PROCEDURA SELETTIVA, PER TITOLI ED ESAMI, CATEGORIA D, POSIZIONE ECONOMICA D1, AREA SOCIO SANITARIA, CON PROFILO DI TECNICO DI NEUROFISIOPATOLOGIA, PRESSO IL DIPARTIMENTO DI NEUROSCIENZE, RIABILITAZIONE, OFTALMOLOGIA, GENETICA E SCIENZE MATERNO-INFANTILI - DINOGMI, INDETTA CON D.D.G. N. 3386 DEL 3 AGOSTO 2022, PUBBLICATO NELLA G.U. N. 69 - 4^ SERIE SPECIALE - DEL 30 AGOSTO 2022

Adempimenti di cui all'art. 19 del D.lgs. n. 33/2013, come modificato dall'art. 18 del D.lgs. n. 97/2016

QUESITI PROVA ORALE

Il giorno **24.02.2023, a partire dalle ore 13.00, presso l'Aula Magna del Dipartimento di neuroscienze, riabilitazione, oftalmologia, genetica e scienze materno- infantili (DINOGMI),** Largo Paolo Daneo 3, piano terra, ha luogo la sesta riunione della Commissione esaminatrice della procedura di cui al titolo per lo svolgimento della prova orale.

La Commissione, in conformità a quanto deciso nella prima seduta, determina i quesiti e predispone i brani per l'accertamento della conoscenza della lingua straniera da porre alle candidate che vengono di seguito trascritti:

Traccia n. 1

- Quesito 1: Ruolo del tecnico di neurofisiopatologia nel percorso diagnostico del paziente con sonnolenza ed episodi di caduta a terra di dubbia interpretazione;
- Quesito 2: Descrivi le epoche di polisonnografie seguenti (allegato n.2);
- Quesito 3: Leggi, traduci e commenta la pagina seguente (allegato n.3);

Traccia n. 2

- Quesito 1: Ruolo del tecnico nel percorso diagnostico del bambino con disturbi parossistici in sonno;
- Quesito 2: Descrivi l'episodio parossistico documentato in un NAP pomeridiano (allegato n.4);
- Quesito 3: Leggi, traduci e commenta la seguente pagina (allegato n.5);

Traccia n. 3

- Quesito 1: Ruolo del tecnico di neurofisiopatologia nella diagnosi del bambino con disturbi dell'inizio e del mantenimento del sonno;
- Quesito 2: Osserva e commenta le pagine seguenti (allegato n.6);
- Quesito 3: Leggi, traduci e commenta la pagina seguente (allegato n.7);

Traccia n. 4

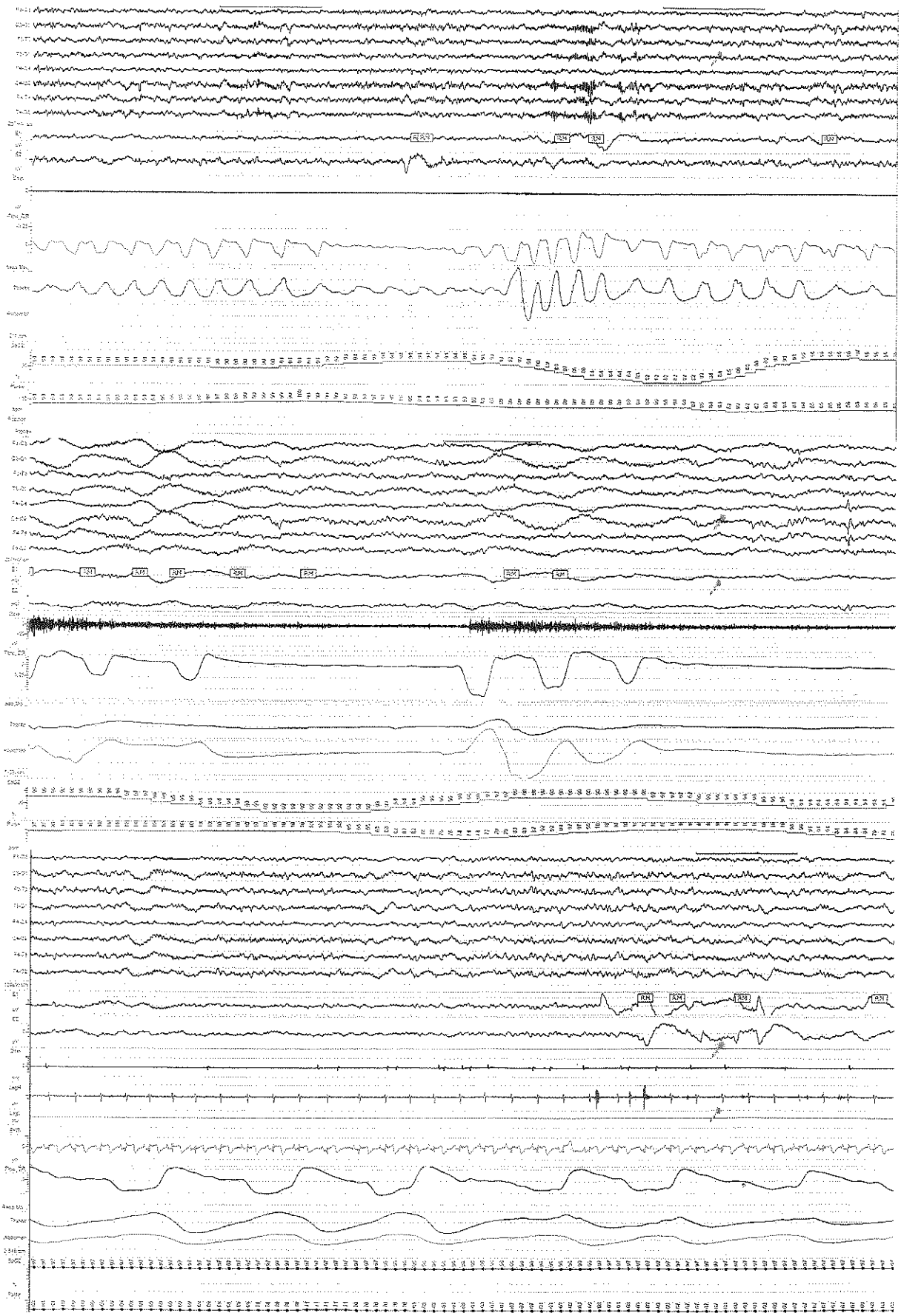
- Quesito 1: Ruolo del tecnico di neurofisiopatologia nello studio dell'ipersonnia dell'età evolutiva;
- Quesito 2: Osserva e descrivi le pagine seguenti (allegato n.8);
- Quesito 3: Leggi, traduci e commenta la pagina seguente (allegato n.9);

Genova, 24.02.2023

La Commissione:

-	Firmato	Prof. Lino Nobili	Presidente
-	Firmato	Dott.ssa Simona Martelli	Componente
-	Firmato	Dott. Francesco Famà	Componente
-	Firmato	Dott. Antonio Cogliandro	Segretario

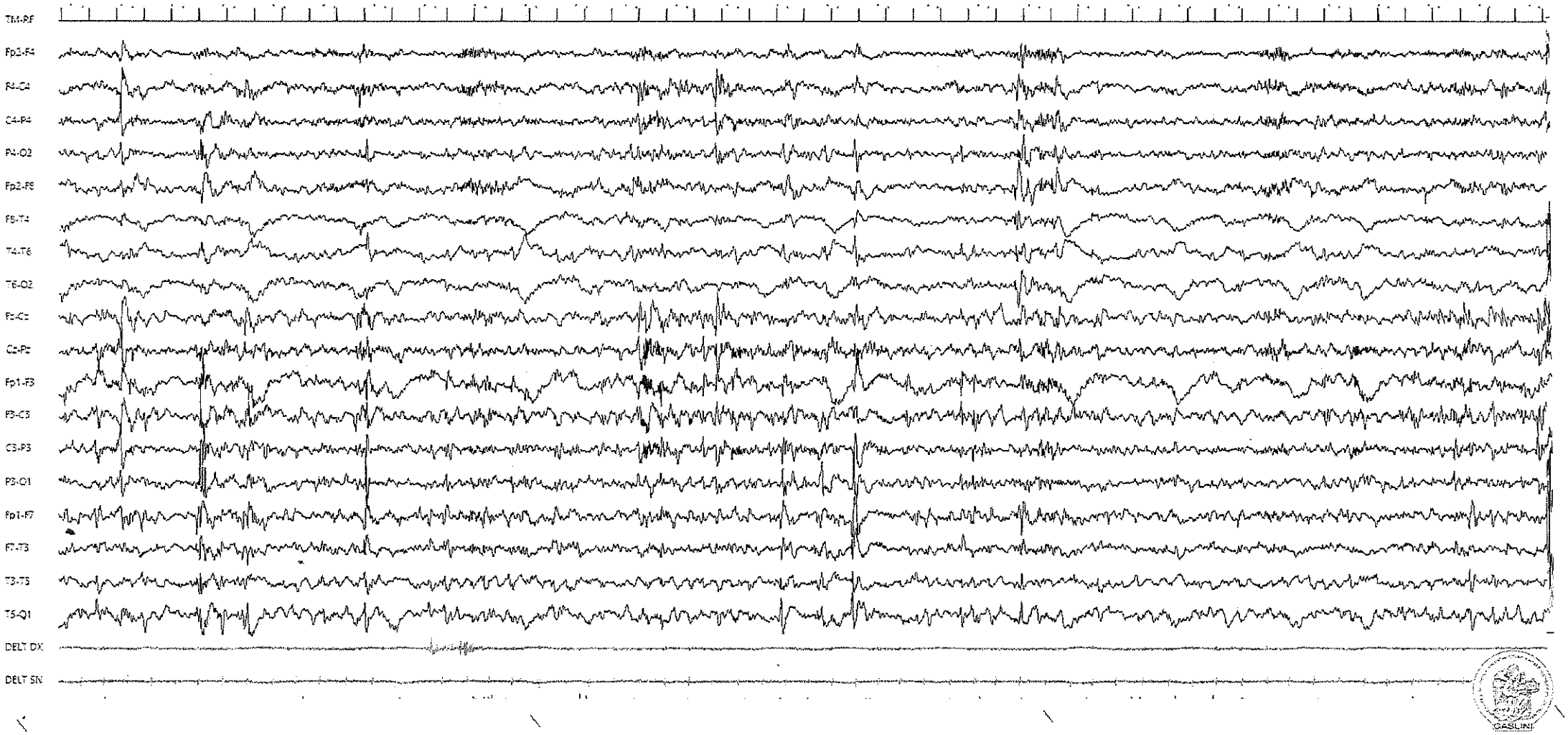
ALLEGATO 2



ALLEGATO 3

1) **Sleep has a significant effect on epilepsy, with NREM sleep facilitating and REM sleep inhibiting** epileptic activity (Ng & Pavlova, 2013; Shouse, Farber, & Staba, 2000). Evidence for the effects of sleep on epilepsy is not only present for sleep architecture but also for its microstructure. Analysis of the cyclic alternating pattern (CAP), an EEG marker of unstable sleep, has shown that epileptic activity is not uniformly increased during NREM sleep, but that enhanced epileptic activity is associated with CAP A1 subtypes that consist of recurrent EEG bursts of slow-wave activation (Parrino, Smerieri, Spaggiari, & Terzano, 2000). Subsequently, the role of slow waves known to orchestrate physiological brain rhythms was investigated in epilepsy (Steriade, 2006). Isolated high-amplitude slow waves were found to be the main driver of interictal epileptic activity during NREM sleep (Frauscher et al., 2015a), likely mediated by EEG hypersynchronization and by the presence of a bi-stable state typical of slow oscillations (Steriade, 2006). On the other hand, in some epileptic conditions, the distribution of interictal epileptiform discharges (IEDs) follows the dynamics of spindle frequency activity throughout the night (Ferrillo, Beelke, & Nobili, 2000; Zubler, Rubino, Lo Russo, Schindler, & Nobili, 2017). In contrast, it was shown that REM sleep with (phasic) and without (tonic) rapid eye movements has distinct suppressive impact on interictal epileptic activity, with the most inhibiting effect being present during phasic REM sleep where EEG desynchronization is maximum (Campana et al., 2017; Frauscher, von Ellenrieder, Dubeau, & Gotman, 2016). Apart from these well-documented relationships, the effect of arousals on sleep and epilepsy remains a crucial issue to be investigated. In particular, it has been shown that epileptic activity (Peter-Derex et al., 2020; Terzano, Parrino, Anelli, & Halasz, 1989), as well as physiological, paraphysiological and pathological motor events share a common trait of arousal-activated phenomena (Parrino, Halasz, Tassinari, & Terzano, 2006).

ALLEGATO 4


TM-PF
Fp2-F4
F4-C4
C4-P4
P4-O2
Fp2-F8
F8-T4
T4-T6
T6-O2
Fz-Cz
Cz-Pz
Fp1-F3
F3-C3
C3-P3
P3-O1
Fp1-F7
F7-T3
T3-T5
T5-O1
DELT DX
DELT SN

FP2-REF
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O2-REF
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T4-REF
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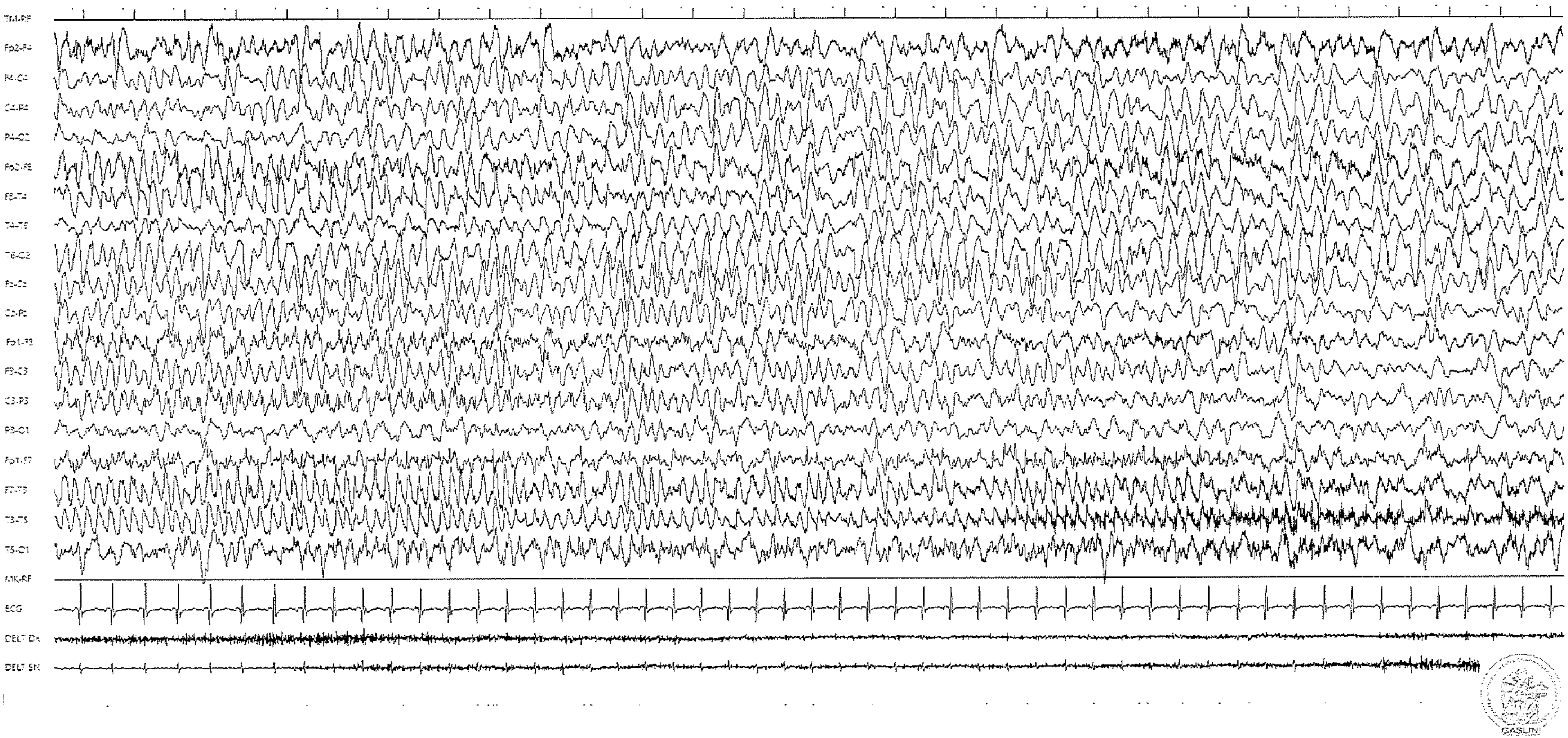
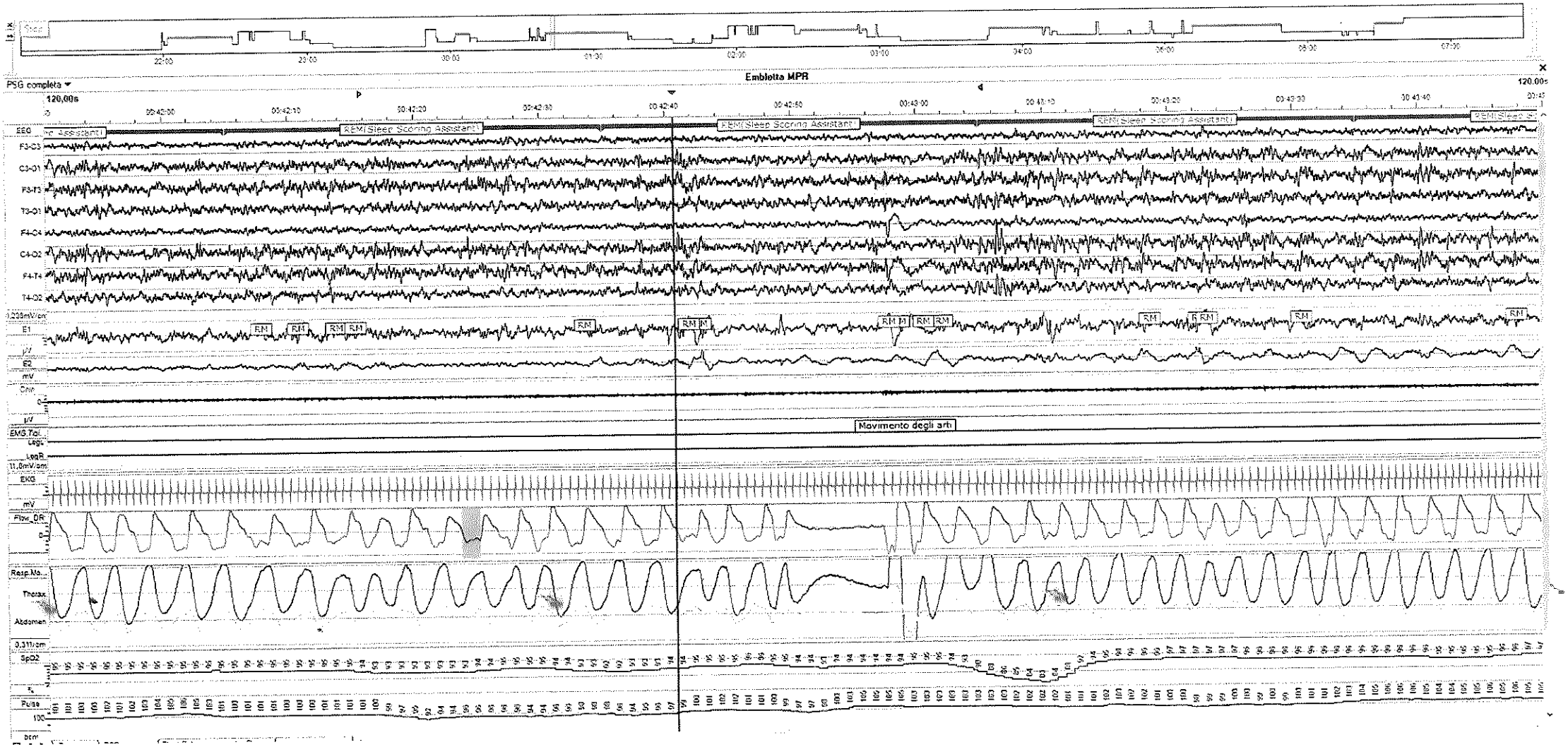
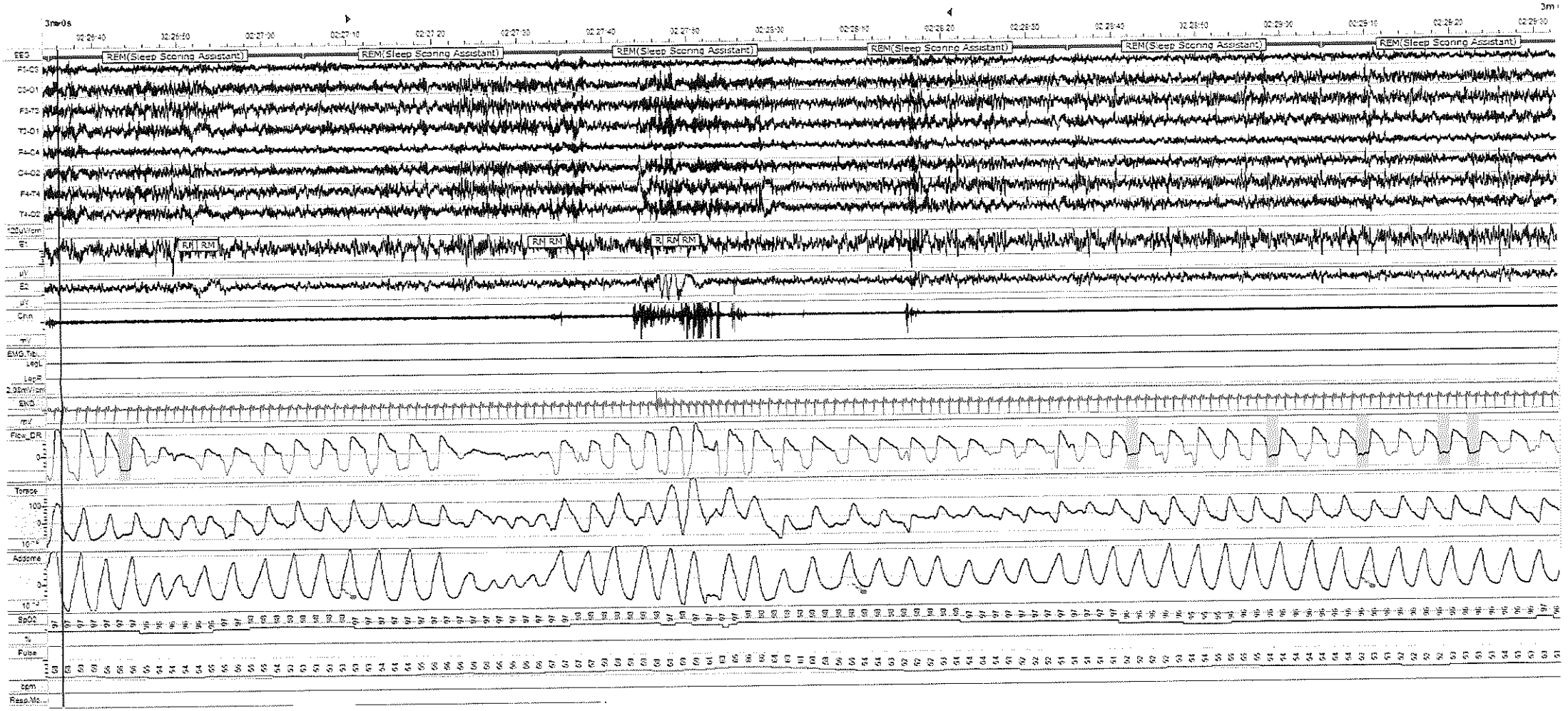

Fp2-F4
F4-C4
C4-P4
P4-O2
Fp2-F8
F8-T4
T4-T6
T6-O2
Fz-Cz
Cz-Pz
Fp1-F3
F3-C3
C3-P3
P3-O1
Fp1-F7
F7-T3
T3-T5
T5-O1
ECG
DELT Dx
DELT SN
GASLINI

ALLEGATO 5

Epilepsy is associated with changes in sleep macro- and microstructure (Sudbrack-Oliveira, Lima Najar, Foldvary-Schaefer, & da Mota Gomes, 2019). These changes are multifactorial, given that epilepsy is not only seizures but rather a complex, multidimensional condition regarding the underlying pathology, neuropsychiatric and sleep comorbidities, and effects of pharmacological and non-pharmacological treatments (Fisher et al., 2014; Liguori, Toledo, & Kothare, 2021; Romero-Osorio, Gil-Tamayo, Nariño, & Rosselli, 2018). However, apart from these manifold factors, evidence suggests that epileptic activity has a direct impact on sleep architecture, sleep continuity and sleep oscillations. Increased wake after sleep onset is the strongest feature observed in patients with epilepsy (PWE; Crespel, Coubes, & Baldy-Moulinier, 2000; Dell et al., 2021; Parrino et al., 2012; Peter-Derex et al., 2020), especially during nights with clinical manifestations. It may result in part from the awakening effect of certain seizures, not only generalized tonic–clonic seizures but also focal, and even paucisymptomatic seizures (Awad & Lüders, 2010; Manni et al., 1997; Yildiz, Tezer, & Saygi, 2012). Seizure-associated changes in sleep parameters also include a decrease in REM sleep quantity and a delay in the first REM sleep episode (Bazil, Castro, & Walczak, 2000; Dell et al., 2021). Sleep architecture disruption is observed at the microstructural level as well. Generalized interictal discharges are associated with alterations in NREM sleep stability as evident by an increased amount of CAP rate and a longer duration of CAP cycles (Terzano, Parrino, Anelli, Boselli, & Clemens, 1992).

ALLEGATO 6


Embletta MPR
PSG completa
REM Sleep Scoring Assistant
Movimento degli arti
EEG
EOG
EMG Tot.
ECG
Flow_OR
Resp Mo...
Thorax
Abdomen
SpO2
Pulse


REM(Sleep Scoring Assistant)
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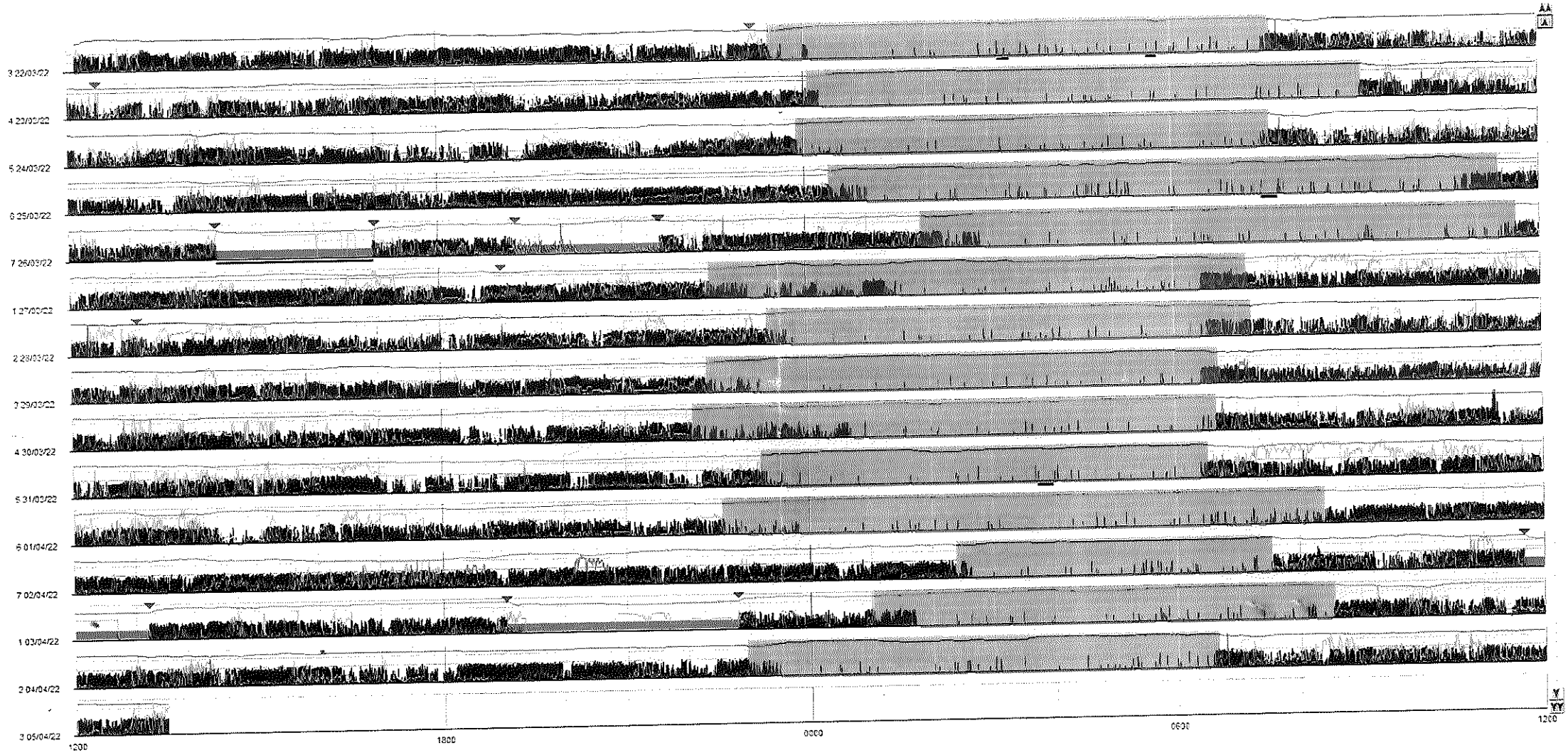
C3-O1
F3-T3
T3-O1
F4-C4
C4-O2
F4-T4
T4-O2
E1
E2
Chin
EMG_Tib
LegL
LegR
ECG
Flow_OR
Thorax
Abdome
SpO2
Pulse
Resp.Rate

ALLEGATO 7

The NREM sleep seems to play a major role in memory and cognition, regulating synaptic homeostasis (Tononi & Cirelli, 2014) and reshaping hippocampal-neocortical network necessary for long-term memory consolidation (Born & Wilhelm, 2012; Buzsáki, 2015; Diekelmann, 2014). The fact that NREM sleep may strongly activate IEDs, including those produced by the mesial temporal regions (Lambert et al., 2018), may have consequences on both synaptic plasticity and hippocampalneocortical dialogue. In childhood epilepsies characterized by a strong activation of IEDs during NREM sleep and cognitive alterations (LKS, continuous spike and waves during slow-wave sleep), an association between cognitive impairment and an altered overnight decrease of slow waves (a sign of altered slow wave homeostasis) has been reported, suggesting that IEDs may prevent the physiological process of synaptic downscaling. This seems to be supported by the improvement of cognitive functions in these patients after recovery of the homeostatic regulation of slow waves (Bölsterli et al., 2011, 2017). On the other hand, IEDs occurring during sleep have been suggested to disturb the coupling between hippocampal ripples, thalamic spindles and cortical slow waves, necessary for long-term memory consolidation (Buzsáki, 1989, 2015). Indeed, recent studies, conducted during the presurgical evaluation of drug-resistant patients with focal epilepsy, showed a link between NREM sleep-related hippocampal IEDs and the impairment of long-term memory consolidation (Lambert et al., 2020, 2021).

ALLEGATO 8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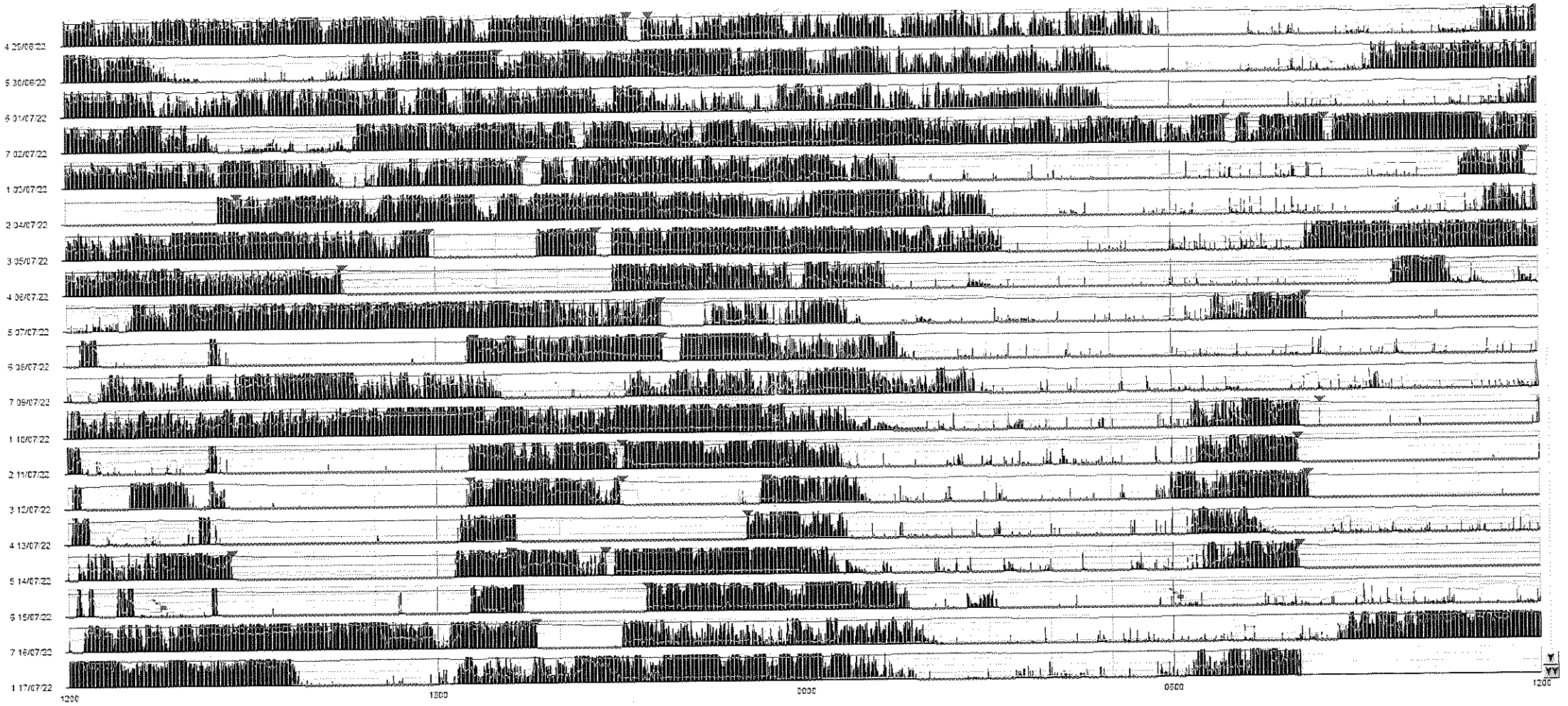
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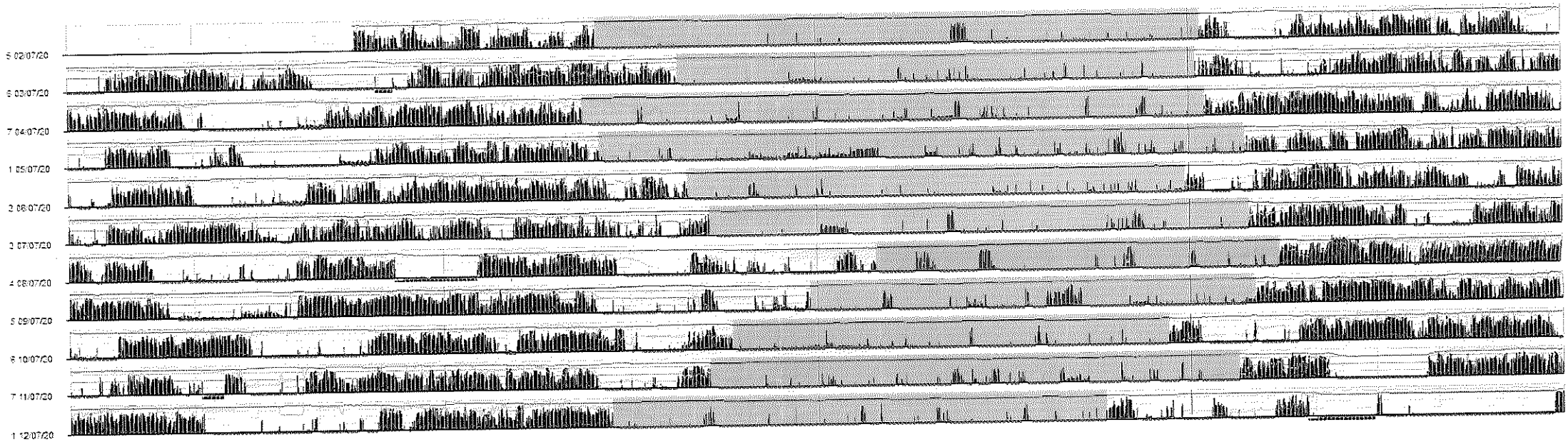
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ALLEGATO 9

1) **Circadian rhythms are part of the internal 24-hr daily cycle of nearly all biological functions.** Circadian patterns in seizure occurrence have been recognized for centuries. Advances in diagnostic technology including chronic intracranial EEG recordings have confirmed the clinical observation of different temporal patterns of epileptic activity and seizure occurrence over the 24-hr period (Baud et al., 2018; Ct, Tk, Ft, & Mj, 2015). The diurnal occurrence of seizures is influenced by several factors, including the type of epilepsy (generalized or focal) and the site of seizure onset (i.e. frontal, temporal, etc.; Khan et al., 2018; Spencer et al., 2016). Generalized seizures have a tendency to occur in the morning following sleep. In focal epilepsies, frontal lobe seizures occur predominantly during sleep, while temporal lobe seizures arise mostly in wakefulness (Hofstra et al., 2011). Of note, these studies do not allow evaluation of whether the observed preferred time of occurrence is modulated by behavioural states (wakefulness versus sleep or drowsiness), environmental conditions or independent effects of the endogenous circadian system. While demonstrating circadian patterns of seizures in humans can be challenging, strong evidence supporting a circadian modulation of seizures is derived from animal models, where rigorous study designs are feasible. In a rat model of limbic epilepsy, the presence of a distinct endogenous circadian distribution of seizures, irrespective of the sleep–wake status, has been shown, and the distribution of seizures relative to time of day resembled the one observed in human mesial temporal lobe epilepsy (Quigg, Straume, Menaker, & Bertram, 1998).