



# Latest Findings in the Mechanisms of Cortical ‘Arousal’: ‘Enabling’ Neural Correlates for All Consciousness

by

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*Electroneurobiología* 2006; **14** (2), pp. 199-210; URL  
<<http://electroneubio.secyt.gov.ar/index2.htm>>

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**ABSTRACT:** This paper differentiates traditional "cortical arousal" concepts into separate components for the functional states termed REM ("rapid-eye-movement sleep"), Quiet-Waking, and Active-Waking, and traces their involved mechanisms in the neuronal and biochemical level, *i. e.*, skipping from consideration any more fundamental issues such as quantum or relativistic biophysics. In a short review of the latest findings, these "classical" mechanisms include (a) *basal forebrain* acetylcholine "final path" projections to the cortex for "basic arousal" in

REM and quiet wakefulness; (b) *brainstem* neurotransmitter systems allowing incoming stimuli to activate "arousal", and recruiting additional systems to turn "basic arousal" into the fuller arousal and capacities of Active Wakefulness state; (c) thalamic intralaminar-nuclei cortical projections that connect anterior and posterior cortex for processing sensory stimuli; (d) thalamic reticular-nucleus which can shut down perceptual transfer between thalamus and cortex or allow gated transfer; and (e) circadian mechanisms in the basal forebrain, hypothalamus and pineal gland that direct transitions from waking to slow-wave sleep to REM and back. (f) More speculatively, it is considered that circadian clocks alternating control between left and right hemispheres may be involved in rotations between slow wave sleep (SWS) and REM sleep.



## Introduction

Inside Anglo-American academe, John Searle (2000; 2005) has been chastising the consciousness researchers for spending so much time on what he calls the "*building blocks*" of the neural correlates of consciousness: what it takes for specific mental 'content' to compete for conscious workspace, as in binocular rivalry. Searle rather calls on us to focus, instead, on the "*unified fields*" question – what makes creatures conscious versus in a deep sleep, coma, or vegetative state, pursued at the neuronal and biochemical level without consideration of any more fundamental biophysical issues, *e. g.* quantum or relativistic. Such mechanisms in this "classical" level are what Searle calls *the* neural correlates of consciousness (NCC). Illustrative of Searle's concerns, Christof Koch, in his otherwise-superb book *The Quest for Consciousness* (2004), spends a measly eight pages or so on Searle's "unified fields" (which Koch calls the "*enabling*" correlates of the NCC) and the rest of the book on Searle's "building blocks", and most of that on *visual* perceptual consciousness. This article, which for the sake of synopsis leaves outside of its scope the issues of non-REM-sleep experience (reviewed in Nielsen 2003) and the troubles posed by the non-mammalian EEG-characterization of sleep-waking states (Crocco 2005), is an attempt to pull together the latest findings on the unified fields/enabling neural correlates for *all* consciousness and to relate the "enabling" NCC to the "building blocks" NCCs.

## 1. Basic Arousal found in REM and Quiet Wakefulness:

### Basal Forebrain Acetylcholine

It has been traditional to consider both *REM* and *waking* states to be *states of consciousness* in the phenomenal sense of having conscious experiences; but to bestow that title to only *waking* states in the medical sense of being conscious of ones surroundings. While REM and waking states share some commonalities in terms of "phenomenal" experiences – "*The dream seemed so real!*" –, there are many "access" differences between REM and waking that affect the quality of the "phenomenal" experiences. Somewhere between REM and "*active waking*" states are the states of "*quiet wakefulness*" (Jones, 1998), such as restfulness, absorption, trance, and meditation. The cortical arousal that is common to REM and to *quiet wakefulness* can be called a state of "basic-cortical-arousal" (my term) or a state of "sustained attention" (Sarter & Bruno, 1998), except that – in the non-lucid REM case – one is unable to intentionally interact with what is being attended.

**Acetylcholine (ACh)** cells in the **basal forebrain (BF)**, just anterior to the hypothalamus, seem to be the "**final common pathway**" (Dringenberg, 1998) for activating the cortex (Jones, 1998), by tonically depolarizing receptors on dendritic spines of rows of cortical pyramidal (projecting-out) cells in adjacent cortical columns (Dringenberg, 1998). This is likely achieved by preventing the escape of potassium ( $K^+$ ) ions from these cells (Jones, 1998). This *tonic effect* on parts of the cortex presumably leads to the conscious state shared by *quiet waking* and REM sleep (Sarter & Bruno, 1998). This makes these cortical cells "trigger happy", so to speak, so that they can easily be activated by relevant stimuli impinging onto them (whether externally derived in wakefulness or internally derived in both wakefulness and REM). The classic term "*arousal*" seems to imply that the whole cortex is activated. Yet such homogeneous activation would be functional mayhem. It seems more like major sections of the cortex being moved from "*at ease*" to "*get ready, get set . . .*," which makes it readily possible for them to serve function in normal ways.

Since ACh projections are active during both REM and quiet wakefulness, perhaps the degree of REM activation is a "*get ready*" state. Then, hypothalamic **histamine** projections to both brainstem and basal forebrain acetylcholine nuclei may be necessary to *further activate* the ACh cells to the "*get set*" stage of quiet wakefulness. **Norepi-**

**nephri**ne and **serotonin** activation may be what allows a further degree of cortical arousal for the "go!" of **active** wakefulness (Speigle, 2004).

Clusters of acetylcholine cells, both in the BF and in the upper brainstem, are very active during both REM and waking states, but virtually silent during slow wave sleep (SWS) (Hobson, 2005; Hobson *et al.*, 2000). During REM activated dream phases, it is only the brainstem *acetylcholine* cells (rather than brainstem norepinephrine, dopamine, etc., cells) that activate the BF acetylcholine cells (Sarter & Bruno, 1998), while during wakefulness, basal forebrain acetylcholine's wide projection to the cortex is moderated by a number of other brainstem projections to it. This leads us to examine pathways that are active during *wakefulness*, but *not* during *REM*.

## **2. Brainstem Neurotransmitter Systems' Ventral Projection to Basal Forebrain**

### **a. allow incoming stimuli to activate "arousal"**

Stimulation of the upper-pons-through-midbrain area wakes a sleeping cat, while destruction of the area puts a normal cat into permanent coma. From the days of Moruzzi and Magoun (1949) on, this upper brainstem area was seen as crucial for arousal. Cortical projection from the *acetylcholine* nuclei in the *brainstem* was generally heralded as *the* consciousness generator, or rather the generator of a necessary (if, perhaps, not sufficient) condition for enabling the observer's connection with the surroundings, while other brainstem area projections from dopamine (DA), norepinephrine (NE), serotonin (5HT), glutamate (GLU), and endorphins (EN), and the posterior hypothalamus histamine (H) system were seen as playing supportive roles (Hobson, 2000; Faw, 1997, 2000).

Now that the *basal forebrain* acetylcholine clusters are seen as playing the main role in the state called cortical arousal (Schiff, 2004), the crucial role of *brainstem* (and histamine) projections to the acetylcholine cells in the basal forebrain seem to be **the means by which incoming stimuli can activate** arousal, motor control, and salience-detecting systems (Dringenberg, 1998).

This involvement is seen most clearly in the process of being awakened by an alarm clock or something else. While you are dreaming, a bright light, a loud noise, an earthquake, a bitter taste in your mouth or the smell of smoke can wake you up. What you see, hear, touch, or taste can activate *brainstem* systems that are activated during wakefulness, but turned way down during both REM and SWS, including, NE, DA, GLU and, to a lesser extent, serotonin. These project to the basal forebrain and surrounding areas to activate *from below* the supportive cortical projections that move one to active wakefulness. What you *smell* activates the basal forebrain area *from above* – from olfactory centers (Jones, 1998). This network of activation from the sensory systems – and a more or less continual bombardment of sensory input throughout your waking day seem crucial to keep you in active wakefulness.

## **b. Brainstem systems link basic arousal system to other systems**

Serotonin seems to activate the cortex directly and other systems indirectly through ACh projections (Dringenberg, 1998), to move the tonic cortical condition from conditions of REM or quiet waking to active waking. A lot of this occurs by way of an "arousal ventral pathway" (Jones, 1998) through the hypothalamus to the basal forebrain. The projection of **NE** from the pontine locus ceruleus to the BF acetylcholine bed seems to be crucial for helping subcortical "salience-detecting" centers, such as the **DA** "reward circuits", amygdala, and hippocampus "recruit" the BF Ach projections to the cortex (Sarter & Bruno, 1998; Dringenberg, 1998). NE also activates *primary sensory areas* and the *right prefrontal* areas – areas whose products become inaccessible to experience or "shut down" during both SWS and REM sleep – seemingly to enhance or prolong wakefulness, including increases in gamma EEG activity (the famous "40 Hz" oscillations) (Jones, 1998) and maintain vigilance to external events. Downward projection of NE enhances muscle tone and may set the stage for active wakefulness and coordinating motor response with vigilance (John *et al.*, 2004).

The heavy projection of basal forebrain acetylcholine cells to the **hippocampus**, made possible by brainstem NE projections, seems a condition to turn the hippocampus functional as to what should be evaluated and consolidated into long-term memory. Indeed, very re-

cent findings indicate that it is the BF acetylcholine projection to the hippocampus that stimulates stem cell neurogenesis into a condition in which it becomes possible to consolidate new memories (Mohapel *et al.*, 2005). This is quite consistent with the long-known fact that BF acetylcholine destruction is linked to the loss of new memory consolidation in Alzheimer's. BF projections to the **cortex** are involved in making those cortical areas more responsive to perceptual and other processing (Jones, 1998) and in helping *consolidate into memory* the representations projected there through convergence upon perceptual glutamate pathways (Rasmusson, 1998).

While the term "*cortical arousal*" implies a very diffuse non-specific ACh projection to the cortex, these ACh projections seem to have both *general tonic* and *specific phasic* characteristics (Szymusiak, 1998). Specific sensory input might further increase acetylcholine activity in specific cortical regions (Szymusiak, 1998). Thus the term "generalized arousal" may be too broad (Sarter & Bruno, 1998). And yet, these projections (and some others to be named) do prepare the cortex for specific sensory input. A modified definition of cortical arousal still seems appropriate.

### **3. The Dorsal Arousal Extension:**

#### **a. Thalamic Intra-laminar Nuclei (ILN)**

The pontine/midbrain reticular formation seems to extend in a "dorsal path" into the thalamus into a cluster of five midline nuclei stuck in the white-matter lamina separating lateral from medial thalamus, collectively called the **intra-laminar nuclei (ILN)**. These thalamic ILN nuclei project to diverse cortical and subcortical areas to tonically activate the cortex. This projection to the ILN and from there to the cortex involves **glutamate** (Parvizi & Damasio, 2001). The ILN projects quite widely to the superficial layers of the cortex, to prepare cortical cell columns to better receive specific input. This dorsal pathway through the thalamus has *classically* been seen as the *cortical arousal system*.

More recently (Schiff, 2004), the ILN projections to the cortex have been found to be *diffuse*, but *not undifferentiated*. The five ILN nuclei project to slightly different parts of the cortex, with the rostral nuclei projecting to prefrontal, posterior parietal and primary sensory areas; and the caudal nuclei projecting to premotor and anterior parie-

tal cortex – and both groups projecting to corresponding parts of the basal ganglia (Schiff, 2004). They seem to be activating different parts of **long-range cortical pathways** (one might think of *Autoban* or Interstate Highways as a simile), especially connecting frontal and parietal lobe, involved in conscious processing and working memory of contents of sensory experience sustained by brain states whose neuroactivity is being processed in more local subdivisions and *cul de sacs* – and their related basal ganglia loop sections. **These long-distance connections are crucial for normal waking state functioning.** Hobson (2000, 2005) contends that these long-distance connections are almost totally shut down during both REM and deep sleep, an assertion not yet verified independently. Anyway, the two main projections to the cortex considered to be "*general arousal*" projections (from the ILN Thalamus and this BF ACh projection) are now seen as having considerable *specificity* (Jones, 1998). Recently it has been shown that the ILN neurons are modulated by posterior-hypothalamic hypocretin (Hcrt) neurons (Schiff, 2004).

## b. Thalamic Reticular Nucleus (RNT)

As part of a one-two arousal punch for this dorsal extension, the brainstem **acetylcholine** projection to another thalamic fixture is crucial for normal waking arousal. Surrounding much of each of the two thalami is a net-like covering called the **reticular** (net-like) **nucleus** of the thalamus (**RNT**), through which to-and-fro projections between the thalamus and cortex pass (imagine wires sticking through a window screen). These passing fibers send collaterals to the "screen" RNT to laterally inhibit transmission through *other* fibers sticking through neighboring parts of the screen. This seems to be a major mechanism employed in selective attention (LaBerge, 2000). During waking states, the RNT thalamus seems in "**transport mode**" (Detari, 1998) and the doors of perception are open, so that one can interact with the outside world. In transport mode, the reticular nucleus of the thalamus serves the basic "**gating**" function of determining which thalamic-cortical "relay" pathways are in play and which are not.

During sleep, the thalamus is in the "**oscillatory mode**" (Detari, 1998). The inhibitory reticular nucleus is massively turned up during sleep, inhibiting cortical pyramidal cells, and thus serving as what appears as a pacemaker for the thalamic spindle oscillations of deep sleep. Both brainstem and basal forebrain (Dringenberg, 1998) **ace-**

**tylcholine** and NE, serotonin and H projections (Detari, 1998) block the generation of these spindles and thus initiate the wakeful state (Steriade & McCarley, 1990; Steriade, 2006; Parvizi & Damasio, 2001), placing the thalamus into "transfer mode".

When the ILN projects to various parts of working memory cortex/basal ganglia loops and when the reticular nucleus of the thalamus allows interaction between sensory systems and cortical areas, the BF Ach projections to those same cortical areas are involved in enabling the same basic mechanisms of memory consolidation found in the hippocampus. The lack of this two-way punch during SWS and REM is probably involved in determining why we have such poor memory of SWS mentation and REM dreams, unless we awake from them.

#### **4. Circadian involvement: SCN, VLPO and Pineal**

We have already noted that sudden or intense sensory stimulation from any modality can activate the waking mechanisms through brainstem and olfactory systems – adding activation beyond the basic ACh activation. But, most of us do not always continue sleeping until something awakens us. We have natural circadian systems that control shifts from waking to SWS to REM and so on. These, in turn, are affected by changes in light duration, physical exercise, hormonal changes, stress conditions, and recent mental activity.

Several inter-connected hypothalamic neuron clusters and the pineal gland are involved in shifts of circadian rhythm, which then control the brainstem arousal and motor areas involving the various neurotransmitters. In **anterior hypothalamus** are the **suprachiasmatic nucleus (SCN)** and **ventro-lateral preoptic area (VLPO)**, the latter being adjacent to the basal forebrain. Rats with damage to the SCN sleep erratically, showing no night-day rhythm.

The **SCN** receives direct retinal input through the retino-hypothalamic tract and indirect visual input from the LGN thalamus, to adjust natural sleep-waking cycles to daily and seasonal shifts in light. The SCN projects to the **pineal gland** to convert serotonin to melatonin, whose available concentration is somehow related in diurnal species to drowsiness in response to waning light. The SCN projects to other hypothalamic nuclei to modulate body temperature and the pro-



duction of hormones consistent with circadian needs. The **VLPO** area has "sleep cells" that project the major inhibitory neurotransmitter **GABA** to turn down a number of arousal and motor centers (Jones, 1998; Morairty *et al.*, 1998). It is increasingly being appreciated that **adenosine** buildup in anterior hypothalamus and basal forebrain (Szymusiak, 1998) may be a major sleep factor (Morairty *et al.*, 1998). Adenosine inhibits the neighboring **ACh** neurons in the basal forebrain (McCarley, 1998). All cellular activity involves adenosine in energy transfer and in second-messenger reactions for hormones and neurotransmitters. The longer one is awake, the more this adenosine builds up. Its **buildup specifically in the basal forebrain** has recently been seen to be directly proportional to sleepiness (McCarley, 1998; Porkka-Heiskanen, 1999). Consistent with these findings is the fact that the main effect of caffeine is on the adenosine second-messenger system, inhibiting the breakdown into adenosine. Thus, GABA and adenosine may be specific slow-wave sleep factors, moving us from wakefulness to SWS.

In **posterior hypothalamus** are the newly examined **hypocretin (orexin)** and **histamine** systems. The **hypocretin** system seems to be a "toggle" switch (Schiff, 2004) by which circadian mechanisms can directly activate the brainstem and basal forebrain arousal and motor systems – in addition to any sensory stimulation. The **hypocretin (Hcrt)** area projects densely to the **SCN**, helping to maintain the full circadian cycle. Lower Hcrt levels leads to lower amplitude of circadian cycles and thus to **narcolepsy** (John *et al.*, 2004). In terms of waking arousal mechanisms, Hcrt projects to both *brainstem* and *basal forebrain* **acetylcholine** centers, likely leading to cortical arousal, during both waking and REM (Kiyashchenko *et al.*, 2002). Hcrt also projects to the ILN neurons in the thalamus to modulate them (Schiff, 2004). The Hcrt-2 neurons project to excite neighboring posterior-hypothalamic **histamine (H)** neurons. Histamine, in turn, projects to the thalamus, to brainstem and basal forebrain arousal centers and to much of the cerebral cortex (Gazzaniga, 2002). Histamine neurons are found to be basically quiet during both REM and SWS sleep, highest during active waking and fairly high during quiet waking. This implicates H for waking but not REM cortical arousal. And the arousal effect of histamine is heavily dependent on Hcrt. Very recent studies of Hcrt, histamine and norepinephrine (NE) show a very interesting interaction. In **narcolepsy** (when one is asleep plus paralyzed), histamine levels are quite low. But, during **cataplexy** (which combines the greatly reduced muscle tone – paralysis – found in REM and narcolepsy, with the awareness of events

around one, found in wakefulness), **H** levels are at the same level as in quiet wakefulness – which catalepsy seems to be – but **NE** levels are very low and **5HT** levels are pretty low. This suggests that **histamine** may be a crucial factor in **waking arousal** – and then **NE**, **5HT** and **DA** – add to waking arousal the “**active**” component of “active waking” (John *et al.*, 2004). Stimulating H centers leads to arousal and, as we all know, antihistamine leads to drowsiness.

## **5. Circadian clocks alternating control between left and right hemispheres may be involved in rotation between SWS and REM sleep**

One of the least developed questions in terms of circadian control of conscious and unconscious states is the alternation between slow wave sleep and REM through the sleep cycle. REM phases are initiated by so-called **PGO** (pontine-geniculate-occipital) spikes from **acetylcholine** cells in the pons. The tonic inhibition of the gamma-motor neuron system, which virtually paralyzes most somatic muscles during REM is triggered by acetylcholine cells in the medulla. But what circadian mechanisms set them off?

There is considerable evidence for a 90-minute or so alternation between left and right hemisphere dominance throughout waking and sleeping states. A very recent field of study has been proposed to demonstrate one extremely simple and low-tech way of detecting which hemisphere is dominant at any one time, which is to restrict the breathing through one nostril at a time and determine which one is actually breathing – or breathing most robustly – at that time. The opposite hemisphere is presumably dominant at that time.

It is now clear that there is “mentation” during both REM and SWS parts of the cycle, but some such as Hobson (2000) contend that REM mentation tends to be much more perception-like, while SWS mentation tends to be much more verbal-thought-like. It might be that, for most people, the SWS mentation is linked to parts of the hemispheric cycles when the left hemisphere is dominant and REM mentation linked to the right. Solving this switch from SWS to REM and back will allow us to pull together a coherent story of *the* neural correlates of conscious states.

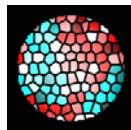
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revista

*Electroneurobiología*

ISSN: 0328-0446