

A Neurochemistry Cusp Catastrophe Model of Abnormal Sleep-Wake Cycles

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Abstract

There are various neurochemical interaction models which attempt to explain abnormal sleep-wake cycles and employ complex feedback loops to do so. There are also general state-space models of Alertness, such as the Hobson AIM model of brain activation. However, the latter does not explain the sometimes abrupt transitions observed in pathological conditions, such as narcolepsy. This paper proposes a simple state-space model based on non-linear neurochemical interactions with the characteristics of a Logistic Cusp Catastrophe. Trajectories across this state-space show various transitions in the sleep-wake cycle including smoothed normal behaviour but also accounts for abrupt pathological transitions.

Keywords: *Narcolepsy; Cusp Catastrophe*

Introduction

There are numerous diseases of the nervous system which manifest abnormal sleep-wake cycles, and there are many explanatory mechanisms identifying various neurochemical species (NCS) involvements [1]. The Hobson AIM model [2] of brain activation provides a state-space model which includes sleep-wake states, but which does not readily explain the sometimes abrupt transitions between states observed clinically. The presence of abrupt state changes suggests an underlying non-linear mechanism characteristic of a cusp catastrophe. A simple model is proposed which originates from logistic growth of competing NCS promoting and demoting neural activation, with the addition of a scavenging mechanism modelled as a sigmoid process.

Method

The model is based upon a Logistic Equation (LE) modified to incorporate a sigmoid scavenging function [3]. In the classic LE there are two controlling parameters, r = the growth rate of wakefulness and k = the waking saturation level. In the Cusp LE (CLE) the sigmoid scavenging function causes these parameters to become generalised: so R captures the overall response of NCS factors which promote wakefulness (A+); whilst K captures the overall response of factors limiting wakefulness (C+). The A and C indicate neurochemical systems most probably dominated by Aminergic and Cholinergic processes, but this is contentious [4].

Results

The state-space cortical alertness is represented by the CLE control surface, shown below. The wakefulness state at any moment is a point on the surface, whilst a trajectory across the surface shows a possible development history of alertness. The vertical scale N measures Alertness, with the bottom surface representing the sleeping state, whilst the upper surface represents the waking state.

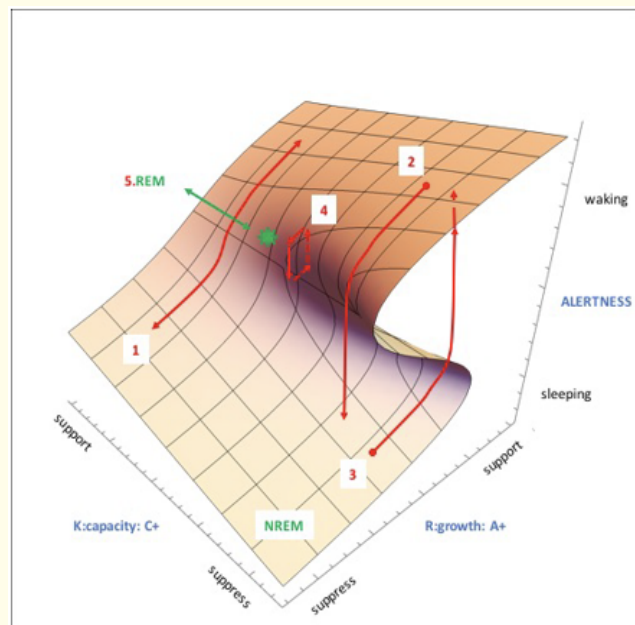


Figure 1

Conclusion

Several trajectories are demonstrated in the figure above:

1. Smooth transitions from wake-sleep states as a “normal” process.
2. Catastrophic transitions from the wake state to the sleep state, reminiscent of narcolepsy.
3. Catastrophic transitions from the sleep state to the wake state (a survival mechanism?)
4. Rapid cycling between sleep and wake states, reminiscent of delirium.
5. An intermediate bifurcation point which provides a convenient model of the metastable REM state.

Trajectory 3 is suggestive of an evolutionary mechanism shaping this control surface. The benefit of abrupt waking in a survival scenario is offset by the pathological trajectory 2 seen in narcolepsy, with an incidence of up to 1 person in 2000.

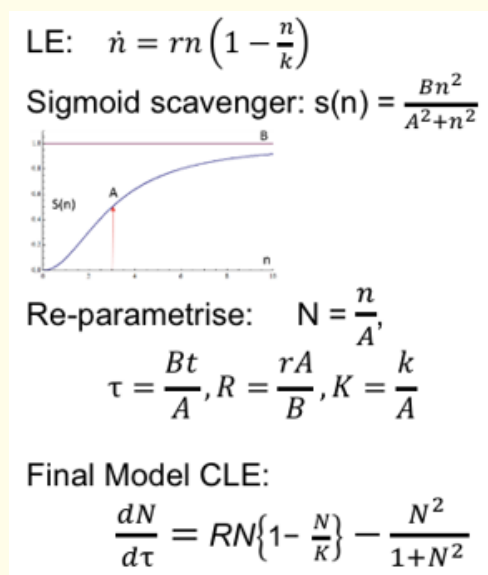


Figure 2

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