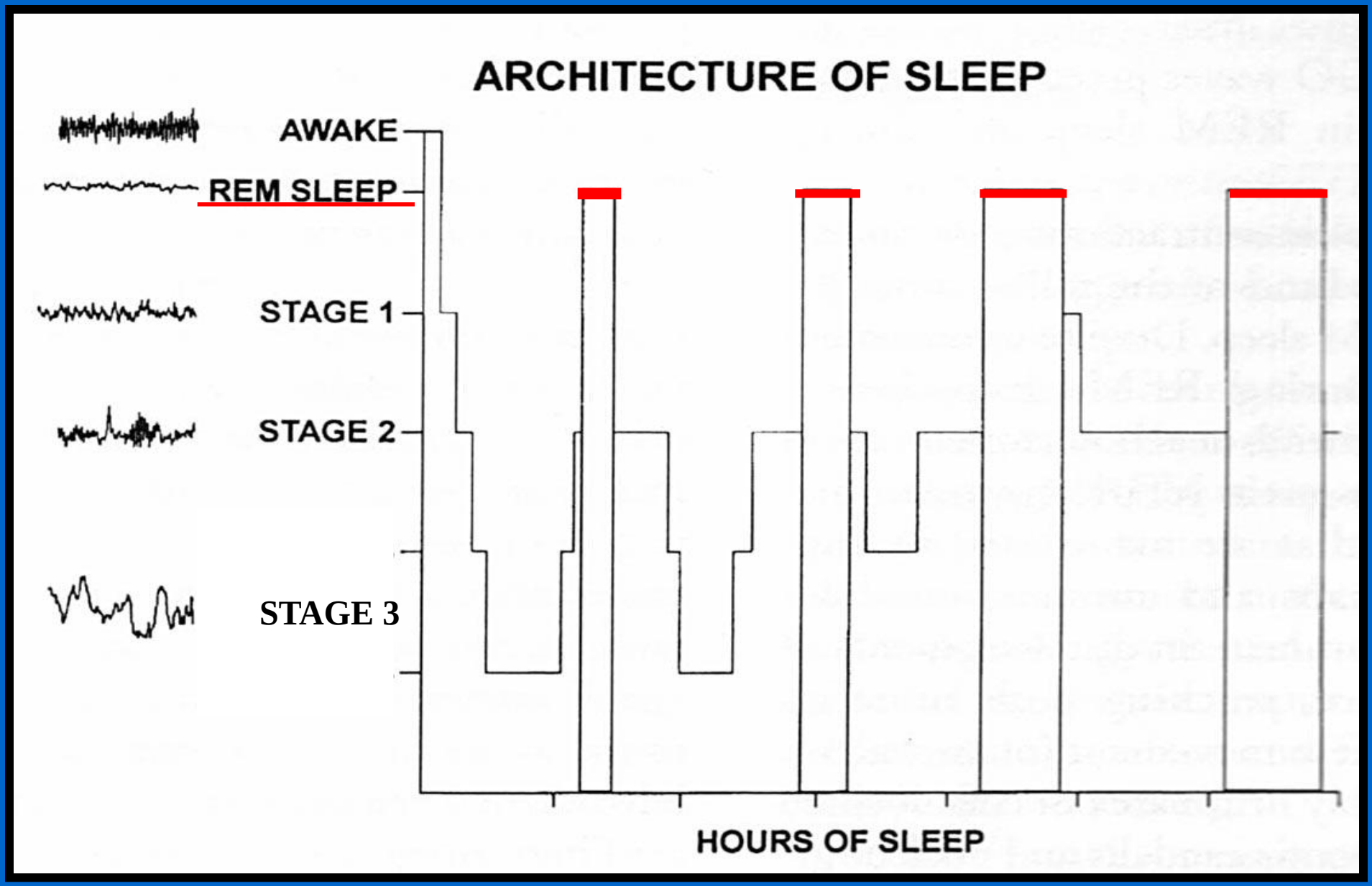
Arquitectura del sueño

- **Sueño no REM** **(75%)**
  - **Sueño superficial: fases N1 y N2** **(55%)**
  - **Sueño profundo: fase N3** **(20%)**
- **Sueño REM** **(25%)**


ARCHITECTURE OF SLEEP
AWAKE
REM SLEEP
STAGE 1
STAGE 2
STAGE 3
HOURS OF SLEEP

Neurotransmisores

	Vigilia	No REM	REM
Ach	⇑	⇓	⇑ ⇑
NA, Ser, His	⇑ ⇑	⇓	0

Hypocretin (orexin) deficiency in human narcolepsy

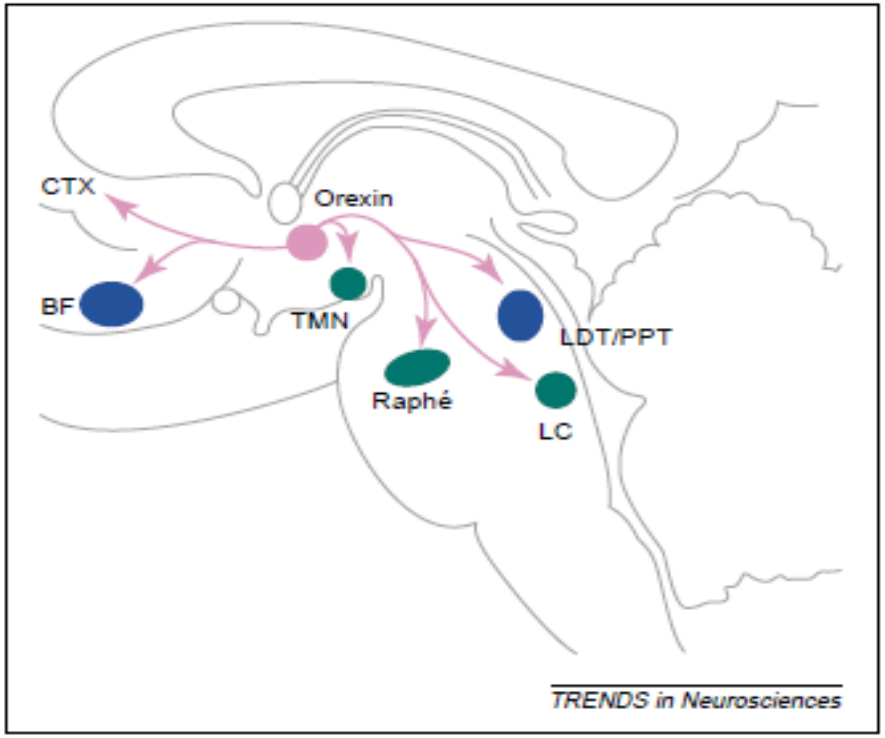
See Commentary p 6

Seiji Nishino, Beth Ripley, Sebastiaan Overeem, Gert Jan Lammers, Emmanuel Mignot

Alterations in the hypocretin receptor 2 and preprohypocretin genes produce narcolepsy in animal models. Hypocretin was undetectable in seven out of nine people with narcolepsy,

analyses were done on
Hypocretin-1 was
inter-individual variat

Lancet 2000



The sleep switch: hypothalamic control of sleep and wakefulness

Clifford B. Saper, Thomas C. Chou and Thomas E. Scammell

Trends 2001

Neurotransmisores

	Vigilia	No REM	REM
Hipocretina	⇑ ⇑	⇓	0
Ach	⇑	⇓	⇑ ⇑
NA, Ser, His	⇑ ⇑	⇓	0

Vol 441|1 June 2006|doi:10.1038/nature04767

nature

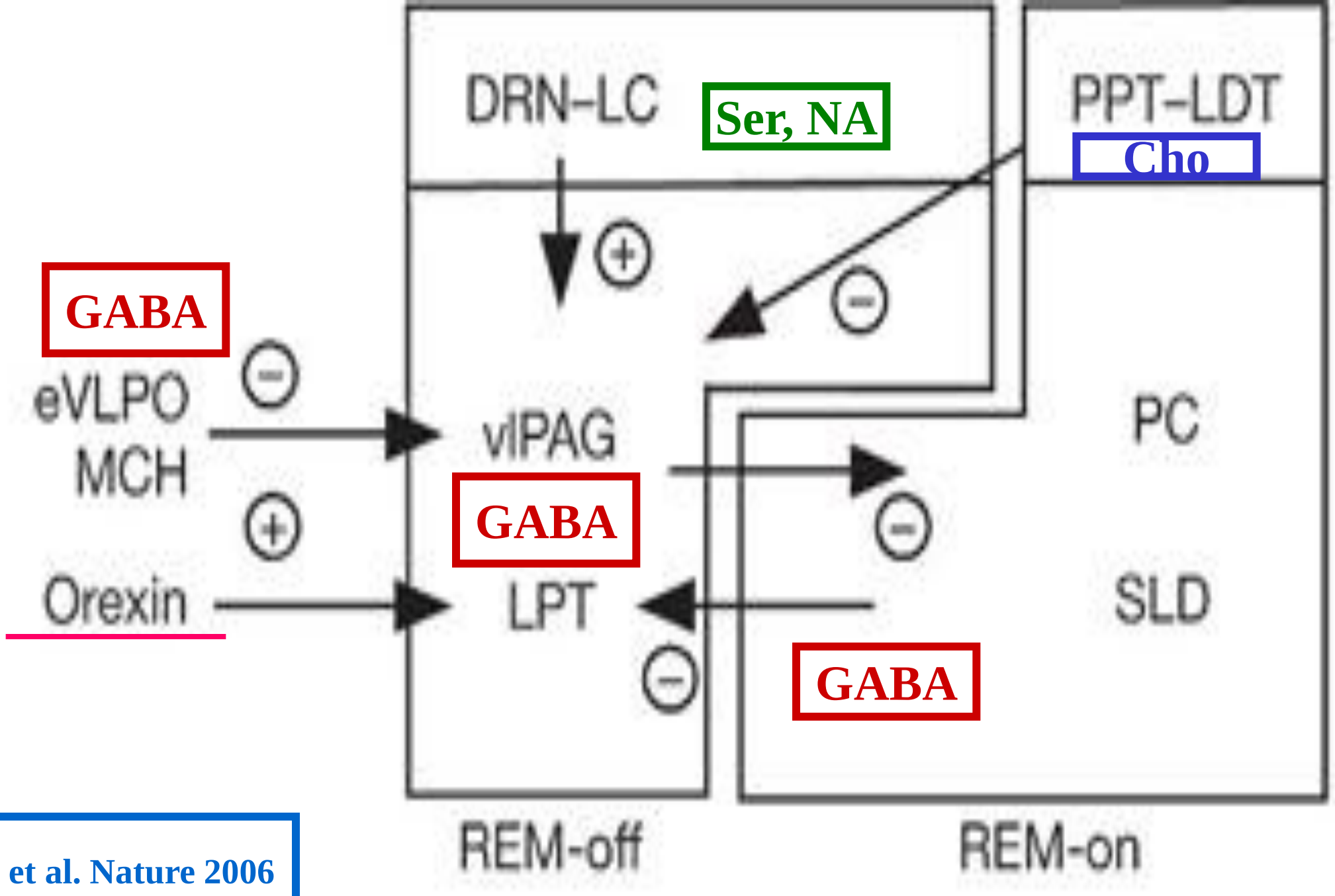
ARTICLES

A putative flip-flop switch for control of REM sleep

Jun Lu[1], David Sherman[1], Marshall Devor[1,2] & Clifford B. Saper[1]

Rapid eye movement (REM) sleep consists of a dreaming state in which there is activation of the cortical and hippocampal electroencephalogram (EEG), rapid eye movements, and loss of muscle tone. Although REM sleep was discovered more than 50 years ago, the neuronal circuits responsible for switching between REM and non-REM (NREM) sleep remain poorly understood. Here we propose a brainstem flip-flop switch, consisting of mutually inhibitory REM-off and REM-on areas in the mesopontine tegmentum. Each side contains GABA (γ-aminobutyric acid)-ergic neurons that heavily innervate the other. The REM-on area also contains two populations of glutamatergic neurons. One set projects to the basal forebrain and regulates EEG components of REM sleep, whereas the other projects to the medulla and spinal cord and regulates atonia during REM sleep. The mutually inhibitory interactions of the REM-on and REM-off areas may form a flip-flop switch that sharpens state transitions and makes them vulnerable to sudden, unwanted transitions—for example, in narcolepsy.

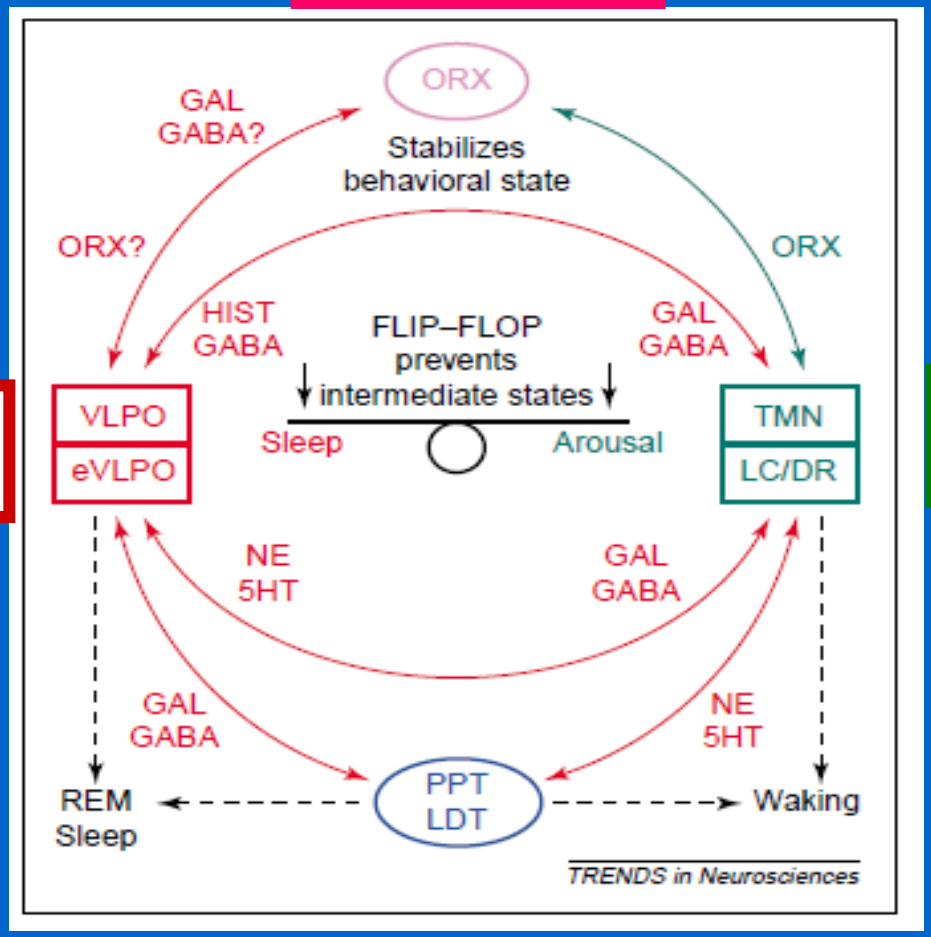
Nature 2006



Lu et al. Nature 2006

Sueño ⇄ **Vigília**

Orexina



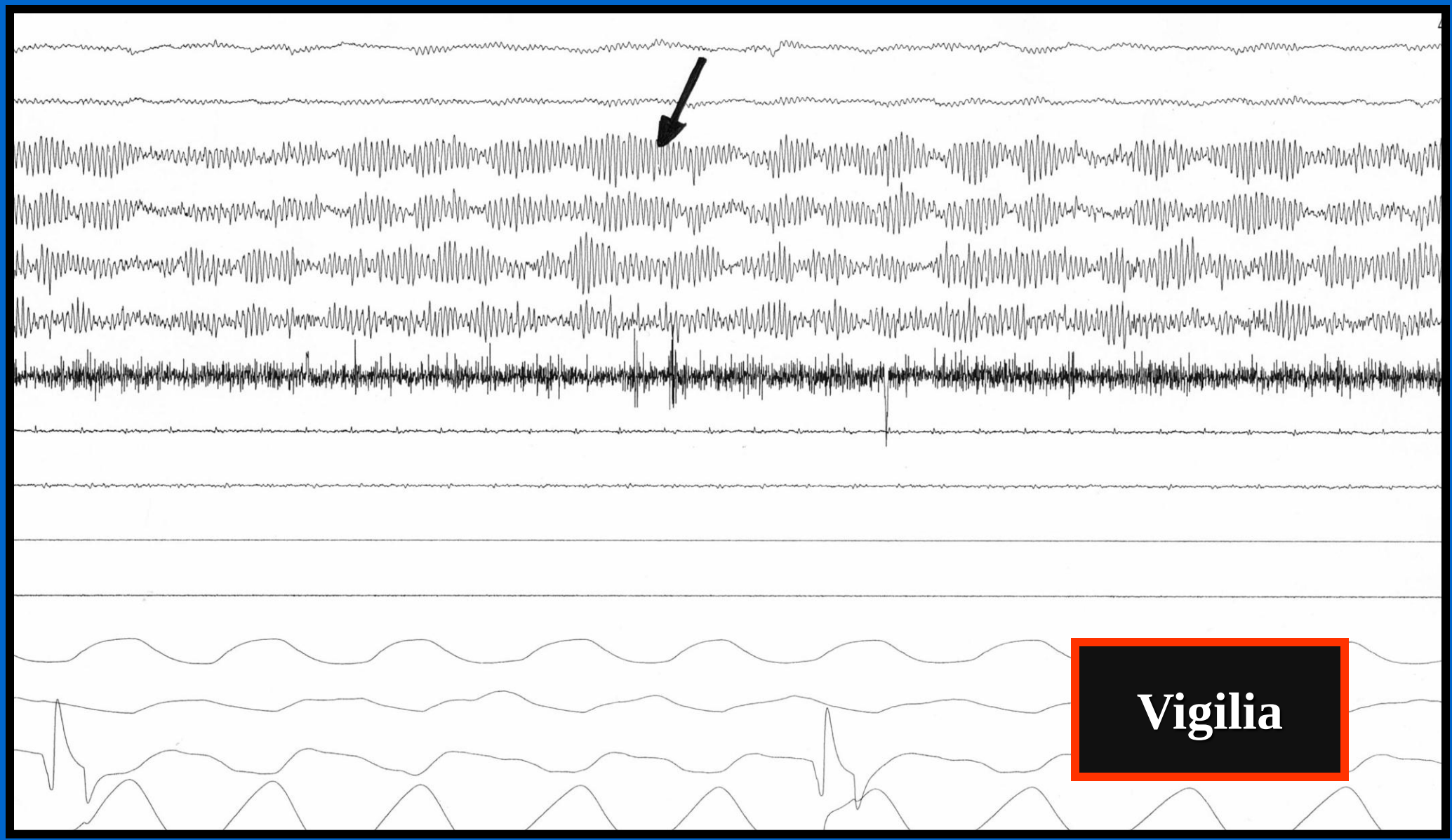
GABA

His, NA, Ser

Ach

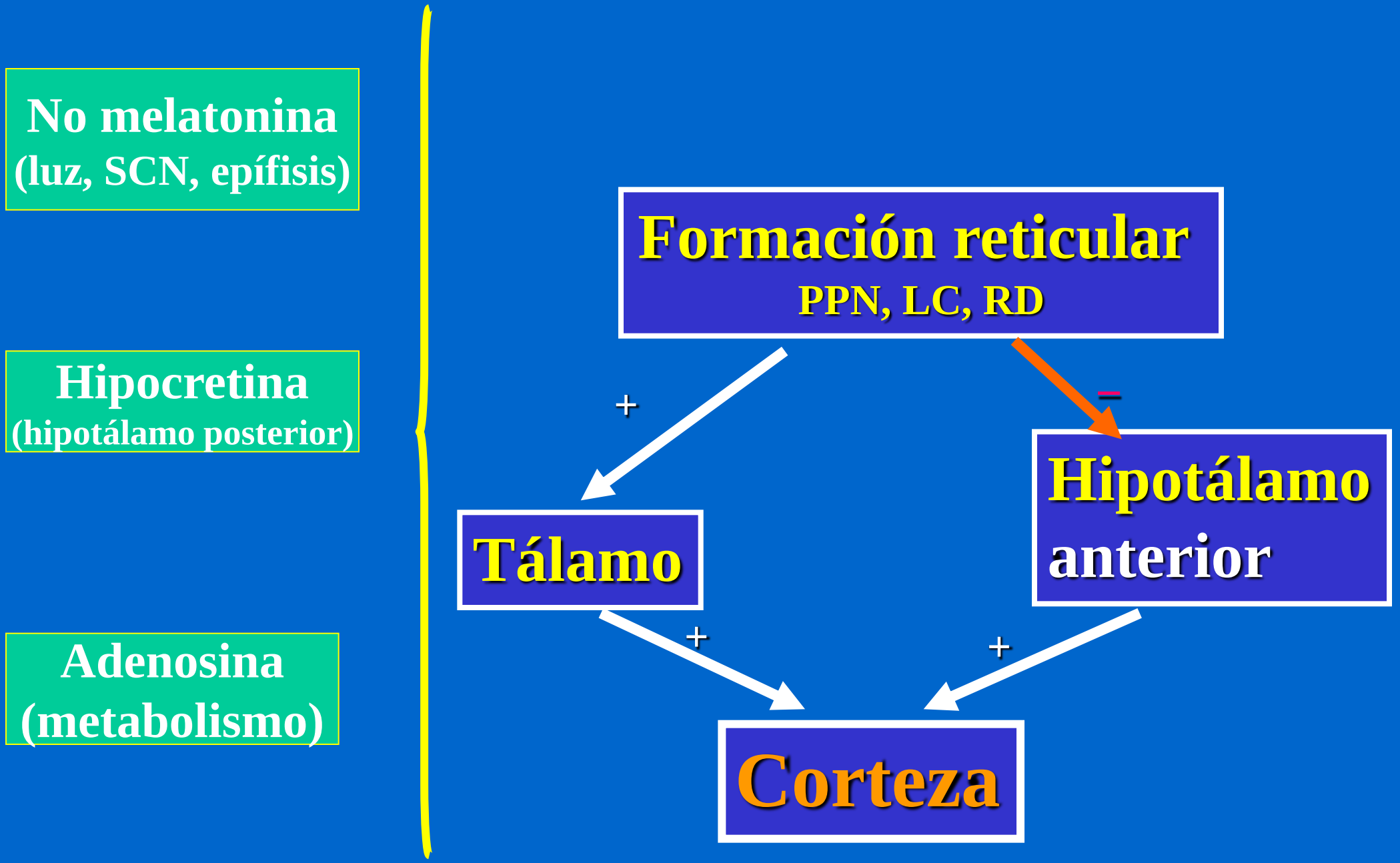
Vigília

- Ritmo alfa
- Tono muscular elevado
- Movimientos oculares rápidos

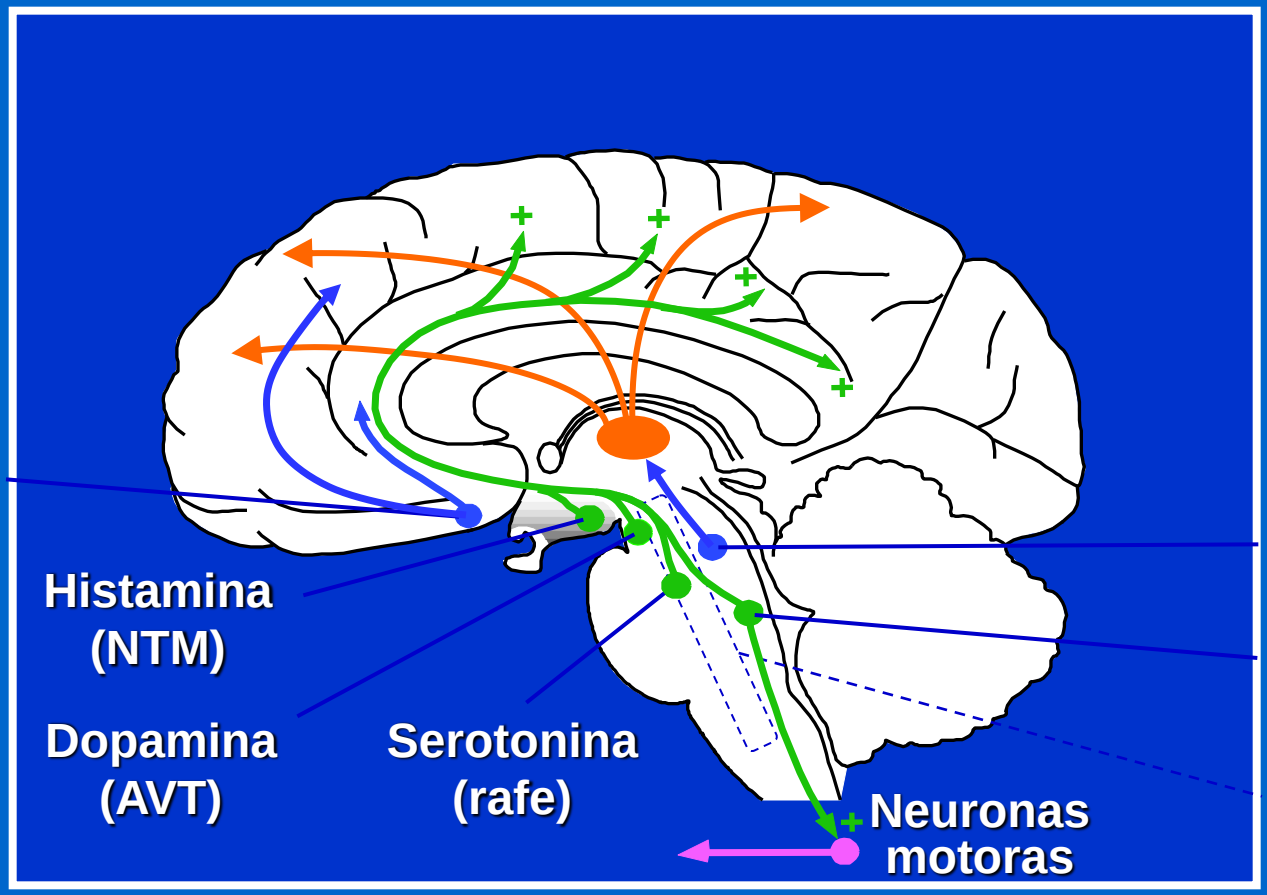

Vigilia

Neurotransmisores

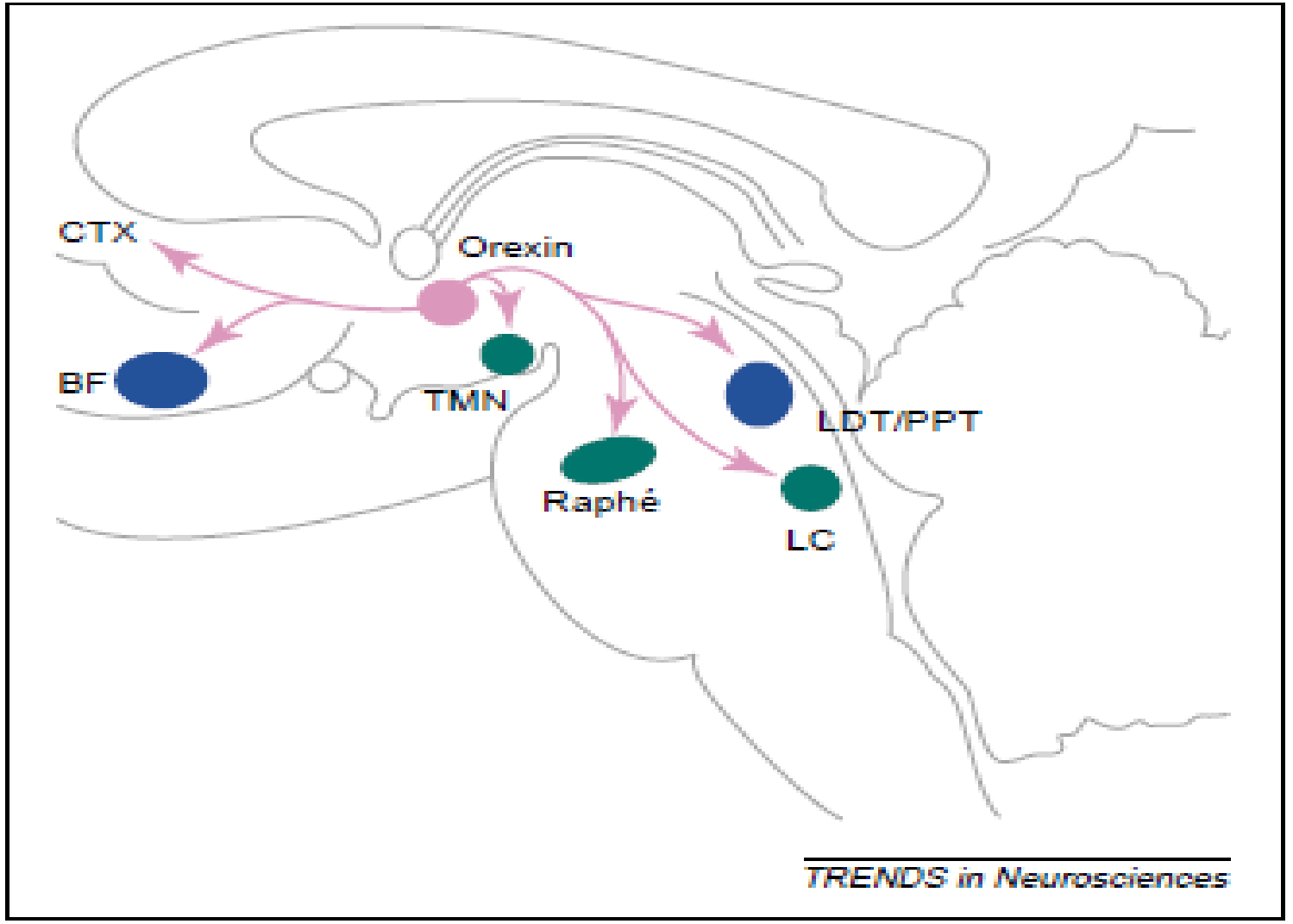
	Vigilia	No REM	REM
Hipocretina	⇑ ⇑	⇓	0
Ach	⇑	⇓	⇑ ⇑
NA, Ser, His	⇑ ⇑	⇓	0


No melatonina
(luz, SCN, epífisis)
Hipocretina
(hipotálamo posterior)
Adenosina
(metabolismo)
Formación reticular
PPN, LC, RD
+
–
Tálamo
Hipotálamo
anterior
+
+
Corteza

Vigília

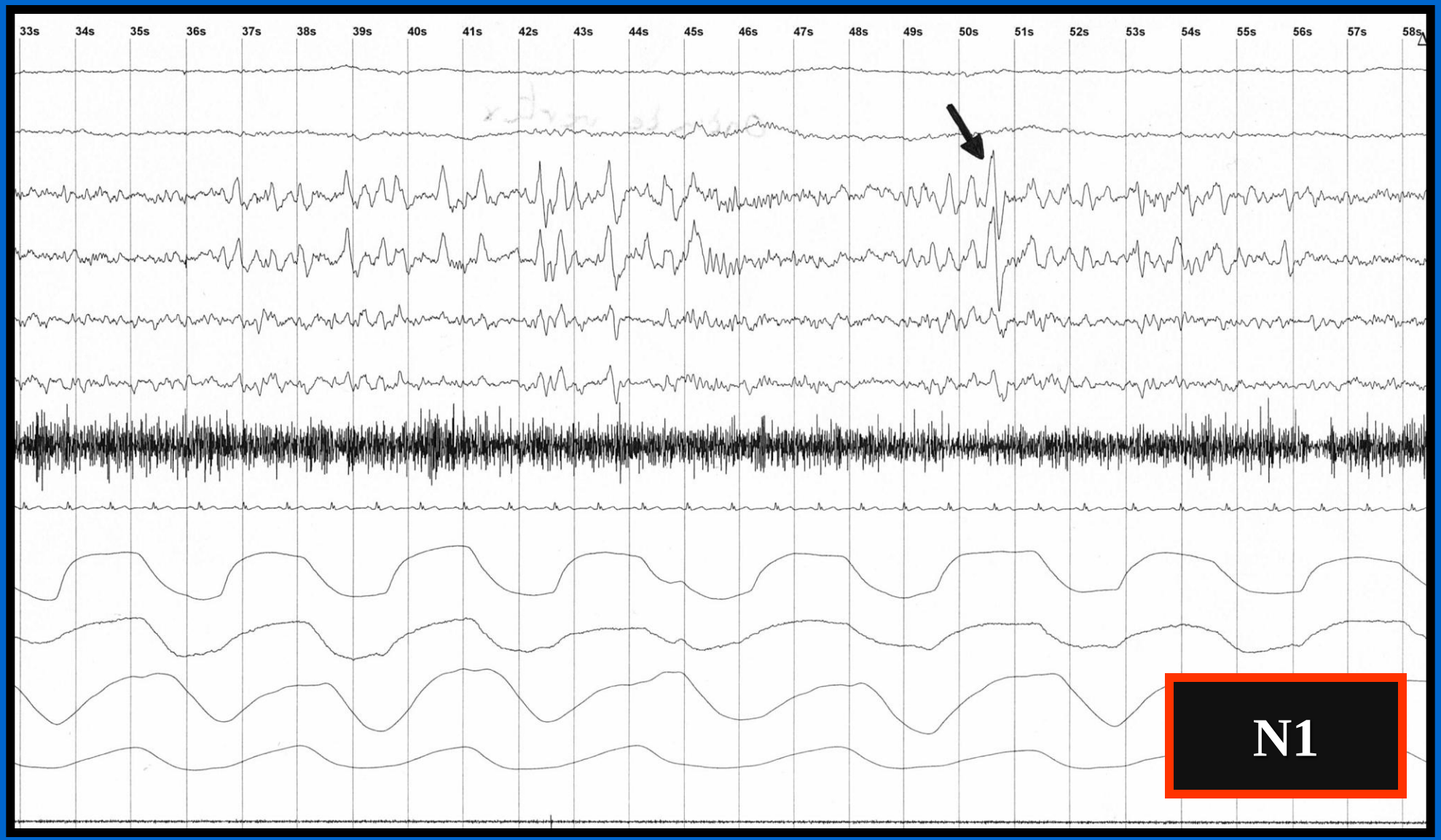


PB = prosencéfalo basal; NTM = núcleo tuberomamilar; AVT= área tegmental ventral; LC = locus coeruleus; PPT = núcleo pedunculopontino; NTLD = núcleo tegmental laterodorsal.


CTX
Orexin
BF
TMN
LDT/PPT
Raphé
LC
TRENDS in Neurosciences

Sueño no REM

- Desaparición ritmo alfa
- Puntas de vértex
- Husos de sueño y complejos K
- Ondas delta de elevada amplitud
- Reducción del tono muscular
- Desaparición de movimientos oculares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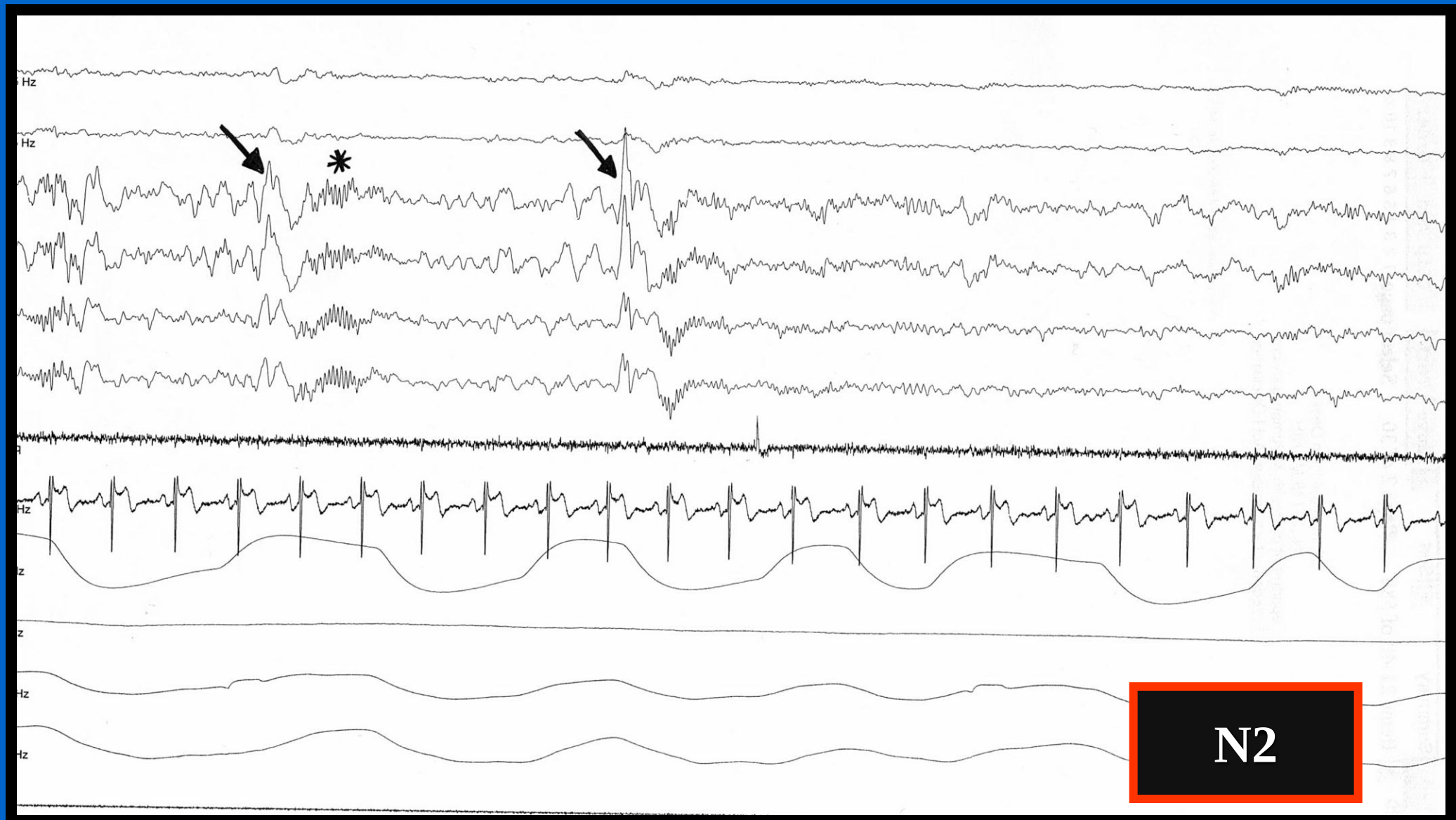
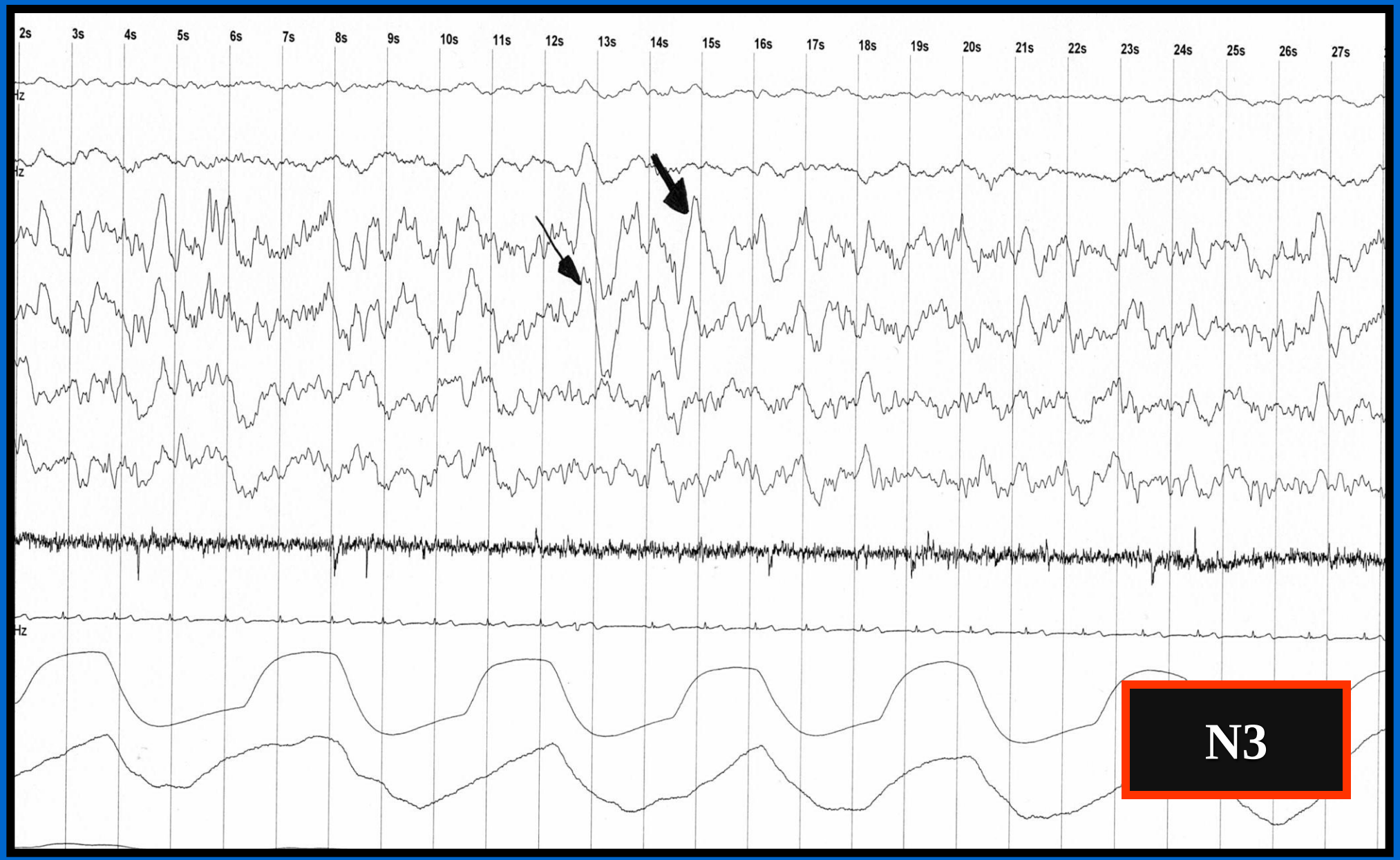
58s
N1


N2


2s
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Hz
Hz
Hz
N3