

## from sleep to attention – lecture 3

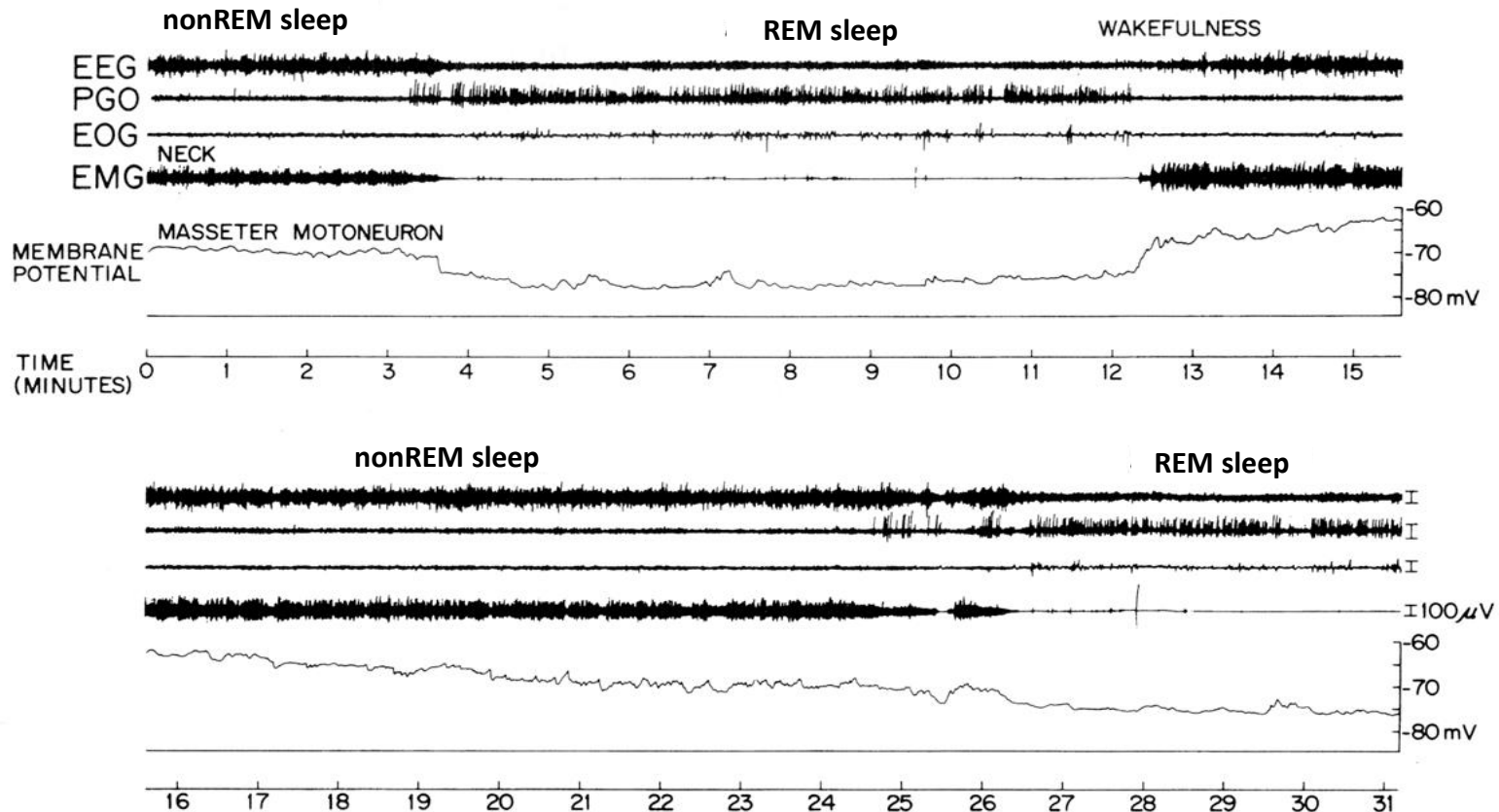
### control of sleep/wake state production I



The Common Sense [senso comune], is that which judges of things offered up to it by the other senses. The ancient speculators have concluded that that part of man which constitutes his judgment is caused by a central organ to which the other five senses refer everything by means of impressibility [impressiva]; and to this centre they have given the name Common Sense. And they say that this Sense is situated in the centre of the head between Sensation and Memory. And this name of Common Sense is given to it solely because it is the common judge of all the other five senses i.e. Seeing, Hearing, Touch, Taste and Smell. This Common Sense is acted upon by means of Sensation which is placed as a medium between it and the senses. Sensation is acted upon by means of the images of things presented to it by the external instruments, that is to say the senses which are the medium between external things and Sensation. In the same way the senses are acted upon by objects. Surrounding things transmit their images to the senses and the senses transfer them to the Sensation. Sensation sends them to the Common Sense, and by it they are stamped upon the memory and are there more or less retained **according to the importance or force of the impression**. That sense is most rapid in its function which is nearest to the sensitive medium and the eye, being the highest is chief of the others. Of this then only we will speak, and the others we will leave in order not to make our matter too long. Experience tells us that the eye apprehends ten different natures of things, that is: Light and Darkness, one being the cause of the perception of the nine others, and the other its absence:--Colour and substance, form and place, distance and nearness, motion and stillness. – **Leonardo Da Vinci**

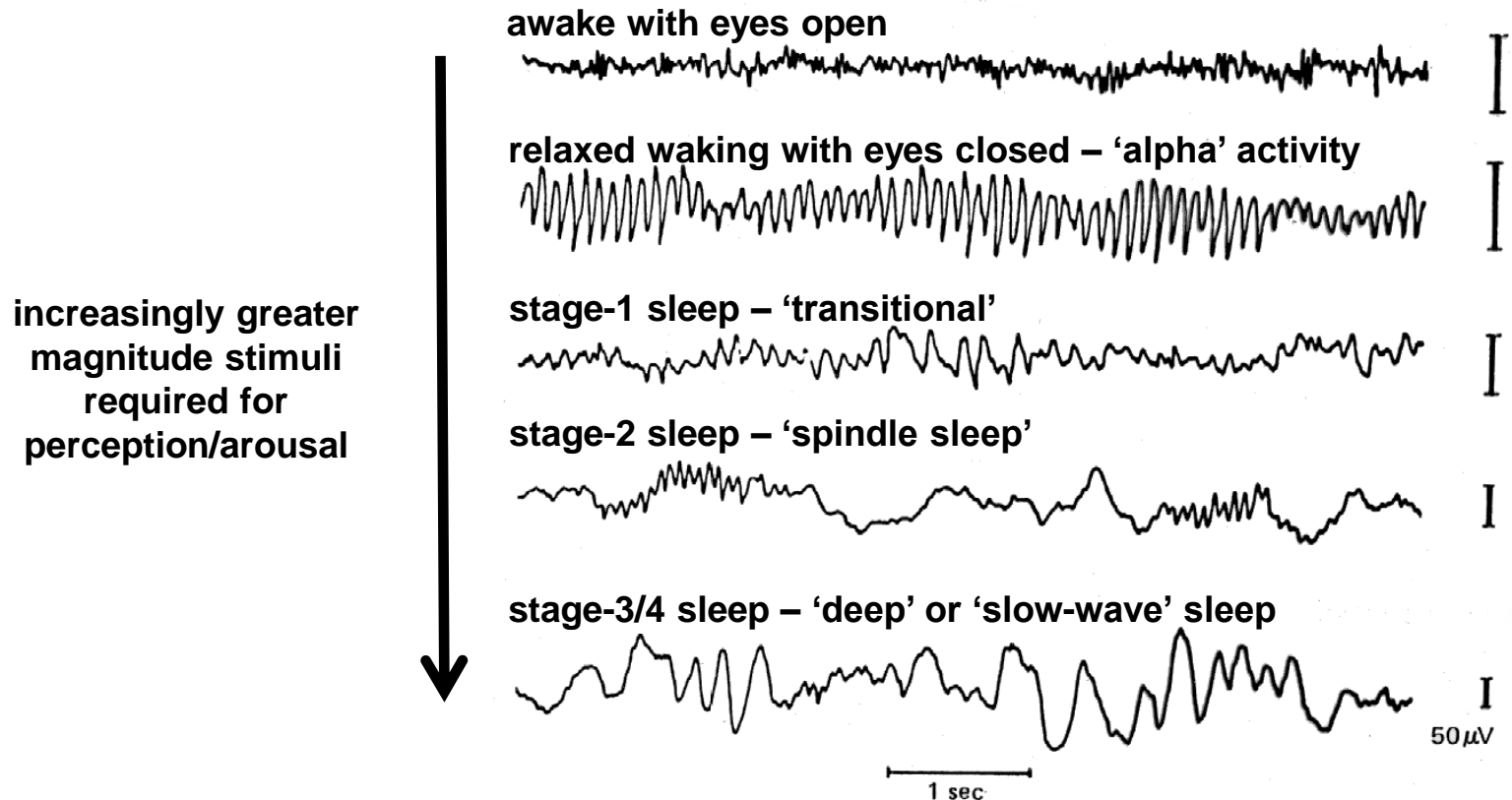
# 'PGO' spikes differentiate the thalamic and cortical patterns of REM sleep and waking

## NORMAL SLEEP

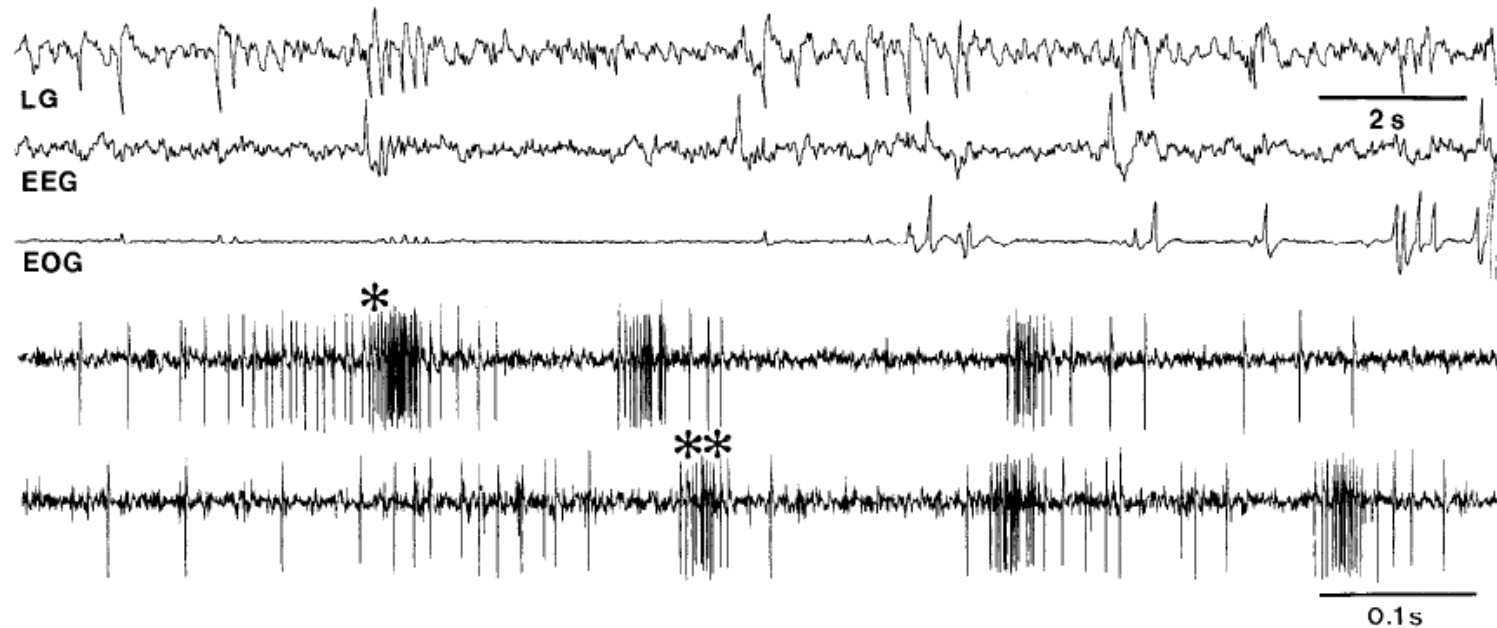


Chase MH, Morales FR (2000). In: Principles and Practice of Sleep Medicine (Kryger MH, Roth T, Dement WC, eds), pp 155-168. Philadelphia: W.B. Saunders, 2000

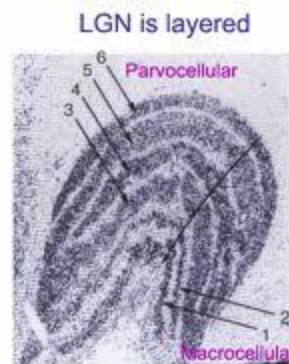
**the basic patterns of cortical EEG/LFP activity in the cerebral cortex  
closely track sleep depth as assessed by arousal**



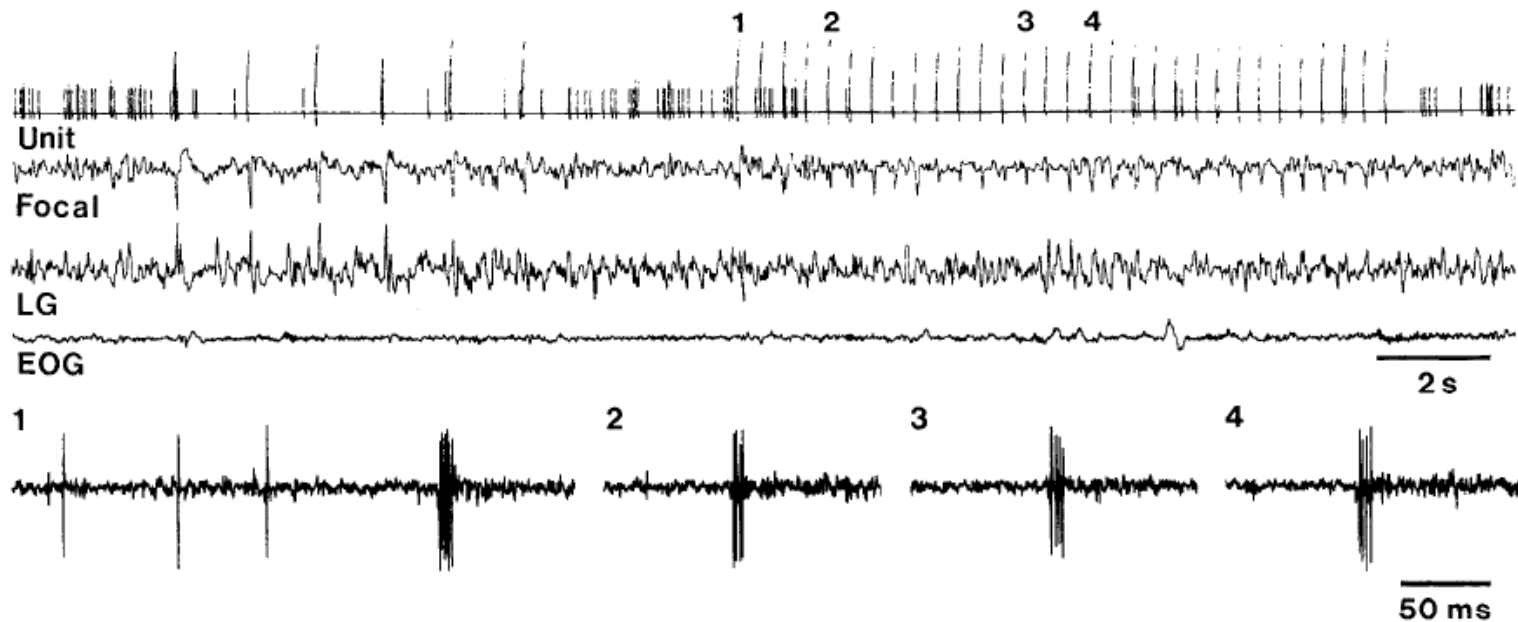
**REM sleep is characterized by phasic events generated by bursting of brainstem reticular formation neurons – these ‘events’ may take the form of eye movements, twitches of non-axial musculature, and/or LFP events in the lateral geniculate nucleus that are called ‘PGO waves’**



Steriade et al., 1990, J. Neuroscience



**PGO spikes can also be elicited in waking states, but primarily in response to surprise stimuli (such as a hand clap) that attracts attention in the form of an orienting response**



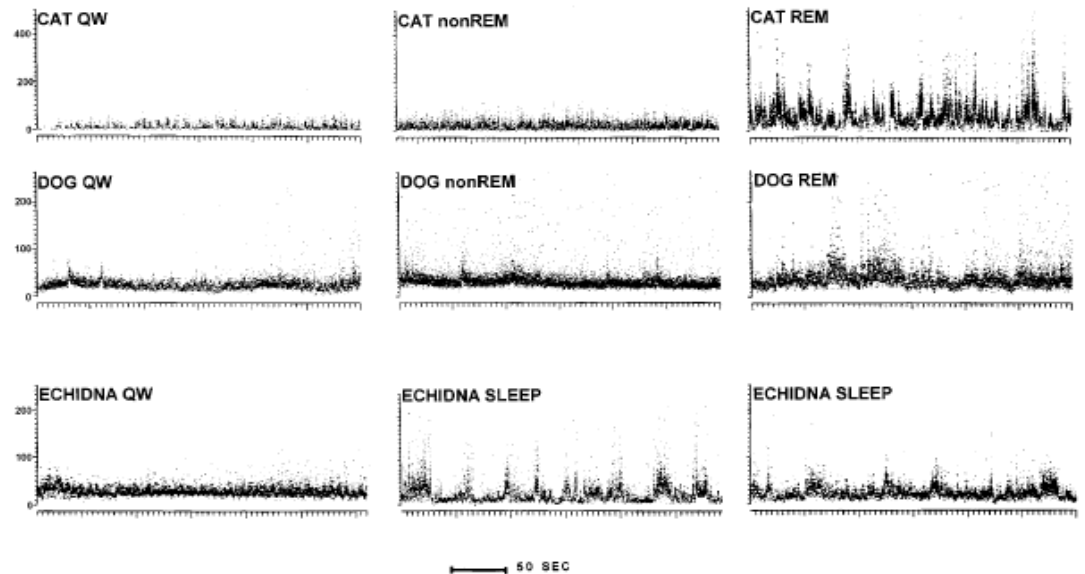
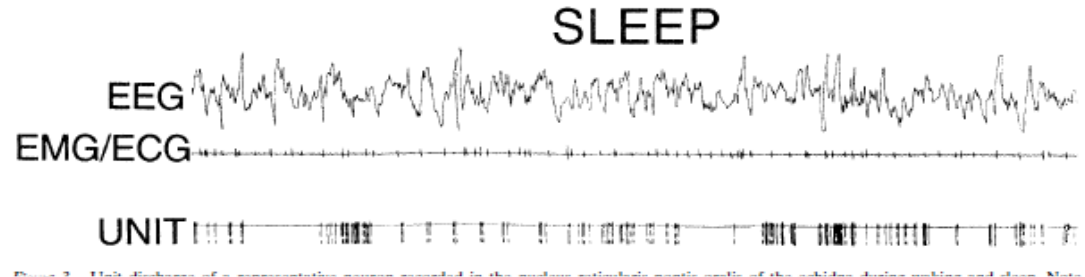
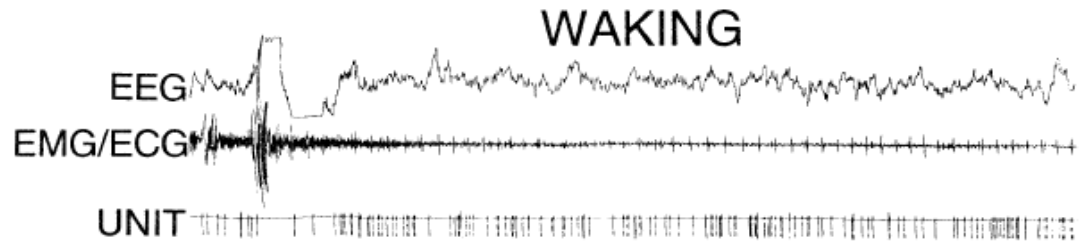
Steriade et al., 1990, J. Neuroscience

**the mysterious echidna, a member of the monotreme order of mammals**



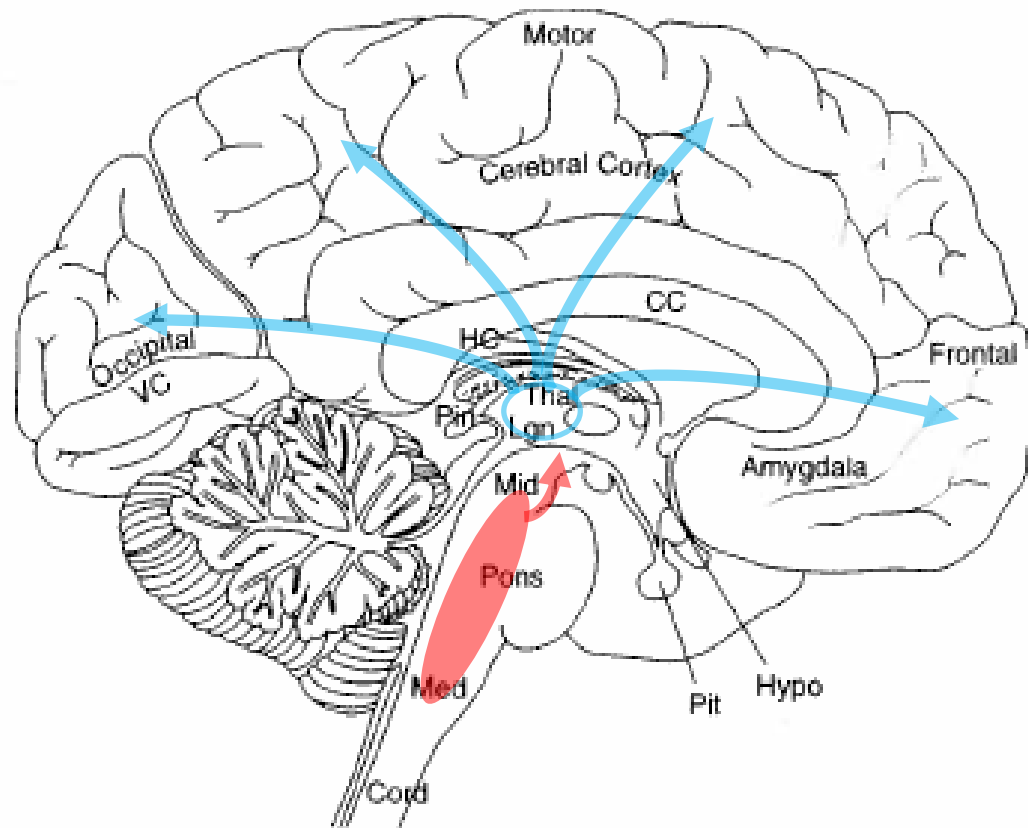
**at first thought to be the only mammal to lack REM sleep**

**later, Siegel shows that echidna non-REM (slow-wave) sleep is punctuated by REM-like brainstem burst events such that sleep in the echidna is actually a mixture of REM sleep and non-REM sleep**

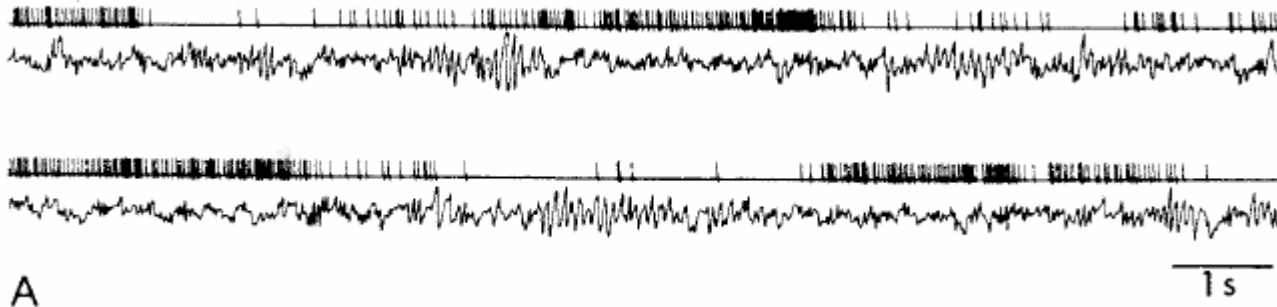


transitions between sleep and wake cortical EEG/LFPs involve interactions between:

- cerebral cortex
- specific thalamic nuclei
- thalamic reticular neurons
- brainstem reticular neurons
- brainstem/basal forebrain neuromodulatory neurons



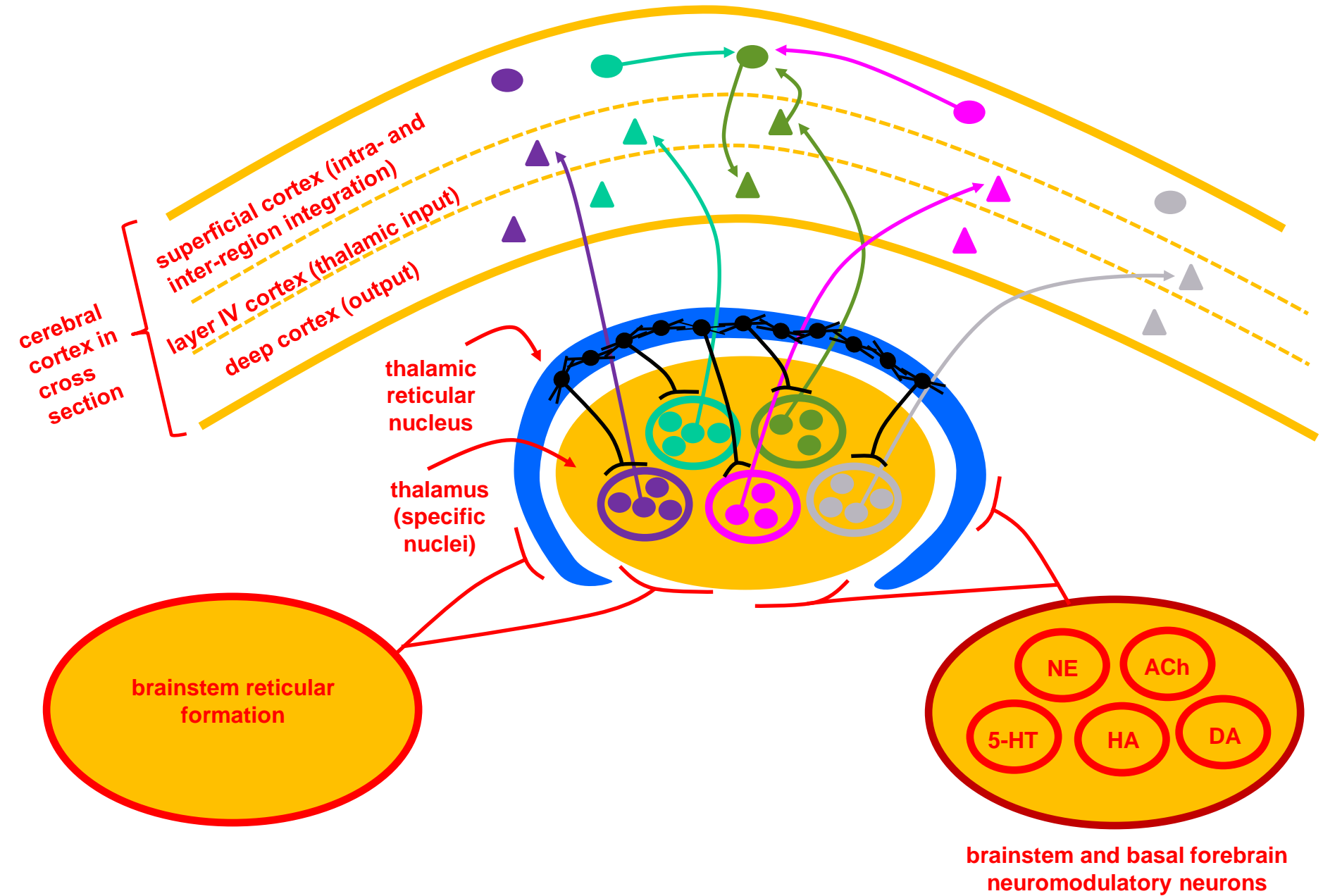
**depressed activity of brainstem reticular formation neurons closely follows the production of spindle and slow-wave cortical EEG events**



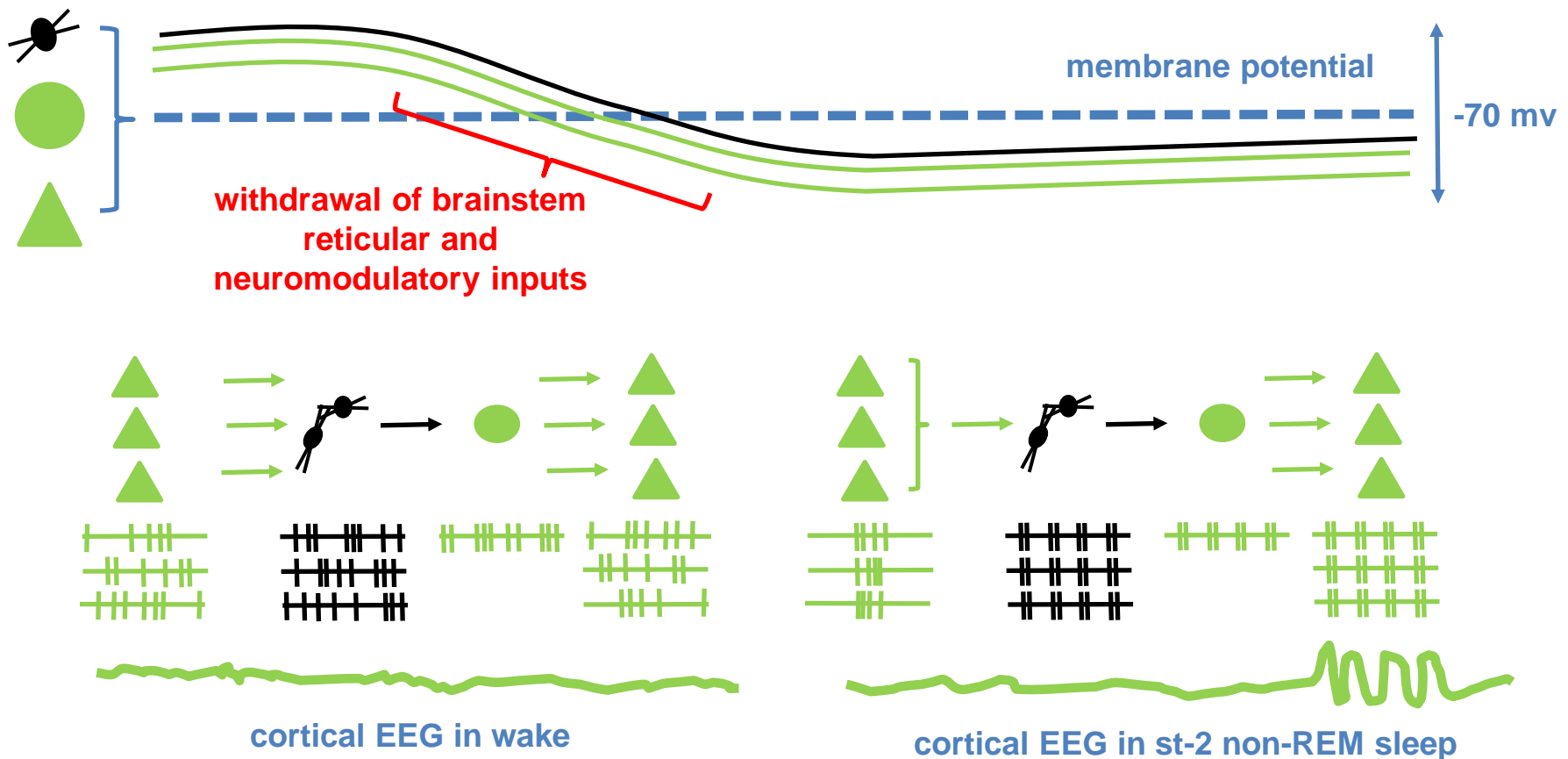
Steriade M (1994). In: Principles and Practice of Sleep Medicine (Kryger MH, Roth T, Dement WC, eds), p 116. Philadelphia: W.B. Saunders, 1994



## the control of cortex-wide activity patterns in schematic



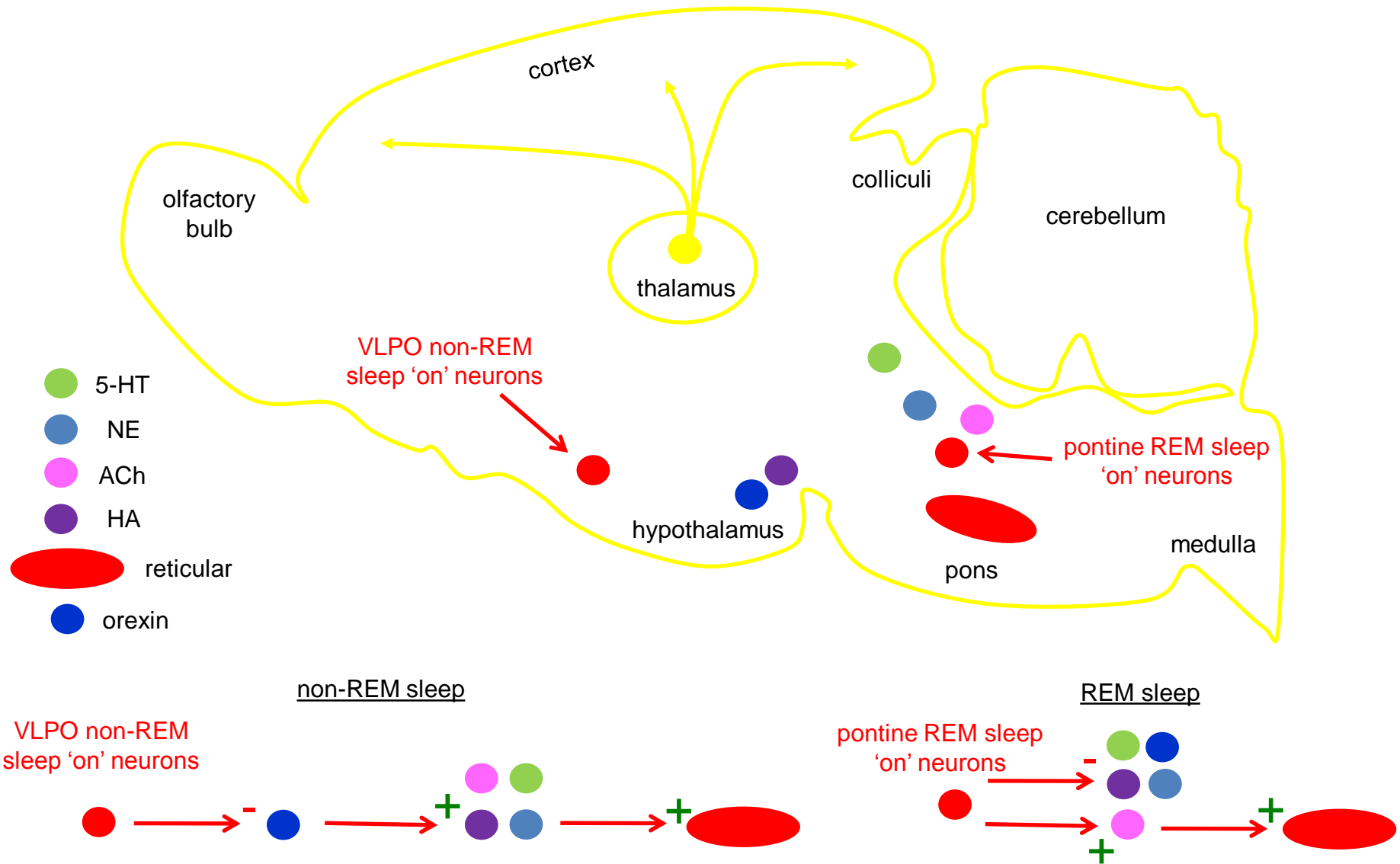
With reduction in depolarizing input from brainstem reticular neurons and neuromodulatory neurons, thalamic neurons respond to excitation by producing spindle oscillations. During non-REM sleep, when such reductions occur, thalamic reticular neurons are excited en masse by cortical neurons. The resulting spindle oscillations of thalamic reticular neurons entrain cortically-projecting thalamic neurons (of the specific nuclei).



**Q: What alters the activity of brainstem reticular neurons and neuromodulatory neurons to yield the patterns of thalamic and cortical activity that accompany wake, non-REM sleep, and REM sleep?**

**A: hypothalamic 'non-REM sleep on' and pontine 'REM sleep on' neurons**

**A: absence of movement and absence of environmental stimuli**



	<u>waking</u>	<u>NREM</u>	<u>REM</u>
cortical EEG / LFP	fast/low-amp/irregular	slow-waves/spindles	fast/low-amp/irreg
trunk muscle tone	high	minimal	absent (paralysis)
eye movements	frequent	none	frequent
heart rate	high/variable	low/regular	high/variable
breathing rate	high/variable	low/regular	high/variable
mentation	vivid	minimal / transient	vivid
hippo. LFP	theta rhythm	slow-waves	theta rhythm
PGO spikes	few	none	frequent
arousal thresholds	low	highest	variable
cortex/thalamus	fast/irregular	slower/burst-pause	fast/irregular
ACh neurons	high rate	lowest rate	highest rate
NE neurons	high rate	very low rate	inactive (REM-off)
5-HT neurons	high rate	low rate	inactive (REM-off)
HA neurons	high rate	very low rate	inactive (REM-off)
DA neurons	moderate rate	moderate rate	moderate rate
VLPO neurons	inactive	highest rates	inactive
REM-on neurons	inactive	inactive	high rate
orexin neurons	high rate	low rate	low rate

\* red coloring denotes major distinctions between wake and REM





