

# Vagus Nerve Stimulation therapy enhances obstructive respirations during sleep

Jerald H. Simmons, MD <sup>(1,2,3)</sup>, Shaaron Barlow, ANP <sup>(1,2)</sup>

1. Sadler Clinic Sleep Disorders Center, The Woodlands, TX, United States. 2. Comprehensive Sleep Medicine Associates, Houston, TX, United States 3. Sleep Education Consortium, Houston TX

## Introduction:

Vagus Nerve Stimulator (VNS) therapy has been used in medication resistant Epilepsy, with improved outcomes, and utilization has increased over the past decade. VNS therapy utilizes an implantable device in the chest with an electrode connected to a wire extending into the neck that wraps around the left vagus nerve. The device has an adjustable program capability that modifies the characteristics of the stimulation parameters.

In our centers, we have OSA patients—with epilepsy—that have VNS devices and we recognized many of these patients have periodic obstructive breathing patterns that correspond to their VNS activation settings. Other groups (4,5,6) have reported similar experience with VNS therapy. We sought to confirm this association and then to explore if changes in the VNS settings could reduce the induction of obstructive respirations while still providing therapeutic benefits for the control of epilepsy.

## Methods:

We reviewed all of our patients who have both VNS devices for the treatment of their epilepsy and who underwent NPSG testing. Of those who were diagnosed with OSA we retrieved their sleep studies for further review. Re-assessment of the sleep studies were done to identify if there was an apparent association between the VNS stimulation pattern and the occurrence of OSA. We then reviewed the PAP titration studies of those patients with OSA to determine if optimal titration of PAP therapy was achieved and we called back the patients for a clinical update to reassess their progress.

Patients who demonstrated a refractory pattern of obstructive respirations—in spite of positive pressure therapy—returned to the lab for a VNS / PAP titration study. This was a three part process. In the first step: Modifications were made to the VNS settings during NPSG testing to confirm the association between VNS stimulation and OSA occurrence. Step two: The VNS was turned off and the CPAP or BiPAP was modified / titrated to settings that properly prevent obstructive respirations during sleep. Third step: The VNS was turned back on which resulting in a resumption of obstructive respirations, and VNS programming was modified until parameters were identified that no longer induced obstructive respirations. The VNS programming was done while patients' were maintained at their optimal PAP-therapy-level identified during step two (while the VNS was turned off).

## Results:

Eight VNS patients met our criteria of having had a NPSG study. Six of these patients demonstrated OSA. All six demonstrated portions of the study in which the OSA occurred in a pattern that correlated to the VNS stimulation rate. PAP titration was performed on all six. In two of these patients' PAP therapy initially prevented OSA from occurring during sleep but their condition worsened over time. Also, clinically they developed increased daytime sleepiness, not originally thought to be from suboptimal treatment of their OSA. One patient presented with difficulties during the initial titration (which stimulated this investigation).

A VNS/PAP titration was performed on each of the three patients brought in for reassessment. All three demonstrated a clear pattern of periodic OSA that was not adequately treated by PAP therapy.

From the VNS/PAP titration studies it was found that lowering the VNS activation charge provided the best results of reducing the induction of obstructive respirations. Two patients were at 25Hz and one was at 30Hz prior to the VNS/PAP titration. Lowering the stimulation charge down to 10 Hz in two patients and 15 Hz in the third patient was required to achieve this effect.

On clinical follow up, all three patients' demonstrated improvement in their complaint of daytime sleepiness, now at about six months post treatment modification (date of this presentation). One patient had an increase in seizure frequency that was addressed by increasing their dosage of Lamotigine without any side effects.

## Conclusion:

We confirm previous reports that OSA is a potential complication of VNS therapy. This can be managed adequately but must be recognized when present. VNS activation parameters may require adjustment to optimize OSA treatment. We have determined that lowering the stimulation frequency to the range of 10 to 15 Hz minimized the promotion of obstructive respirations by the VNS device. Malow et al (5) have reported a similar observation in one patient.

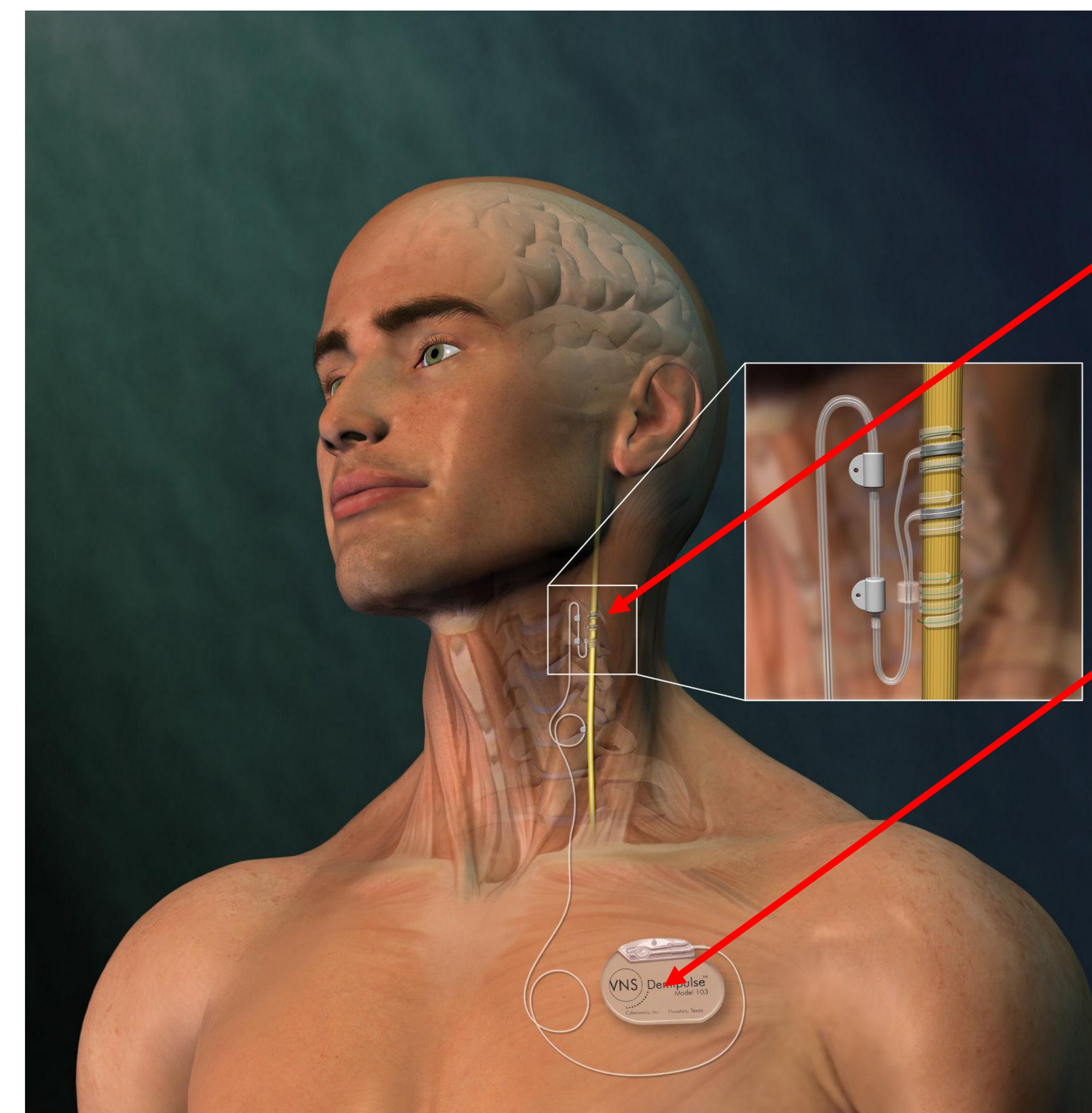
No established recommendations have been previously presented to accommodate the difficulties of VNS therapy when enhancement of OSA occurs. We present here our VNS/PAP titration approach that has thus far worked well in three patients.

It would be advantageous to develop an enhanced VNS device with a variable activation pattern. This variable mode would be different during sleep than those during the awake hours. This would allow for maximum utilization of VNS therapy without precipitating complications such as OSA.

Further study is clearly needed on this topic since VNS is a useful treatment in Epilepsy patients and enhancement of VNS utilization is a worthy endeavour for select clinical conditions.

## References:

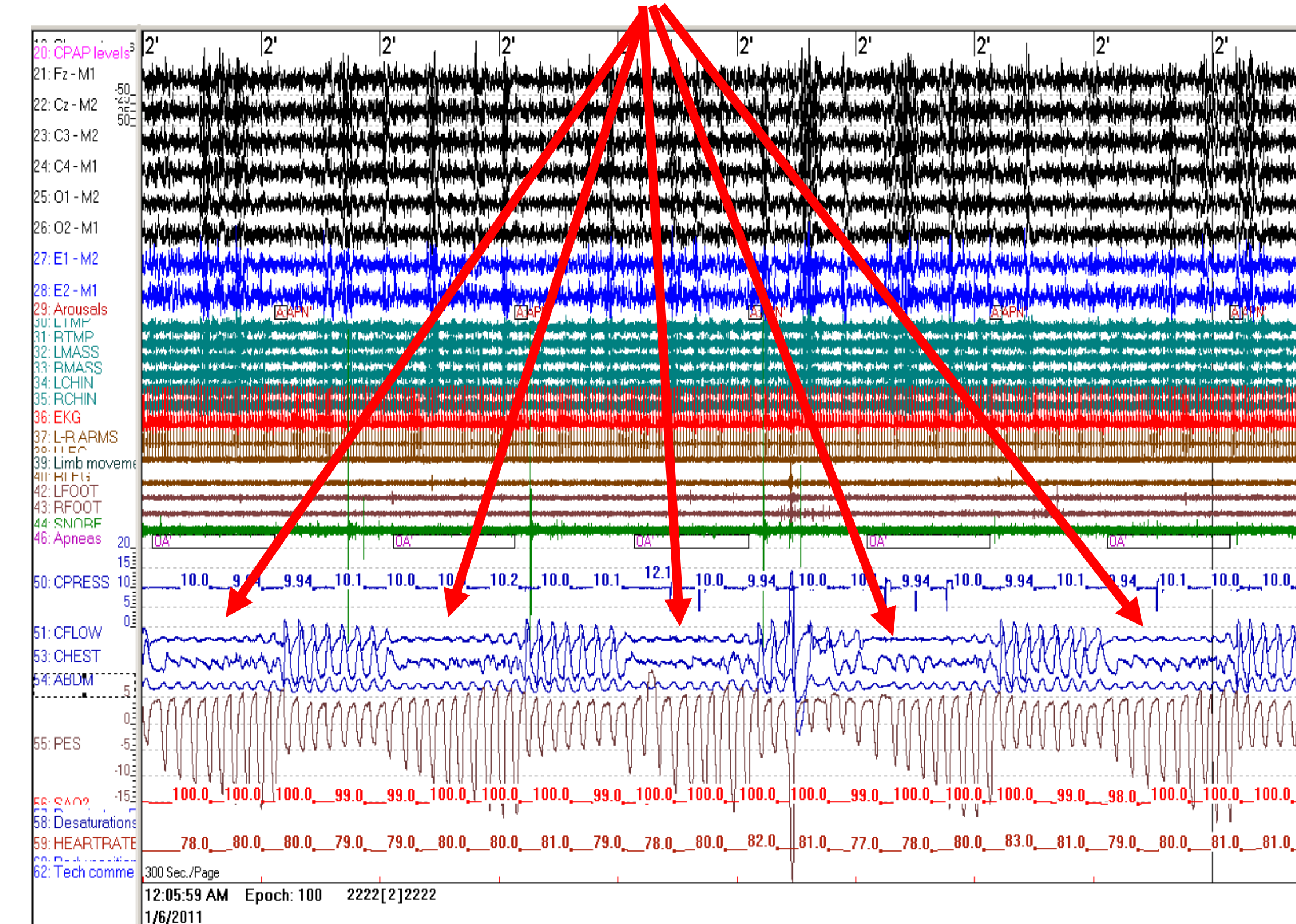
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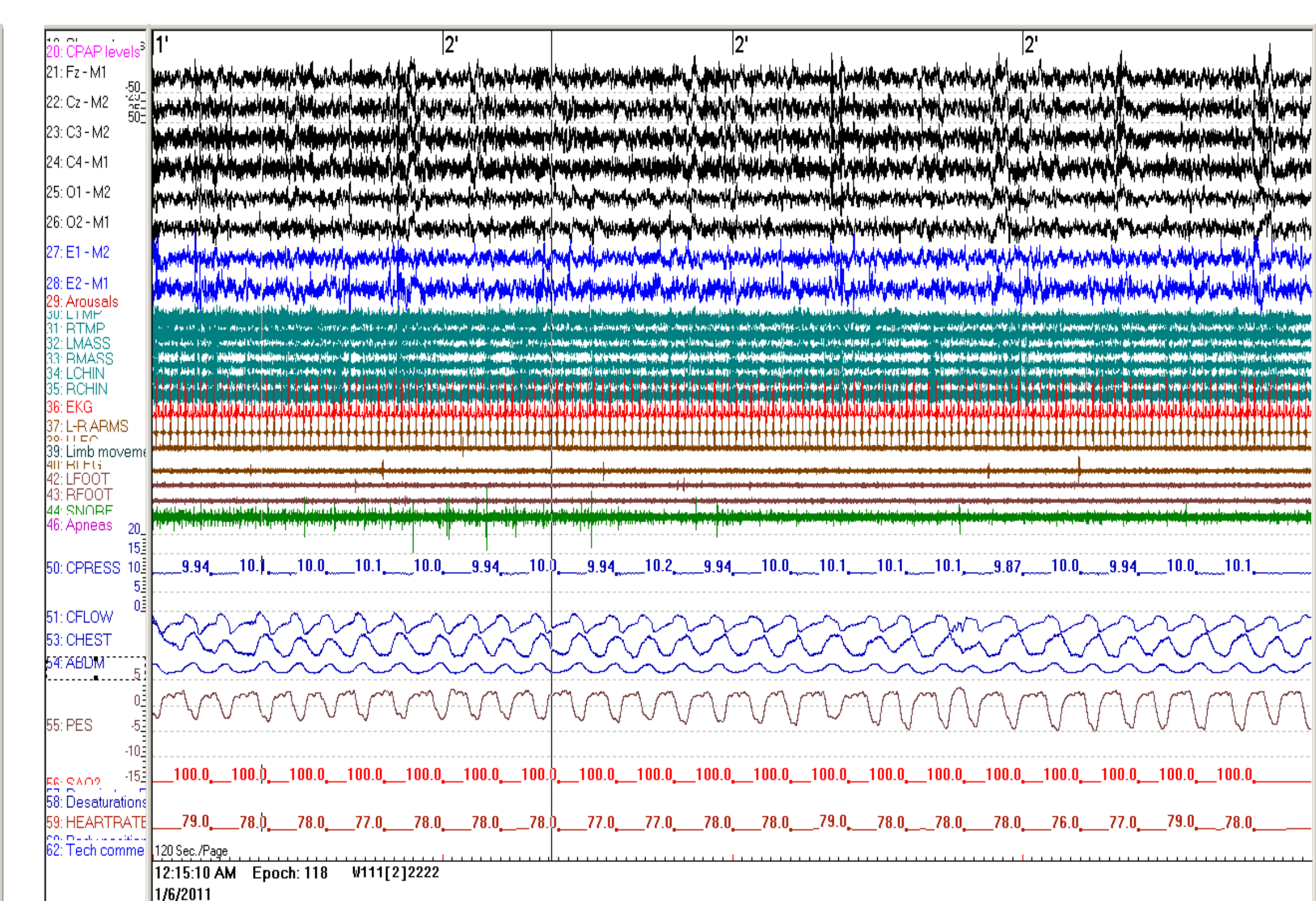
VNS electrode  
Placed on the vagus nerve

VNS Generator device

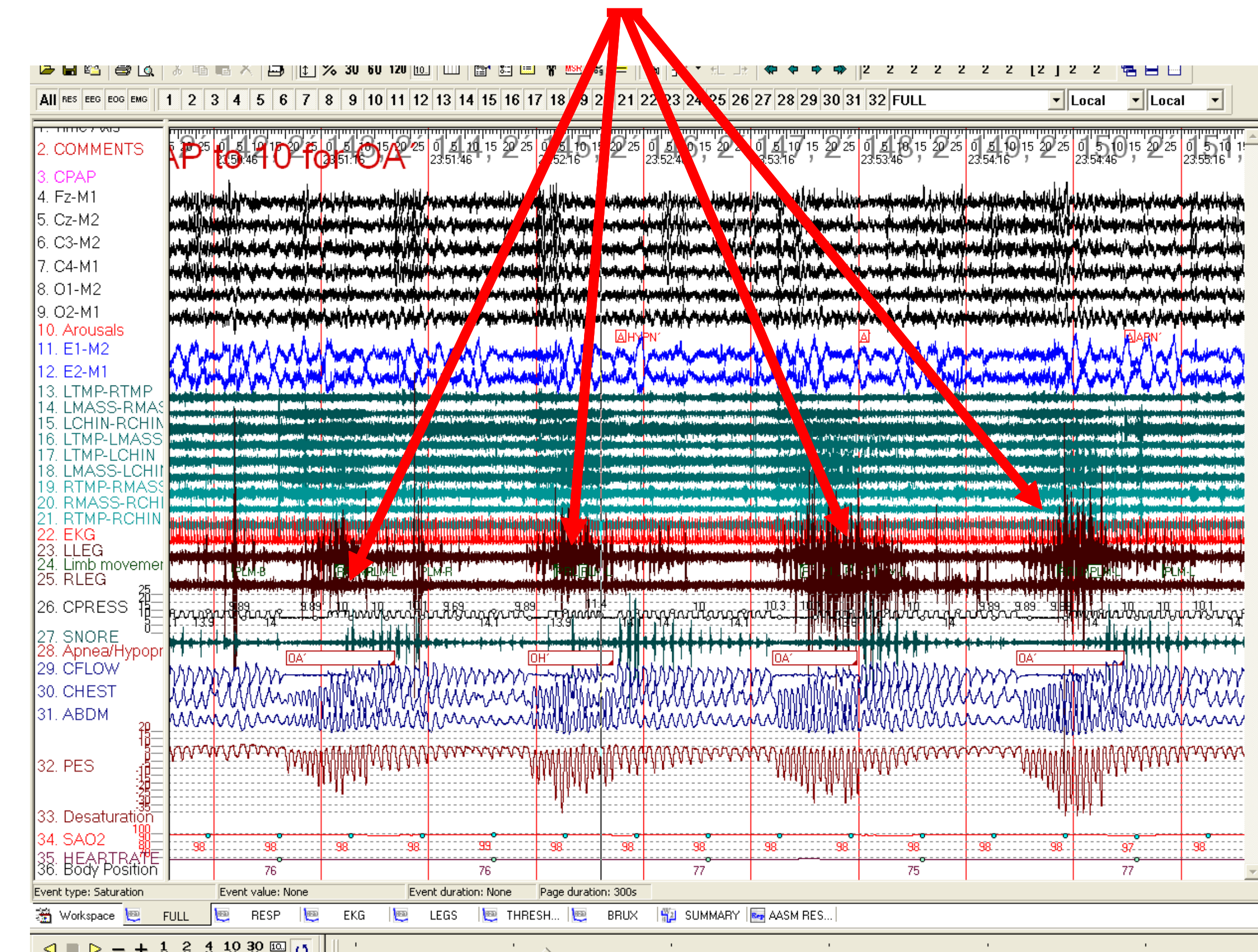
## VNS Activation



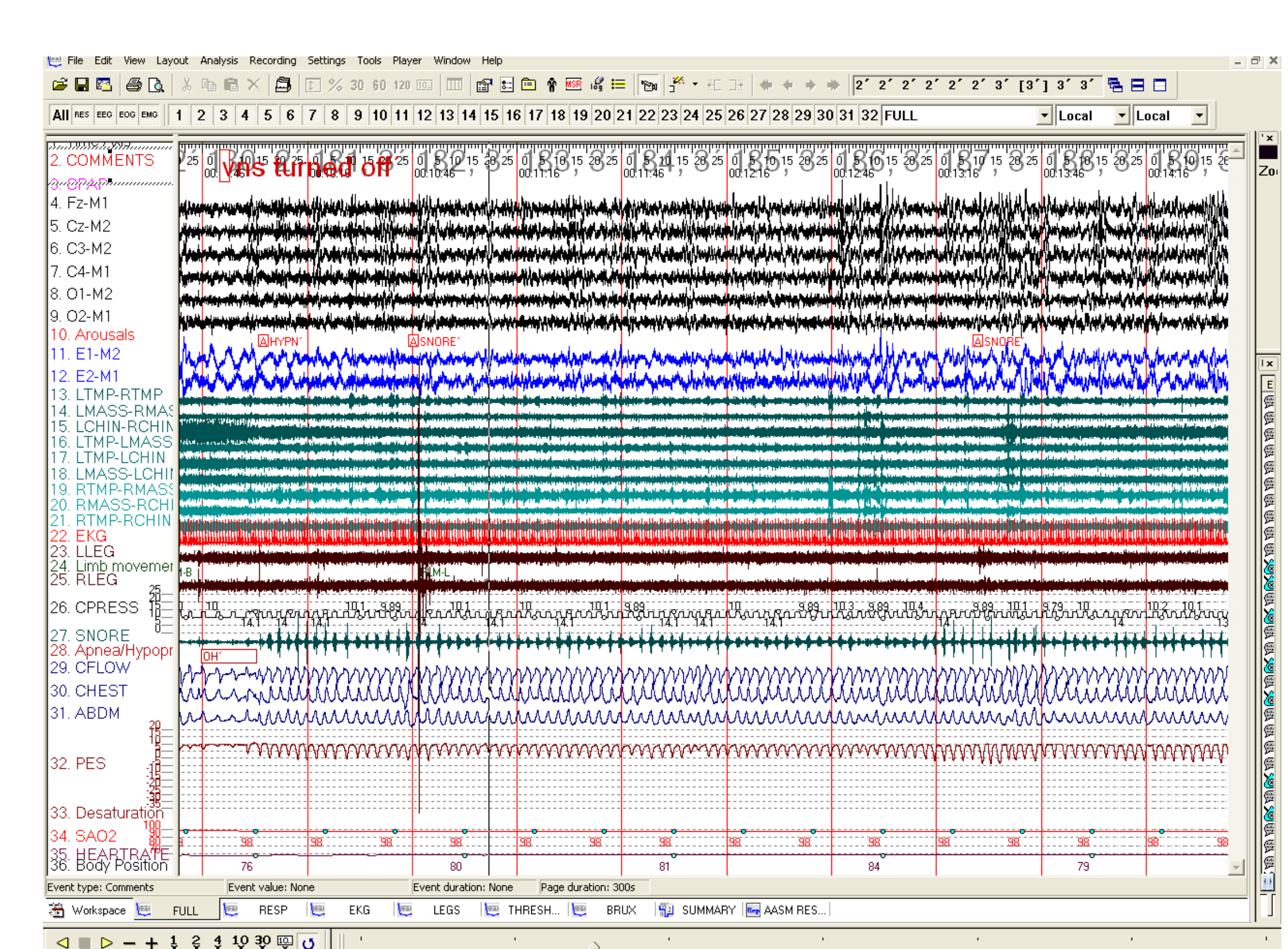
## VNS Turned Off



## VNS Activation



## VNS Turned Off



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