

# The Missing Link: Commentary on LaBerge's Triangular Circuit

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PSYCHE, 4(18), December, 1998

<http://psyche.cs.monash.edu.au/v4/psyche-4-18-newman.html>

**KEYWORDS:** attention, consciousness, fast (40 Hz) oscillations, thalamic reticular nucleus, thalamocortical circuit.

**COMMENTARY ON:** LaBerge, D. (1997). Attention, awareness, and the triangular circuit. [\*Consciousness and Cognition\*, 6, 149-181.](#) (See also [La Berge's precis for PSYCHE.](#))

**ABSTRACT:** LaBerge (1997) proposes that underlying attention and self-awareness is a triangular circuit involving interconnected posterior cortical, thalamic and prefrontal neural ensembles. While the target paper makes only passing reference to it, the reticular nucleus of the thalamus is an integral part of the triangular circuit he posits. Indeed, it is essential to a complete understanding of his model.

## 1. Introduction

That I share LaBerge's (1997) interest in "Attention, Awareness and the Thalamocortical Circuit" is testified to by an electronic seminar I led for the Association for the Scientific Study of Consciousness (ASSC) entitled "Thalamocortical Foundations of Conscious Awareness" (Newman, 1996). I refer the reader to Figures 1 & 4 contained in my target paper for the seminar. They are reproduced from LaBerge's (1995) book, *Attention: The Brain's Art of Mindfulness*. Figure 1 illustrates the central position of the thalamus in the brain. Figure 4 is a schematic of the basic thalamocortical circuit (<http://www.phil.vt.edu/ASSC/newman/>). If able to access these figures, you will be struck by a prominent feature in both--missing from the diagrams of the triangular circuit found in LaBerge's (1997) target article--the *reticular nucleus* (RN).

This might seem a pedantic observation. After all, the minutiae of neuroanatomy are probably best left aside when presenting an already complex circuitry to a multi-disciplinary audience. I will argue, however, based upon LaBerge's earlier work, that the target paper omits a **key element** of his triangular circuit for conscious attention and awareness.

## 2. The Missing Link

Virtually all thalamocortical loops pass through RN (as briefly noted by LaBerge). As they do so, they give off axon collaterals to it (refer to Fig. 4). The loops themselves are excitatory, whereas RN cells (which project to the thalamic relay cells, not the cortex) are inhibitory. LaBerge, Carter and Brown (1992) ran simulations of three variations upon this T-C/RN circuitry and found that all produced an enhancement of the firing of target over flanker cells in parallel T-C loops "owing mainly to the interactions arising from reticular nucleus neurons" (p. 323). They concluded the "slight initial advantage of the target location over that of the flanker is assumed to be induced by higher order (i.e., prefrontal) processes that influence afferent input to the principal cells of the pulvinar via parietal cortex" (ibid).

Here, then, is a *complete* triangular circuit for the posterior cortex. Central to the "interactions" which LaBerge et al. (1992) describe is *recurrent inhibition*, which takes place between RN cells, inducing oscillations within the thin, extended sheet formed by bi-polar RN dendrites (refer to Fig. 4). This oscillatory activity is rapidly transferred to the T-C loops, and thence, to the cortex. What role do these oscillations play in arousal and attention? In the alpha range (8-13 Hz) they initiate the descent into sleep. Again, LaBerge (1995),

During the early stages of sleep, hyperpolarization of these [thalamocortical and reticular nucleus (RN) cells] is induced by a decrease in the acetylcholine (Ach) neurotransmitter projected from the brain stem [reticular formation] . . . The rhythmic characteristic . . . is promoted by the "pacemaker" activity of RN cells. Since the meshwork of RN cells spreads the rhythmic bursts to virtually all thalamocortical cells, the spindle oscillations will become synchronized across the entire cerebral cortex . . . by virtue of the circuit properties of the reticular nucleus (p. 180).

It is important to note that the reticular nucleus and reticular *formation* are distinct structures, although they both are considered to be components of the "unspecific" reticular activating system (RAS) involved in the generation of the various cortical EEG states associated with deep sleep, dreaming and waking (Jasper, 1960; Scheibel, 1980; Steriade, 1996).

### 3. The Role of Synchronous Oscillations in Attention and Awareness

What happens when brain stem Ach *increases*? Well, predictably, the animal wakes up, or if already awake, becomes hyperalert to stimuli in their environment; and their EEG is "desynchronized", i.e. low-frequency oscillations (less than 10 Hz) are replaced by fast, irregular gamma activity (more than 30 Hz). As LaBerge (1997) notes, such "neuromodulatory centers in the brain stem . . . are necessary . . . preconditions for states of attention and consciousness," but adds that although these brain stem nuclei "project richly to thalamic nuclei . . . the distribution of the[ir] fibers is too diffuse to account for . . . selective attention" (p. 158).

According to Scheibel (1980), there is at least one thalamic nucleus for which this is not the case: RN. Based upon his and Skinner and Yingling's (1977) elucidation of an extended reticular-thalamic activation system, Baars and I (Newman & Baars, 1993; Newman, 1995a, 1995b, 1997) have developed a model which includes the reticular formation as an integral "player" in global (i.e., conscious) attention, mediated via the thalamocortical circuit. Research with cats (Steriade, Curro Dossi, Pare & Oakson, 1991) has shown these brain stem (Ach) nuclei to potentiate fast (40 Hz) oscillations in thalamocortical systems. Crick & Koch (1990), based upon a variety of studies, hypothesized that synchronous 40-Hz oscillations could provide the basis for "binding", i.e. the integration of feature-detection activities in multiple cortical areas into a single object of attention. In humans, Kinomura, Larsson, Gulyas and Roland (1996) have demonstrated that subjects' fMRIs showed significantly higher levels of activity in *both* the thalamic portion of the reticular activating system and midbrain reticular formation (MRF) during an attentional task.

What exactly is the role of this extended activation system if (as seems to be the case) cortical activity is central to generating the explicit *contents* of our awareness? Crick (1984) hypothesized that the T-C/RN circuit could act like an "attentional searchlight", selectively heightening, and binding together, a succession of areas in the cortex representing a salient object of perception. His early candidate mechanism for this did not pan out, but recent experiments suggest 40-Hz binding could serve such a purpose. Such binding has been largely attributed to reentrant, corticocortical circuits; but Munk, Roelfsema, Engel and Singer (1996), in experiments with cats, found that activation of the reticular formation facilitated "oscillatory activity in the gamma frequency range and enhanced the stimulus-specific synchronization of neural spike responses in the visual cortex of cats" (p. 271). Thus, this extended reticular-thalamic activating system would appear to contribute to the selective enhancement and binding of "specific stimulus configurations" without altering them, much like a searchlight focuses our attention on center stage without altering the performers' actions. (For a thorough review of these robust, but still controversial, findings, refer to the archived ASSC e-seminar, "Temporal Binding, Binocular Rivalry and Consciousness", at <http://www.phil.vt.edu/ASSC/esem.html>).

## 4. Top-Down Control

It may seem that I am arguing for an alternative, reticulo-thalamic system as the "source of attentional control" LaBerge (1997) associates with prefrontal areas (p. 158). Actually, though, I am simply elaborating upon his triangular circuit model. The great bulk of PFC projections to the thalamus go to the medial dorsal nucleus (MD). MD does not share extensive projections with other thalamic nuclei involved in specific sensory and motor circuits, although it does share some with PPC. LaBerge (1990, 1997) reviews several PET studies in humans showing that, in tasks requiring sustained focal attention to complex visual stimuli, three thalamic regions were especially active: the pulvinar, MD and the "ventrolateral nucleus, which connects with both the frontal areas and the basal ganglia" (p. 163). This is not entirely accurate. The ventrolateral nucleus does receive inputs from PFC via the basal ganglia, but projects to the motor/premotor cortex *behind* PFC. As LaBerge notes, another piece of data that does not seem to fit with his PFC circuit model comes from Posner and Petersen (1990), who propose the anterior cingulate cortex as the site of executive control, based upon other PET studies showing its heightened activation during similarly demanding attentional tasks.

Recent anatomical data suggest a more elaborate circuitry which, once again, does not contradict LaBerge's (1997) model so much as elaborate upon it. In a series of studies, Anthony Grace and his colleagues at the University of Pittsburgh, employing new tracing techniques in rats, have found that PFC influences upon the thalamus are "gated" via a strategic nucleus in the basal ganglia which sends parallel, topographic projections to MD, RN **and** the anterior cingulate. In a recent paper, they conclude,

it is possible that the RTN [reticular nucleus] areas identified in these experiments selectively control the MD-PFC activity. Thus, the RTN region that receives inputs from the [basal ganglia] system appears to control the activity of the thalamocortical regions that are targeted by the same component of [that system, which] may ultimately adjust the level of rhythmicity [oscillatory activity] within the RTN-MD system (O'Donnell, Lavin, Enquists, Grace & Card, p. 2164). [<1>](#)

Thus, with a few loop-the-loops added in "to achieve a sufficiently intense and long-lasting activation in a column cluster," LaBerge's (1997) triangular circuit for attentional control has a plausible neurophysiological basis--but not without the reticular nucleus. Newman and Grace (in press) will elaborate upon the extended model I outline here. In our view, LaBerge's triangular circuit is a necessary, but not sufficient, basis for awareness of an object in relation to self schemas generated in higher cortical areas.

## Notes

<1> For additional information about and references to this research, see A.A. Grace's webpage: <http://www.pitt.edu/~neurosci/faculty/grace/grace.html>.

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