what the three functions $n(t)$, $R(t)$, and $C(t)$ model. Hobson, McCarley and Steriade have given a detailed account what processes in the brain justify a 2-function Lotka-Volterra model (even if generalized beyond what Hobson and McCarley had originally proposed). If we try to construct a model for $C(t)$ by incorporating the negentropic, information processing features of the Tononi paradigm, then we still need to consider what happens neurologically in the brain (and where!) and how it relates to awakening from “REM-sleep”.