5th Paris Appeal Congress, 18th of May, 2015
Royal Academy of Medicine, Belgium

IDIOPATHIC ENVIRONMENTAL INTOLERANCE: WHAT ROLE FOR ELECTROMAGNETIC FIELDS AND CHEMICALS?

Electrohypersensitivity and multiple chemical sensitivity: two clinic-biological aspects of the same disorder?

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Inclusion criteria

1. Absence of known pathology.
2. Reproductibility of symptom occurrence under the influence of electromagnetic fields (EMFs) and/or multiple chemicals whatever their incriminated source.
3. Regression or disappearance of symptoms in the case of EMF and/or multiple chemical avoidance.

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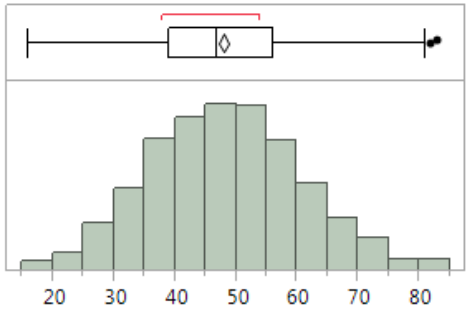
EHS and/or MCS-self reporting patients : A prospective clinical study

Total investigated	**1216**
Total presently analyzed:	**839**
Neither EHS nor MCS:	29
Not evaluable:	83
Evaluable:	**727**
EHS:	521
MCS:	52
EHS+MCS:	154

Sex ratio:

495F (68%)

232M (32%)



Age:

Mean: 47.9+/-12.4

Median: 47 [16-83]

Origin: Patients from France, Europe, Northern America, Other …

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Age and sex ratio in EHS and/or MCS self-reporting patients

	EHS	MCS	EHS/MCS
n	521	52	154
Mean age	48.2+/-12.9	48.5+/-10.3	46.7+/-11.2
Median age and [extremes]	48[16-83]	47 [31-70]	46 [22-76]
Sex ratio	344F/177M	34F/18M	117F/37M
percentage	66%	65%	76%

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Histamine in the peripheral blood of EHS and/or self-reporting MCS patients

	EHS	MCS	EHS/MCS
N	521	52	154
Histamine >10 nmol/l	182/491 (37%)	18/44 (36.7%)	59/142 (41.5 %)

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Histamine release by neuroinflammation-associated cells

In the healthy brain the “bulk” concentration of histamine is very low. Upon brain injury, degeneration or infection, the inflammatory response may trigger degranulation of **mast cells**, leading to a massive release of histamine in the blood and in the cerebrospinal fluid, leading to an increase of blood brain barrier (BBB) permeability through oxidative stress.

Rocha et al. Front Cell Neurosci. 2014 Apr 30;8:120.

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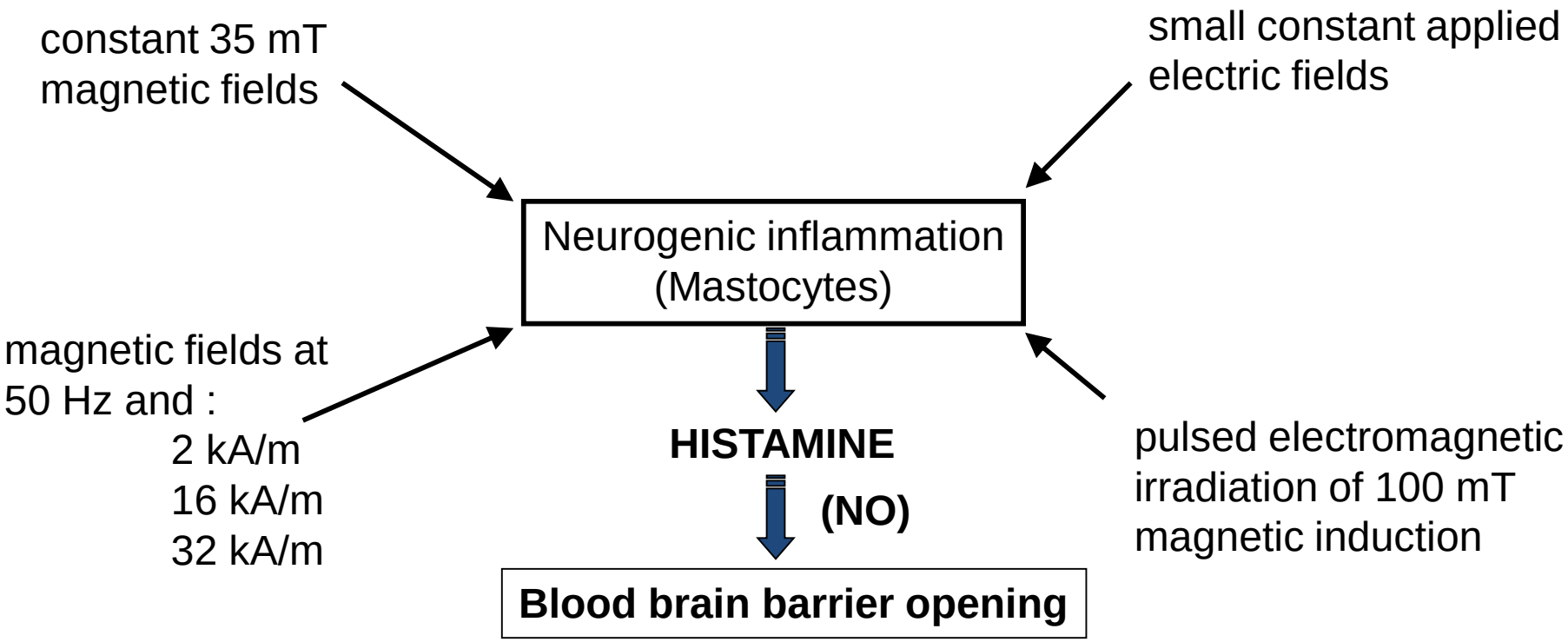
Stress-induced neurogenic inflammation

involving histamine synthesis by inflammatory cells and mast cell degranulation - Histamine release as a key mechanism in Electromagnetic field intolerance syndrome (EMFIS)



W.G. Mayhan. Role of nitric oxide in histamine-induced increases in permeability of the blood-brain barrier. Brain Research 1996, 743 , 70-76

S. Gangi and O. Johansson. A theorical model based upon mast cells and histamine to explain the recently proclaimed sensitivity to electric and/or magnetic fields in Humans. Medical Hypotheses. 2000, 54, 663-671.

Auto-antibodies in the peripheral blood of EHS and/or MCS self-reporting patients

	EHS	MCS	EHS/MCS
n	521	52	154
Anti-O-myelin	109/477 (28.8%)	8/47 (17%)	33/140 (23.4%)
Anti-Hsp 70 >5 ng/ml	91/486 (18.7%)	4/52 (7.7 %)	36/142 (7.6%)
Anti-Hsp 27 > 5ng/ml	123/476 (25.8 %)	6/52 (11.5 %)	42/132 (11.5 %)
Anti-O-myelin and/or anti-Hsp70 and/or anti-Hsp27	197/457 (43.1 %)	12/48 (25 %)	65/127 (52 %)

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How to interpret the increase in anti-O-myelin, anti-Hsp70 and anti-Hsp27 auto-antibodies?

Protein can be modified by oxidation. Extensive oxidative leads to denaturation and loss of biological activity, while initial step of oxidation may change their specificity du to the chemical alteration of the paratope*.

Our hypothesis is that **under the influence of electric and/or electromagnetic fields and/or chemicals, oxidation of cerebral proteins may progress to auto-immunoreactivity** leading to the occurence of auto-antibodies against O-myelin and the chaperone proteins Hsp70 and Hsp27, which therefore lose their cell defensive properties.

* Bozic B. Ann N Y Acad Sci. 2007 Aug;1109:158-66.

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S100B protein and nitrotyrosin in the peripheral blood of EHS and/or MCS self-reporting patients

	EHS	MCS	EHS/MCS
N	521	52	154
S100B >0.105 µg/L	73/495 (14.7%)	6/51 (19.7%)	28/142 (10.7%)
NTT* >0.9 µg/ml	77/259 (29.7%)	6/29 (26%)	22/76 (28.9%)
Increased S100B and/or NTT	133/250 (53.2%)	12/22 (54.5%)	46/73 (63%)
Increased histamine, S100B and/or NTT	220/327 (71.8%)	27/36 (75%)	91/125 (79.1%)

*Nitrotyrosin is a marker of peroxinitrite ($ONOO^-$) production :

$O2^{\bullet -} + NO^{\bullet} \longrightarrow ONOO^-$

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How to interpret S100B and NTT increase?

DATA	interpretation
S100B	BBB opening* (cerebral hypoperfusion)
NTT	BBB opening** (oxidative stress)

*Marchi N et al. Clin Chim Acta. 2004 Apr;342(1-2):1-12;
Koh SX and Lee JK. Sports Med. 2014 Mar;44(3):369-85.

**Tan KH et al. Neurochem Res. 2004 Mar;29(3):579-87.
Phares TW et al. J Immunol. 2007 Jun 1;178(11):7334-43;
Pacher P. Physiol Rev. 2007 Jan;87(1):315-424;
Yang S. J Mol Neurosci. 2013 Oct;51(2):352-63.

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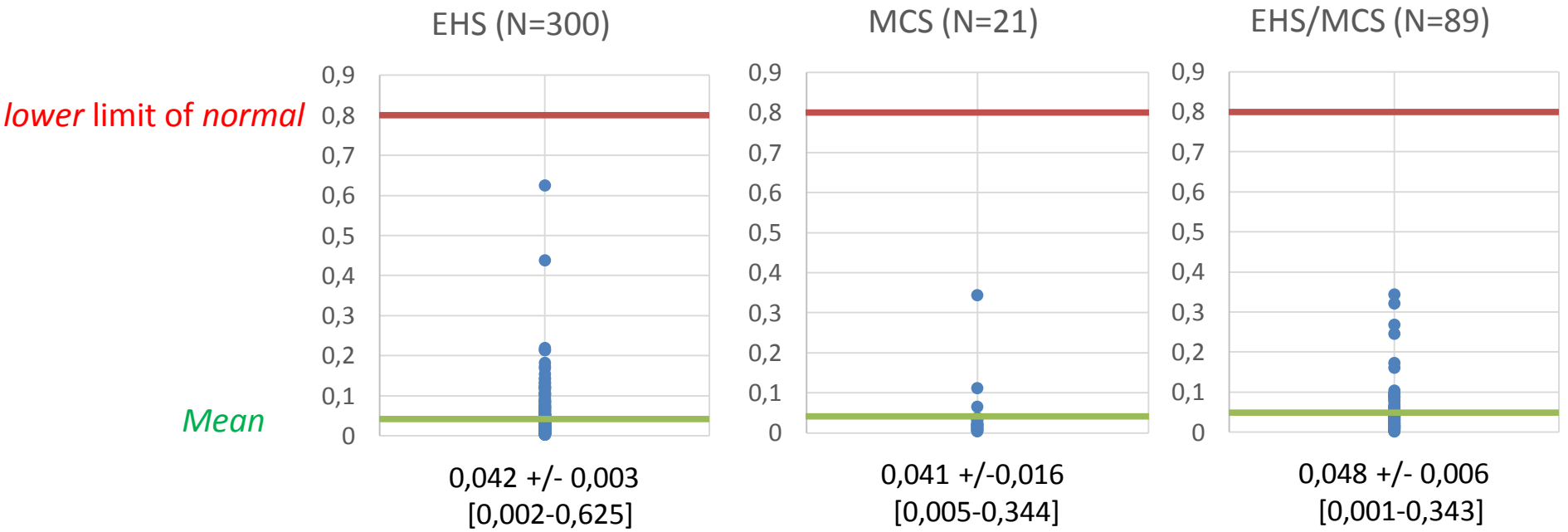
24H urine melatonin/creatinine ratio in EHS and/or MCS patients



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Cerebral hypoperfusion in EHS and/or MCS self-reporting patients

Cerebral hypoperfusion is not specific but is a quasi-constant fundamental abnormality similar to that found in Alzheimer's disease and other neurodegenerative disorders

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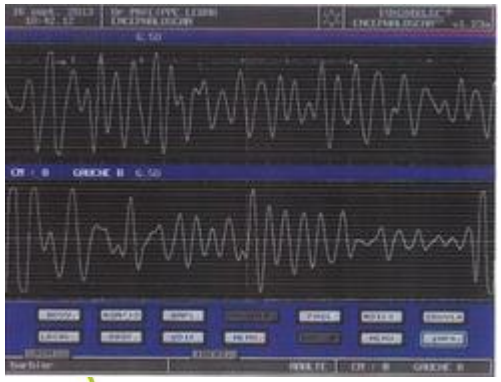
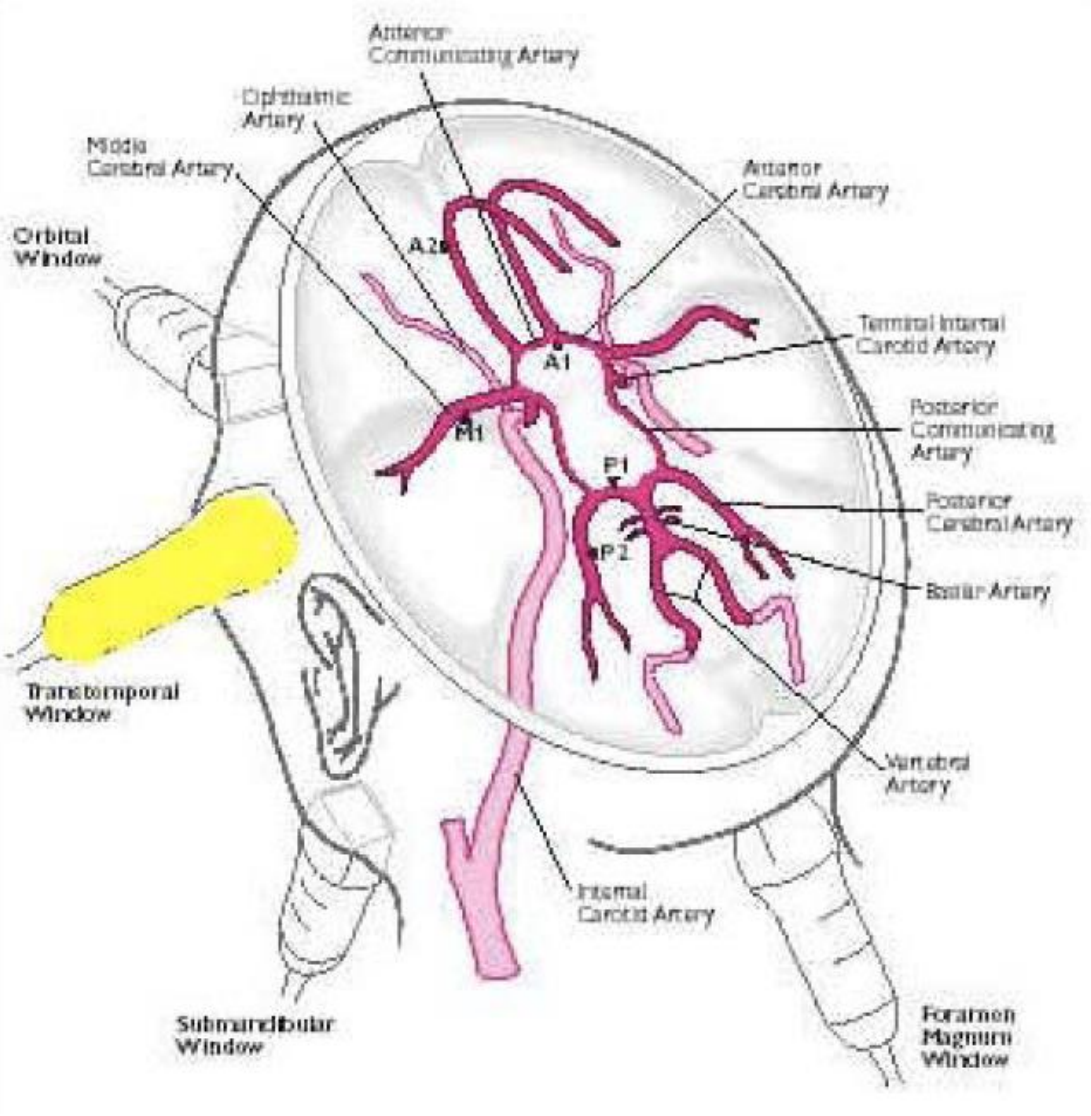
Echodoppler of the middle cerebral artery

Explore 60% of the hemisphere





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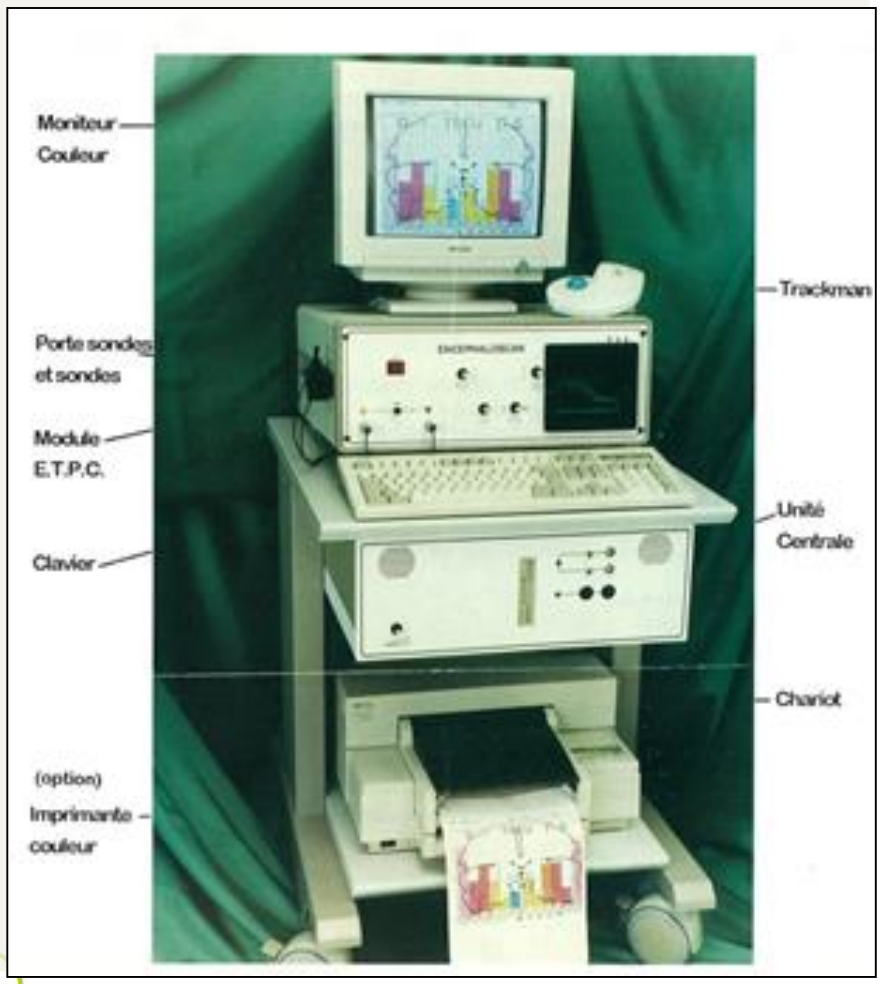
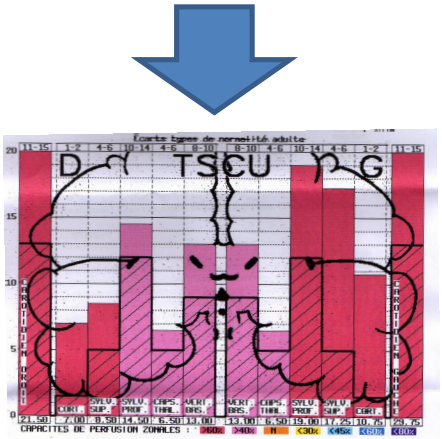
Centimetric ultrasound recording of cerebral pulsativity : Encephaloscan



- Combination of a computer with a cerebral tomosphygmograph
- Source emitting pulsed ultrasounds
- Explore the temporal lobes



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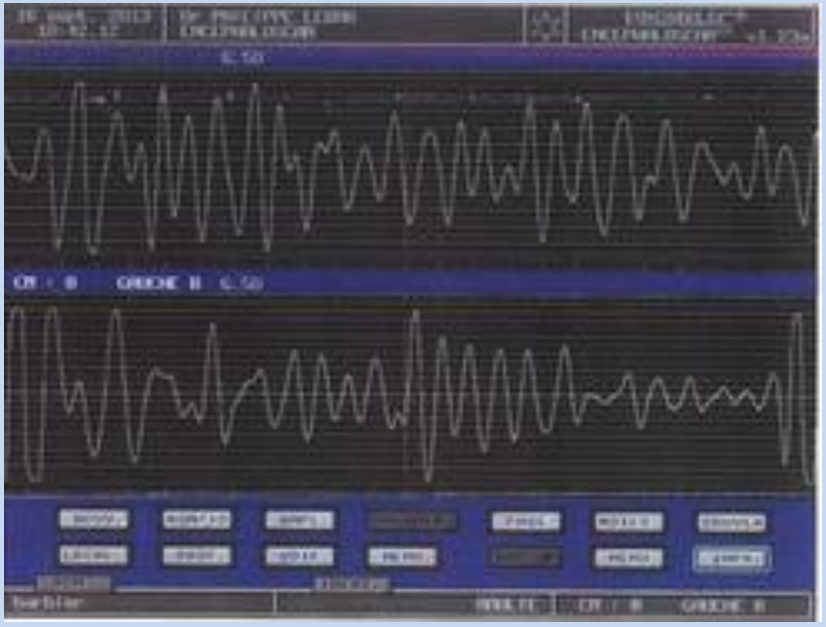

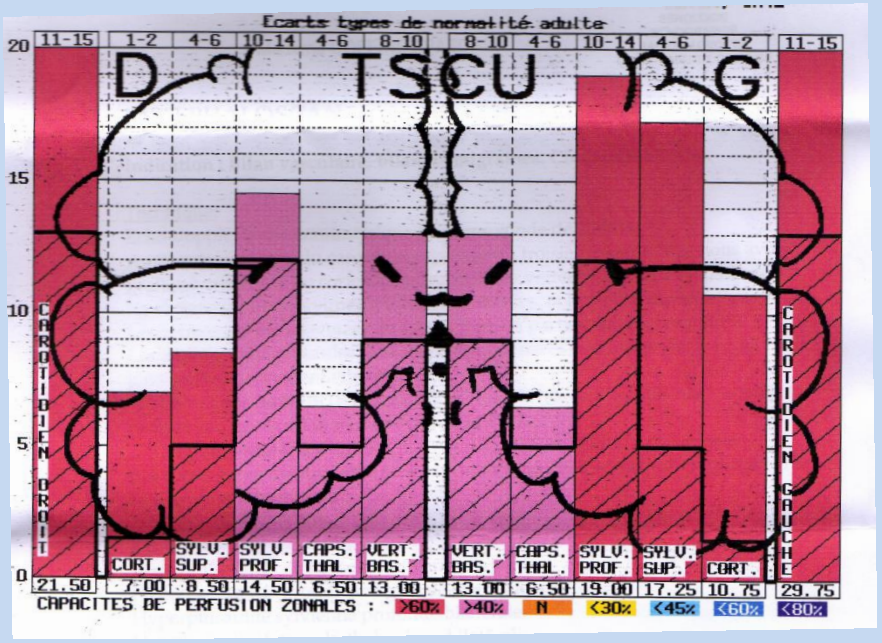
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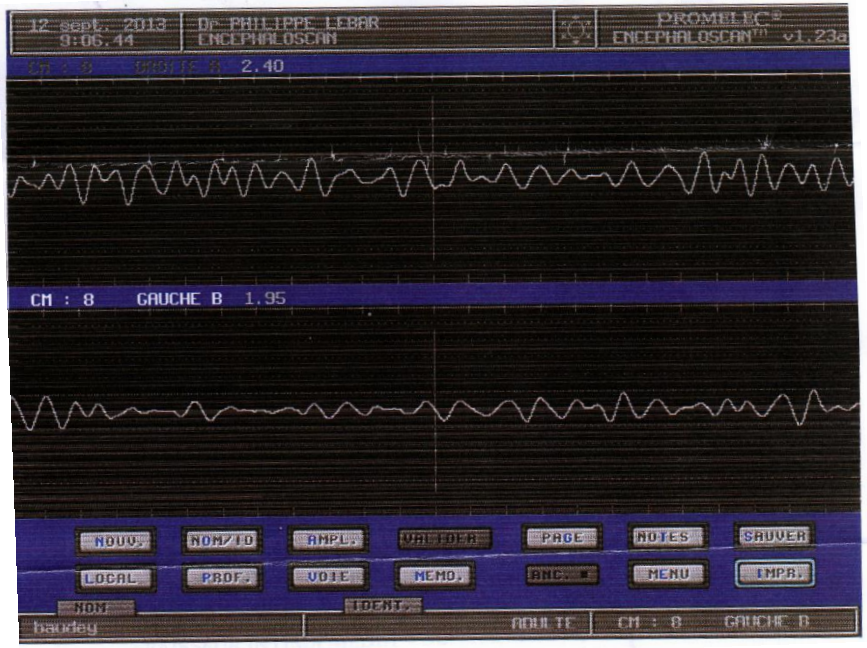
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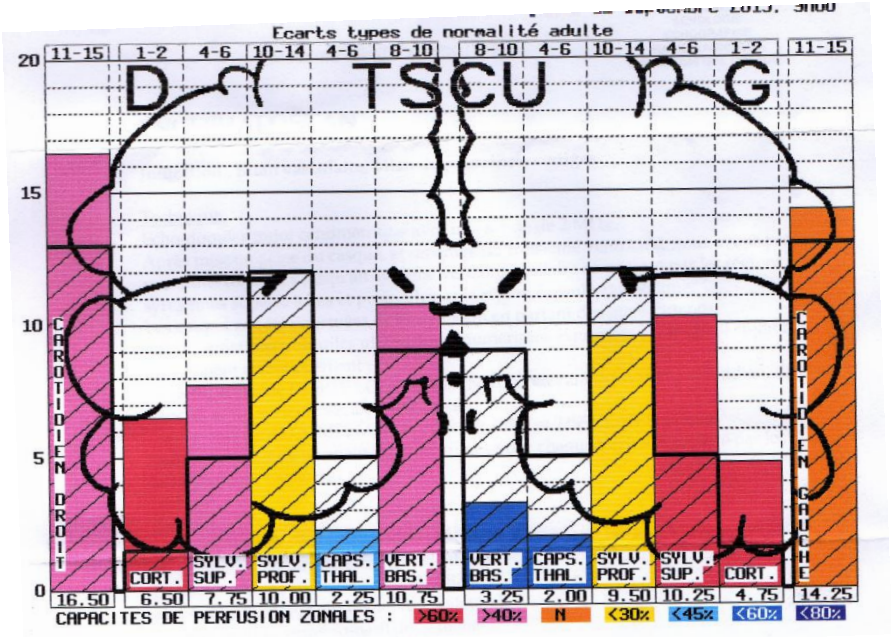
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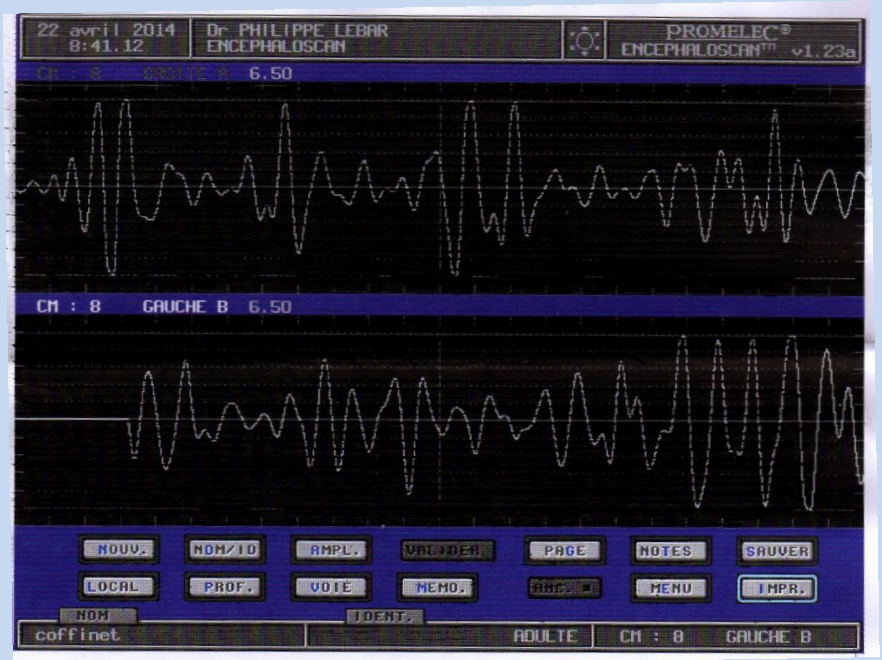
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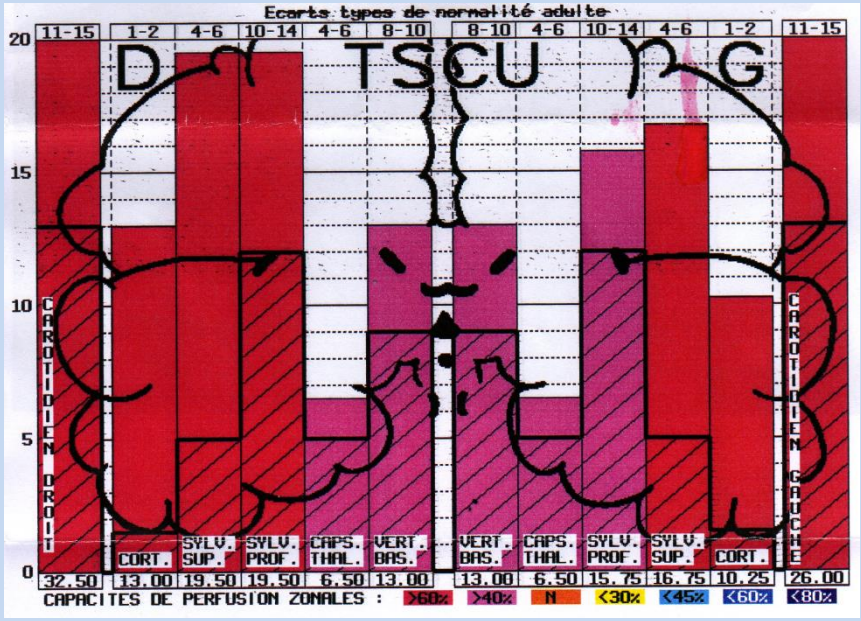
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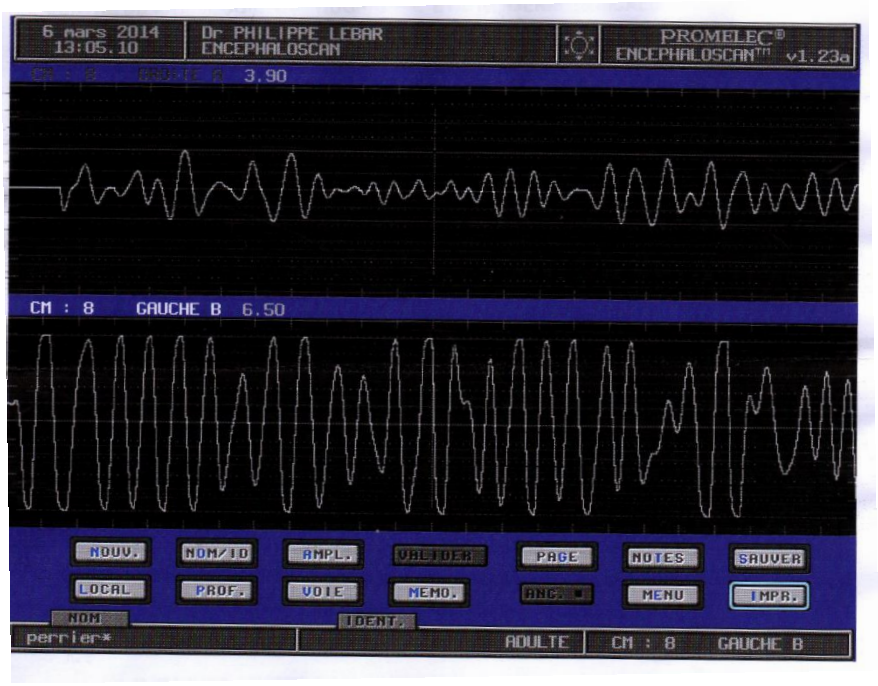
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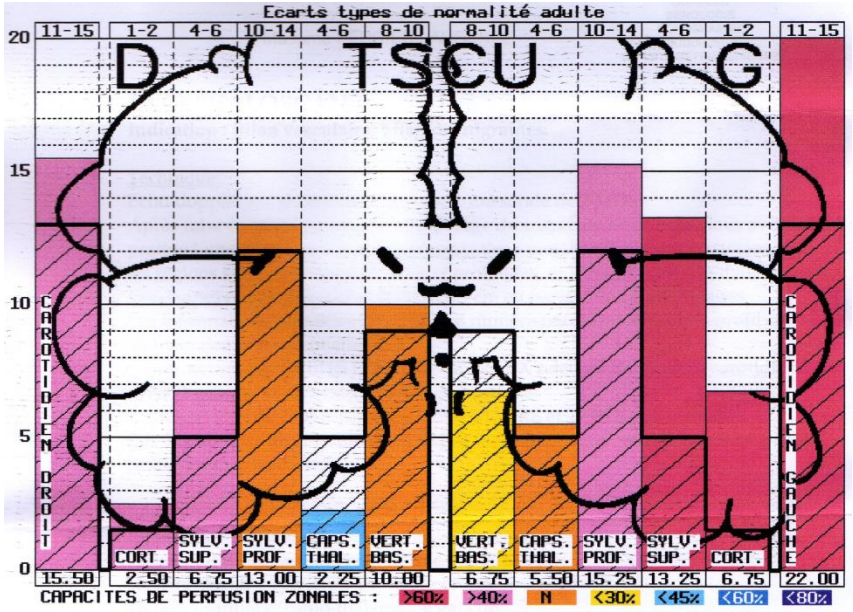
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Are EHS and MCS syndromes two aspects of a unique pathologic disorder involving the limbic system and/or the thalamus?

1. Similar symptomatic pictures
2. Similar biological abnormalities
3. Opening of the Blood Brain Barrier in both cases
4. Association in the same patient
5. Similar therapeutic results

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Scientific arguments strongly suggesting that self-reported EHS and/or MCS are causally-related to EMF and/or chemical exposure

1. No already recognized pathology
2. Appearance and disappearance of clinical symptoms as well as imaging and biological abnormalities depending on electromagnetic source exposure
3. Biological abnormalities detected in humans identical or similar to those observed in experimental animals submitted to EMFs
4. Association of MCS with EHS
5. Limited or no value of negative retrospective interview-based epidemiological studies because of a lack of science-based inclusion criteria and immediate and/or retrospective memory deficiencies

Thank for your attention !

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