

Integrated Quantum Therapy in an Epileptic Child after 13 Years of Inherited Real Risk of Epilepsy in Evolution: A Case Study

Marco Marchionni*, Simone Caramel†, Sergio Stagnaro‡

ABSTRACT

The comprehension of the pathogenesis of epilepsy finds a new impulse in studies on non-linear dynamics of EEG signals and in the growing genetic molecular evidence of the mitochondrial origin of this disease. These data are consistent with the information known by Quantum Biophysical Semeiotics that clinically investigates microcirculation both on a functional (i.e. by studying its non-linear dynamics), and on a structural viewpoint. Microcirculatory dysfunctions reflect those of a genetically altered mit-DNA and of a functional mitochondrial cytopathy known as Congenital Acidotic Enzyme-Metabolic Histangiopathy, originating from this mutation and which is particularly intense both in patients with epilepsy and in those with Inherited Real Risk of epilepsy. Preclinical diagnosis of Inherited Real Risk of epilepsy presents the opportunity to examine subjects with a predisposition to this disease since birth in order to perform an efficient pre-primary and primary prevention. In our case report, an integrated quantum therapy applied to a child with Inherited Real Risk of epilepsy in strong evolution provides encouraging satisfactory results.

Key Words: epilepsy diagnosis, mitochondrial dysfunction, primary prevention, quantum therapy, deterministic chaos

DOI Number: 10.14704/nq.2016.14.2.947

NeuroQuantology 2016; 2: 338-347

Introduction

The Pathogenesis of Epilepsy: An Overview

The complex pathogenesis of epilepsy has not yet been fully explained in a unique, consistent, and harmonic form according to shared scientific opinion (Yu and Pearl, 2013; Papetti *et al.*, 2013; Frye, 2015). During the past few decades,

scientists have tried to explain epilepsy as a primary metabolic disorder. According to the actual scientific knowledge, the latest ring of each metabolic or biochemical influence able to modify predisposition to epilepsy is the polarization level of the neuronal membrane, i.e. the difference in potential between the inside and the outside of a cell. Currently, this highlights the important role of the mitochondrion in the etiopathogenesis of epilepsy. In particular, a key role is played by the sodium-potassium pump, also called ATP-dependant Na⁺/K⁺ (Na⁺/K⁺ ATPase) pump: this is an enzyme located in cellular membranes which is strictly related to mitochondrial activity. For this reason, epilepsy is supposed to be a mitochondrial disease in some studies (Zsurka

Corresponding author: Caramel Simone
Address: via Doberdò, 3 – 31020 Fontane di Villorba TV, Italy.
Phone: + 39 338 8129030
Fax: +39 0422 1830918
e-mail ✉ info.sisbq@mail.com

Relevant conflicts of interest/financial disclosures: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Received: 17 February 2016; **Revised:** 26 March 2016;

Accepted: 05 April 2016

eISSN 1303-5150



and Kunz, 2010; Folbergrová and Kunz, 2012; Lee, 2012; Zsurka and Kunz, 2015).

Mitochondrial dysfunctions cause cellular dysfunction, also known as mitochondrial cytopathy. Mitochondrial cytopathies (Schmiedel *et al.*, 2003) are the basis of mitochondrial diseases, which are often caused by mutations, acquired or maternally inherited, in the mitochondrial DNA or nuclear genes that code for respiratory chain complexes in mitochondria. There is compelling evidence for the direct involvement of mitochondria in certain neurodegenerative disorders, such as Parkinson's disease (Banerjee *et al.*, 2009; Guerra de Souza, 2016). Friedreich's ataxia (FRDA) (Baron *et al.*, 2007), amyotrophic lateral sclerosis (ALS) (Baron *et al.*, 2007), Huntington's disease (Graeber and Muller, 1998), myoclonic epilepsy (Khurana *et al.*, 2013), and temporal lobe epilepsy (TLE) (Waldbaum and Patel, 2010). This suggests that the critical factor which determines the survival of neurons in neurodegenerative disorders is the degree of mit-DNA damage and the maintenance of an appropriate mit-DNA copy number (Graeber and Müller, 1998; Baron *et al.*, 2007). Mitochondrial diseases involve multiple organs and show heterogeneous and unpredictable progression. The most common clinical presentation of mitochondrial diseases is encephalomyopathy, and epileptic seizures can frequently occur as a presenting sign of mitochondrial encephalopathy. Whether mitochondrial dysfunction or epilepsy is the cause or consequence is still debatable. Epileptic phenotypes vary in different mitochondrial diseases (Kang *et al.*, 2013). Oxidative stress and mitochondrial dysfunction are emerging as key factors that not only result from epileptic seizures, but may also contribute to epileptogenesis as well (Waldbaum and Patel, 2010). The mitochondrion has an essential role in neuronal excitability and survival. It is a key cellular structure involved in a great number of metabolic functions such as ATP synthesis by oxidative phosphorylation, tricarboxylic acid cycle or fatty acid oxidation, Fe/S Cluster production, and so on. These pathways are fundamental for biological processes such as cell proliferation or death. Some mitochondrial dysfunctions of the central nervous system are responsible for several neurological diseases and age-related neurodegenerative disorders, including Alzheimer's and Parkinson's diseases (Gurses *et al.*, 2014).

The above-cited genetic and biochemical

data highlight different mechanisms by which mitochondrial bioenergetics is altered in the hereditary defects of complex I (Papa *et al.*, 2009). Mitochondrial diseases are frequently caused by a disruption of the respiratory chain. Nevertheless, other mitochondrial functions, including organelles dynamics or metabolite transport, could also be involved in such pathologies (Molinari, 2010). In addition to being responsible for energy production, mitochondria also play a crucial role in the maintenance of intracellular calcium homeostasis and generation of reactive oxygen species and mechanisms of cell death, but data about the role of mitochondria in epilepsy are relatively scarce. Mitochondrial genome analysis is rarely carried out in the investigation of various diseases, including epilepsy. In mesial temporal lobe epilepsy (MTLE) - hippocampal sclerosis (HS) - cases, mitochondrial genome analysis showed evidence of mitochondrial dysfunctions caused by important mutations associated with epilepsy (Zhao *et al.*, 2008; Zhang *et al.*, 2013). There are similar evidences in Early Infantile Epileptic Encephalopathy (Palmieri, 2008).

Another key factor seemingly not related to the pathogenesis of epilepsy originating from mitochondrial mutations is the predictability or non-predictability of epileptic seizures. Throughout the years, many non-linear analyses have been conducted on various epilepsy cases (Ferri *et al.*, 2001; Litt and Echauz, 2002). Children with electrical status epilepticus during slow-wave sleep (ESES), for example, show a profound alteration of their EEG dynamics with the occurrence, during sleep, of low-dimensional chaotic structure able to modify their brain functioning during sleep (Sabesan *et al.*, 2003; Chaovalitwongse *et al.*, 2005; Yambe *et al.*, 2005; Zhu *et al.*, 2014). Using different statistical measures, results agree and are consistent with one another: EEG non-linear signals are chaotic under normal physiological conditions, while signals tend to linear regularity in epilepsy cases (Wang *et al.*, 2007; Good *et al.*, 2010). Epileptic seizures can therefore be predicted through the chaotic analysis of EEG signals (Rosso, 2007; Raiesdana *et al.*, 2008; Gao *et al.*, 2011; Korsakova *et al.*, 2011).

The mitochondrial nature of epileptic diseases and the loss of non-linear deterministic chaos in epilepsy are two core aspects of the clinical investigation of Quantum Biophysical Semeiotics (QBS) and of its bedside diagnosis, which presents the opportunity to obtain



important data for a better comprehension of the pathogenesis of epilepsy, its genetic predisposition, and its Inherited Real Risk (Stagnaro, 2004a). This will support performing an original pre-primary and primary prevention.

Experimental Section: Method and Material

Inherited Real Risk of Epilepsy: Physiopathology

The background for all mitochondrial diseases studied by QBS is a particular cell suffering - or functional mitochondrial cytopathy (Stagnaro, 1985), known as Congenital Acidotic Enzyme-Metabolic Histangiopathy (CAEMH). If and where the CAEMH is most intensely present, this is a sign of a locally highly altered mit-DNA and, consequently, of tissue suffering and functional impairment, according to mitochondrial heteroplasmy, both endo- and inter-cellular. The result is the formation of one or more QBS constitutions which predispose the organism to some of the most serious degenerative diseases, such as type 2 diabetes, mellitus, cardiovascular diseases, any form of cancer, solid or liquid, and neurodegenerative diseases (Stagnaro and Neri, 2004).

The QBS constitution (Stagnaro, 2004a), whose presence can be diagnosed in every individual since birth by means of a stethoscope, is the initial preclinical stage of a disease which can evolve into an Inherited Real Risk (IRR) of a specific pathology (Stagnaro, 2009). It indicates that the degenerative preclinical stage (sometimes called for example: pre-diabetes or pre-cancer stage, etc.) is dangerously approaching the clinical stage, which is the appearance of the disease itself.

Constitutions do not necessarily evolve into disease, as not all individuals with the pathological constitution develop its corresponding disorder in their life. When it does happen, however, the first thing to be examined is the IRR: an indicator of a possible slow and gradual shift to a pathological condition, which is always worsened, but never caused, by its environmental risk factors. IRR diagnosis therefore has a crucial role in QBS, allowing to diagnose the presence of a Real Risk of epilepsy already since birth, and thus confirming the mitochondrial nature of this disease, both if hereditary or acquired, as already pointed out by several authors in the previous section. To understand the pathogenesis of epilepsy,

especially at its initial preclinical stage, according to QBS, it is necessary to analyze the data provided by clinical microangiography (Stagnaro, 2011), a QBS branch which studies microcirculation both on the functional and on the structural viewpoint.

Studying microcirculatory dynamics and structures and analyzing the blood flow in arterioles, venules, nutritional capillaries, as well as examining the Endo-arteriolar Blocking Devices (EBDs), which are blood regulatory devices, provides important qualitative and quantitative information for a fine and detailed diagnosis. EBDs (Stagnaro, 2011) are tiny contractible and physiologically flexible structures which alternately open and close to regulate blood flow in microcirculation, that is the local flow-motion.

What reveals structural mutation, or microcirculatory remodeling, which grows together with functional mutation, is the presence of pathological EBDs. These are anatomically and structurally pathological, stiffer, and more obstructing than the physiological ones. Therefore, the opening and closing system of these 'tiny valves' is not flexible, harmonic, and contractible as in the physiological one, but stiff and obstructing. This has repercussions on the flow-motion, as can be seen in non-linear dynamics of microcirculatory oscillations, which are physiologically of a deterministic chaotic type, but they lose complexity already in the stage preceding the disease, i.e., pre-clinical diagnosis of IRR of pathology, while gradually tending to linearity when the disease has set in (Stagnaro and Neri, 2004; Stagnaro 2011).

This is confirmed by the progressive reduction in functional and structural complexity which reflects functional and structural mutations in microcirculation. Structural complexity is geometrically represented by the non-linear dynamic system equilibria observed. This equilibrium physiologically consists of a chaotic attractor (phase space reconstructed equilibrium), which can deteriorate into more simple (low-complexity) orders, such as a periodic equilibrium - limited cycle attractor (pathological IRR) - or a fixed equilibrium (disease at a chronic stage) (Stagnaro and Neri, 2004).

In the case of an IRR of epilepsy, statistical invariants measuring microcirculatory non-linear dynamics, i.e. fractal Dimension (fD) evaluation (Stagnaro-Neri *et al.*, 1996; Stagnaro-Neri and Stagnaro 1994; 1997), reveal a loss in complexity



which shows biological evidence in microcirculatory activity already since birth. This loss is constant in time and it might slowly reduce in conjunction with a slow progressive degenerative preclinical process (IRR evolution into disease). After crossing a critical threshold, this process could degenerate in an epileptic seizure or an epileptic equivalent. The measure of deterministic chaos is therefore a useful diagnostic tool both in preclinical and clinical stages of epilepsy.

Microvessel oscillations are also crucial to investigate the physiopathology of epilepsy and reveal the key role of mit-DNA mutation and of CAEMH in classifying epilepsy as a mitochondrial disease. This is important to define a pre-primary and primary prevention strictly related to its pathogenesis in view of an improvement in mitochondrial respiration, tissue oxygenation and tissue protection as well as of a retroactive genetic intervention into the altered mitochondrial genome. In other words, microcirculation is where *all human tragedies begin and end*. The genetically altered mit-DNA causing the CAEMH, which in turn can generate one or more QBS constitutions able to evolve - although not necessarily - into pathological IRRs, has always equivalents in the behavior of biologic systems, and in microcirculation in particular. Besides genetic mutation, QBS diagnosis allows to identify specific corresponding local mutations in microcirculatory activity and in its structures (i.e., EBDs, anastomosis (AVA)).

Like all IRRs, the IRR of epilepsy too can undergo many stages with different preclinical variants: from initial stage Real Risk to Real Risk in strong evolution, which is the stage before the formation of the epileptic focus and the appearance of the disease. This progressive degeneration happens simultaneously with and reflects what happens in microcirculation, the number of pathological EBDs increases with time, vasomotility and vasomotion progressively dissociate from each other, although the organism continuously produces new compensatory processes for microcirculatory remodeling. So, while the situation is getting worse and worse in the micro-world, nothing yet reveals outside in the macro-world, for example in the blood flow along vessels and arteries. This is the main reason why only the QBS bedside diagnosis is able to objectively verify pre-metabolic syndromes, because it is able to quantify microcirculatory dynamics and structures and give them a

diagnostic meaning and scope. Pathological EBDs continue to increase in number, and vasomotility and vasomotion become more and more different, microcirculatory remodeling is always greater, more continuous and intense, until the organism finally exhausts its compensatory action. This is the point at which the epileptic focus appears and the micro-world mutations reveal into the macro-world as matter-energy-information supply (blood flow) to the corresponding parenchyma. At this point, the disease sets in and can be clinically diagnosed, even with conventional instrumental analyses.

Patients with epilepsy, even those who don't suffer from seizures, show a limited brain area (generally a single one), where the microcirculatory remodeling takes place, and which is characterized by clear microcirculatory and parenchymal mutations. Only in cases where flow-motions are highly reduced by microvascular structural and functional mutations, which are typical signs of a remodeling event, can we talk about an epileptic focus, worsened but never caused by known environmental risk factors. The main purpose of the well-structured and refined activity of the tissue microvascular unit supported by the normal viscosity of the local interstice is the physiological regulation of blood flow in vessels under 100 micron, whose role is essential in matter-energy-information supply to the corresponding parenchyma.

It is now clear that any mutation in the refined microcirculatory process has serious repercussions on tissue economy, whose pH reduces, while hydrogen ionic H⁺ concentration consequently increases, and this is a typical sign of a compromised mitochondrial respiration causing histangic acidosis. QBS diagnostic approach now allows physicians to identify Constitutions and IRRs since birth, that is to say decades before the possible appearance of the corresponding diseases, and this presents the opportunity to perform a rational and detailed pre-primary and primary prevention.

Inherited Real Risk of Epilepsy: QBS Diagnosis

Traditional physical semeiotics does not provide diagnosis of epilepsy in either patients who do not suffer from seizures nor in seizure-affected individuals if the evaluation is made at a distance from seizures. On the contrary, the Reflex-Diagnostic-Auscultatory Percussion, which is the



basis of QBS, allows physicians to diagnose epilepsy and/or the epileptic focus, both in individuals who just seem to be healthy because they have no symptoms and in those who suffer from epilepsy. In other words, the QBS approach provides the means to easily recognize an epileptic focus long before its clinical appearance. It follows that QBS has a crucial role in the prevention of epileptic seizures. It should be noted, however, that epileptic symptoms and seizures will appear only if the corresponding IRR is particularly serious, i.e. it evolves into an epileptic focus.

To diagnose an IRR of epilepsy, physicians should check in sequence the oculo-gastric aspecific reflex, which is asymmetrical, and the cerebro-gastric aspecific reflex in the cerebral hemisphere previously identified, as well as verify whether a microcirculatory activation type I, associated, is present near the epileptic focus. We should also check QBS preconditioning to verify whether the epileptic disease has already set in (Stagnaro and Neri-Stagnaro, 2004).

Patients with epilepsy have the highest Congenital Acidotic Enzyme-Metabolic Histangiopathy (CAEMH) value. Besides CAEMH factor, the following reflexes have a crucial role for the diagnosis of epilepsy: the oculo-gastric aspecific reflex, the microcirculatory activation type I, associated, near the epileptic focus, and the cerebro-gastric aspecific reflex. We explore in details the last one.

Keeping fingers tight together and slightly flexed, physicians should apply nails on frontal region skin, first on the right and then on the left, then crawl up until the corresponding midpoint of the scalp, proceeding from the frontal to the occipital region with very slow movements. When a stimulus (i.e., a mean digital pressure), is exerted directly on skin projection of epileptic focus, a cerebro-gastric aspecific reflex is obtained, which can be further increased with light stimulation, apnoea test, and forced breathing.

We can consider at least 3 significant parameters of this reflex assessment: the latency time (the seconds lasting from the moment of the stimulus to the moment in which the stomach dilates), the intensity (that represents the maximal dilation of the stomach, calculated in centimeters), and the duration. The duration (D) of the cerebro-gastric reflex represents the time in seconds elapsing from the moment of the dilation of the stomach to the moment in which the stomach comes back to the basal position at rest. Compared to "healthy" parameters (the duration of the reflex varies between 3 and 4 seconds in healthy subjects), the duration (D) of the reflex is increased in case of Inherited Real Risk of epilepsy (4 sec. $\leq D < 6$ sec.), and this increase is even higher in case of overt pathology (Table 1) with different growing values depending on its severity and clinical evolution (pathology in evolution: 6 $< D \leq 7$); (advanced stages of pathology: D > 7).

Table 1. Duration of Cerebro-Gastric Aspecific Reflex for the diagnosis of epilepsy

Diagnosis	Duration (in seconds)
Healthy subject	3 < D < 4
Inherited Real Risk of cerebral pathology (pre-clinical stages)	4 $\leq D < 6$
Pathology (early clinical stages)	D ≥ 6
Pathology (in evolution)	6 < D ≤ 7
Overt pathology (advanced stages)	D > 7

Legend: D = duration of the reflex in seconds.

Reflex-Diagnostic-Auscultatory Percussion for cerebral diseases, based on the ancient Auscultatory Percussion technique (Caramel, 2014), is not only useful for epilepsy, but also for the most frequent and serious pathological processes originating from the central nervous system: cerebral cysts, cancer, acute and chronic ischemia, senile cerebral involution, headache, pituitary gland disease, Alzheimer's disease, Parkinson's disease, ALS and even rare diseases

such as Friedreich's ataxia.

Inherited Real Risk of Epilepsy and Primary Prevention

QBS approach is not only diagnostically relevant, but also useful for an adequate therapeutic monitoring, so as to verify effectiveness of treatments that can be suggested for an appropriate primary and pre-primary (Caramel, 2011) therapeutic prevention. There is a class of



treatments (i.e., green therapy) that, if adequately combined and integrated, should allow to make a pathological IRR, like the IRR of epilepsy, a residual factor (Stagnaro, 2004b; Marchionni *et al.*, 2013a, 2013b, 2014; Stagnaro and Caramel, 2011b, 2012a, 2012b, 2013a, 2013b, 2013c, 2013d, 2013e; Caramel *et al.*, 2014). To make a risk residual does not mean to remove it completely, but to reduce it to a minimum, i.e. to prevent it from growing and evolving into a disease. This category includes: modified Mediterranean diet combined with daily healthy physical activity, histaminic protectors (i.e., conjugated melatonin with the recipe of Di Bella – Ferrari, substances to improve tissue and mitochondrial oxygenation, coenzyme Q10, vitamins (i.e., vitamin C, orange squeezed juices with the addition of 7 lemon drops), and ascorbates (i.e., potassium ascorbate, sodium ascorbate).

The latest frontier discovered by QBS for pre-primary and primary prevention is a class of treatments known as 'blue therapy' (Stagnaro, 2013) which can heal CAEMH, constitutions, and IRRs completely, if the corresponding diseases have not set in yet. This strengthens and confirms the existing theories on wave genomics, according to Peter Gariaev (1994), and on genetic/genomic reversibility of nuclear and mitochondrial DNA, and especially the principle of Recursive Genome Function introduced by the American cross-disciplinary scientist and technologist Andras Pellionisz (Stagnaro and Caramel, 2011a). Clinical and experimental evidence of success in healing many pathological IRRs, among which the IRR of epilepsy, comes from the application of millimeter waves in 'Body Resonance Recording' (BRR) mode (Cem-Tech, now Ak-Tom) (Caramel *et al.*, 2014), from quantum therapy with sodium bicarbonate (Stagnaro, 2015a) (taking advantage of the extraordinary possibilities provided by quantum medicine combined with frequential information acquired by ad hoc substances, where water acts in harmony with the principle of water information memory), from noni juice (Caramel *et al.*, 2015) and from sulfurous water (Caramel, 2012).

Finally, the QBS experimental evidence highlight the importance of prayers for the health of patients: this practice plays a key role in the following case study reported below (Caramel and Stagnaro, 2011a).

Results and Discussion

Inherited Real Risk of Epilepsy in Strong Evolution: A Case Study

We report the case of an 11-year-old child with the following pathophysiological basis on May 20th 2013. The patient came to the hospital for tests after episodes characterized by unresponsiveness, with fixed gaze, and slowly falls to the ground, without reporting any serious trauma or head injury, lasting a few seconds, sometimes preceded by complex hallucinatory visions and followed by headache, dizziness, and sometimes vomiting, and slurred speech. The EEG Clinical Report provides the following output: background rhythm characterized by alpha at about 9 cycles per second, irregular, unstable, bilateral, symmetrical and reagent; interspersed on the temporal-parietal regions of the right by activity type PO of medium size with a tendency to spread to the contralateral homologous regions; IPN registers 2 PO bouffées of about 3 cycles per second for a period of 2-3 seconds. The clinical diagnosis is: paroxysmal abnormalities on the right parietal temporal regions with a tendency to spread; bouffées IPN. The suggested therapy is: Levetiracetam 500 in increasing dosage every week.

The child's parents decide to start the prescribed antiepileptic therapy on February 1st 2014, only after an additional episode of seizure. The initial therapy is with Levetiracetam, 125 mg, twice per day.

On February 17th 2014, the senior author (Camponeschi *et al.*, 2013), founder of Quantum Biophysical Semeiotics, visits the child. The QBS diagnosis is: Intense CAEMH in brain trigger-points; Inherited Real Risk of Brain Disorder; Inherited Real Risk of Epilepsy in evolution. After this QBS diagnosis the following therapy is applied: Cem-Tech (now Ak-Tom) treatment, i.e. millimeter waves (Caramel *et al.*, 2014) or Extremely High Frequencies (EHF) in the mode of Background Resonance Recording (BRR) on the following trigger-points (Figure 1):

- 1st yellow radiator: CAEMH trigger point (2 cm above the right external ear orifice, that is the skin projection area of CAEMH);
- 2nd yellow radiator: on the skin projection of the epileptic focus (in this specific case, upon the superior parietal region; 1 cm above the CAEMH trigger-point).

This is just one application in BRR mode: 60 seconds for capturing body radiations plus 10 minutes of re-radiation.

The patient continues a preventive therapy (resumed in Table 2) at home. On May 9th 2014 the child is monitored in the hospital for a 3 days check-up, after 2.5 months from first EHF-BRR treatment, with the following results:

- routine blood tests: within normal range;
- 3T MRI Brain Skull: no specific diagnostic abnormalities;
- cardio-tests ECG: no specific diagnostic abnormalities.
- extended video EEG monitoring: vigil and sleep; track with no specific diagnostic abnormalities.

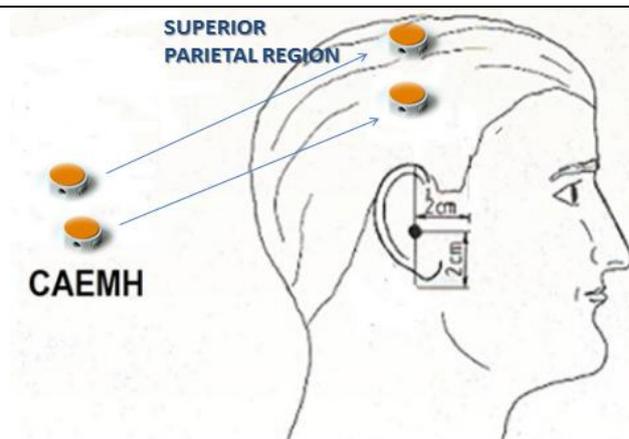


Figure 1. Trigger-point for the application of EHF-BRR treatment through yellow radiators: one on the skin projection area of CAEMH and one on the skin projection area of the epileptic focus (in this case, the superior parietal region)

Table 2. Therapeutic Monitoring of the Child from May 2013 to February 2016. Therapeutical monitoring from May 2013 to February 2016. Legend: BRR – EHF = Background Resonance Recording – Extremely High Frequency * The customized blue therapy includes: radiations from water energized by sodium bicarbonate and lemon drops, radiations from sulphurous water; radiations from noni juice, radiations captured from the epileptic focus area and CAEMH of the child stored in a BRR - Ak-Tom card.

Date of visit	QBS duration of cerebral-gastric reflex (see Table 1)	QBS diagnosis & therapeutical monitoring	Conventional Therapy	QBS therapy
May 20 th 2013			none	
February 1st 2014			Levetiracetam 125: 2 per day	
February 17 th 2014	D = 6 s	Inherited Real Risk of epilepsy in evolution	Levetiracetam 125: 2 per day	one BRR-EHF application
May 9 th 2014	D = 4,5 s	Inherited Real Risk of epilepsy (reduced)	none	Customized blue therapy *
November 2014	D= 4,5 s	Inherited Real Risk of epilepsy (reduced)	none	Customized blue therapy *
February 2015	D= 5,5 s	Inherited Real Risk of epilepsy (increasing)	none	Customized blue therapy*
May 2015	D= 6 s	Inherited Real Risk of epilepsy in evolution	none	Customized blue therapy*
September 2015	D= 6 s	Inherited Real Risk of epilepsy in evolution	none	one BRR – EHF application; “Integrated Quantum Therapy”: Customized blue therapy* integrated by ‘Iron Complex’ & ‘Cell Integrity Brain’
October 2015	D= 4 s	Inherited Real Risk of epilepsy (strongly reduced)	none	Holy Rosary ; “Integrated Quantum Therapy”: Customized blue therapy* integrated by ‘Iron Complex’ & ‘Cell Integrity Brain’
February 2016	D ≤ 4 s	Inherited Real Risk of epilepsy at minimal level (residual)	none	Holy Rosary ; “Integrated Quantum Therapy”: Customized blue therapy* integrated by ‘Iron Complex’ & ‘Cell Integrity Brain’

Indeed, the prolonged 24 hours video monitoring EEG provides the following results: absence of anomalies; no epileptic forms and asymmetries. The maneuver performed in the induction of symptoms to verify a diagnosis of a functional nature gives no effect. The hospital gives the indication to discontinue the epileptic therapy currently underway at low dosage, so the therapy discharge is to suspend the current anti-epileptic therapy with low dose: no therapy at discharge.

The patient spends one more year without any other clinical symptoms, but has relapse episodes in May, 2015.

The senior author visits the child again in September 2015. The QBS tests reveal an impairment of Fe-S clusters (Stagnaro, 2015b, 2015c), therefore the customized therapy resumed in Table 1 is integrated with 'Iron Complex' (from September 26th 2015 to October 18th 2015, 1 tablet per day; from November 19th 2015, 1 tablet per week) and 'Cell Integrity Brain' (from September 26th to January 19th 2016, 2 tablets per day; from January 20th 2016, 1 tablet per day), a food supplement of vitamins (B1, B6, C, E), extract of turmeric and bacopa, L-gluthatione, Myo inositol and Cawiar powder. Moreover, as of September 2015, parents and child start praying the Holy Rosary every evening. After this integration, which is useful for joining some neuronal pathways signals not yet reached by the previous treatments, the health condition of the child improves and the episodes of relapses disappear from October 2015 to February 2016 (Table 2). The QBS diagnosis at the beginning of February 2016 is as follows:

- Oculo-gastric aspecific reflex: physiological reflex values;
- Cerebro-gastric aspecific reflex: physiological reflex values;

- Microcirculatory activation near the epileptic focus: high physiological activation.

From QBS viewpoint and in accordance with the above mentioned collected parameters, the inherited risk of epilepsy has reduced and become residual and it will be fully healed as well as the genetic restructuring process will be completed. In fact, the mitochondrial genetic restructuring (Caramel and Stagnaro, 2011b, 2011c) is still continuing because there is a high physiological microcirculatory activation near the epileptic focus. The current therapeutic monitoring reveals that during the treatment some, but not all, flu viruses, can cause intense tissue acidosis. Although viruses can bring about epileptic seizures, the long term benefits of the ongoing therapy have not reduced.

Conclusion and Directions for Future Research

The most recent studies on the pathogenesis of epilepsy related to maternally inherited mitochondrial DNA genetic mutations confirm the previous clinical and experimental evidence of Quantum Biophysical Semeiotics, which is not only able to diagnose epilepsy at the bedside when the disease has set in, but also to evaluate its Inherited Real Risk already since birth and before its clinical manifestation, thus allowing to perform an efficient customized pre-primary and primary prevention, as evidenced by the study case here reported. Diagnosis and therapeutic monitoring of other similar cases in the near future will allow more accurate and in-depth assessments that integrate the current state of the art with the original preventive diagnostic here introduced.

Acknowledgments

We would like to express our gratitude to Ms Mara Germano, whose English language expertise supported this work considerably.

References

- Banerjee R et al. Mitochondrial dysfunction in the limelight of Parkinson's disease pathogenesis. *Biochim Biophys Acta*. 2009 Jul; 1792(7): 651-63.
- Baron M et al. Mitochondrial dysfunction in neurodegenerative disorders. *Biochem Soc Trans* 2007; 35(5): 1228-31.
- Camponeschi et al. A tribute to Sergio Stagnaro. *A Scientific Odyssey*. *Neuroquantology* 2013; 11(2): 289-304. DOI: [10.14704/nq.2013.11.2.676](https://doi.org/10.14704/nq.2013.11.2.676)
- Caramel S. Manuel's story. *QBS Magazine*, 2011. Available at: <http://www.sisbq.org/qbs-magazine.html>
- Caramel S. Meccanismi d'Azione Sconosciuti dell'Acqua Termale Sulfurea. *JOQBS*, 2012. Available at: <http://www.sisbq.org/uploads/5/6/8/7/5687930/h2s.pdf>
- Caramel S. Auscultatory Percussion of the Stomach, according to Sergio Stagnaro. *Handbook*, 2014. Available at: http://www.sisbq.org/uploads/5/6/8/7/5687930/card_iogarap_handbook.pdf
- Caramel S and Stagnaro S. Two Prayers Experiment: the effectiveness of different kinds of prayer through QBS assessments. *JOQBS*, 2011. Available at: http://www.sisbq.org/uploads/5/6/8/7/5687930/two_prayers_2011.pdf
- Caramel S and Stagnaro S. The Genetic Reversibility in Oncology. *JOQBS*, 2011. Available at: http://www.sisbq.org/uploads/5/6/8/7/5687930/reverse_oncology.pdf
- Caramel S and Stagnaro S. QBS and mit-Genome's fractal dimension. *JOQBS* 1-27, 2011. Available at: http://www.sisbq.org/uploads/5/6/8/7/5687930/joqbs_mitgenome.pdf
- Caramel S et al. Background Millimeter Radiation Influence in Cardiology on patients with metabolic and pre-metabolic syndrome. *Journal of Infrared and Millimeter Waves*, Shanghai, China, Vol.1, February 2014. Available at: http://journal.sitp.ac.cn/hwyhmb/hwyhmben/ch/readerr/view_abstract.aspx?flag=1&file_no=120750&journal_id=hwyhmben
- Caramel S et al. *Morinda citrifolia* Plays a Central Role in the Primary Prevention of Mitochondrial-dependent Degenerative Disorders. *Asian Pac J Cancer Prev* 2015; 16(4): 1675.
- Chaovalitwongse W et al. Performance of a seizure warning algorithm based on the dynamics of intracranial EEG. *Epilepsy Res* 2005; 64(3): 93-113.
- Ferri R et al. Non-linear EEG analysis in children with epilepsy and electrical status epilepticus during slow-wave sleep (ESES). *Clin Neurophysiol*. 2001; 112(12): 2274-80.
- Folbergrová J and Kunz WS. Mitochondrial dysfunction in epilepsy. *Mitochondrion*. 2012; 12(1): 35-40.
- Frye RE. Metabolic and mitochondrial disorders associated with epilepsy in children with autism spectrum disorder. *Epilepsy Behav* 2015; 47: 147-57.
- Gao J et al. Facilitating joint chaos and fractal analysis of biosignals through nonlinear adaptive filtering. *PLoS One* 2011; 6(9): e24331.
- Garaiev P et al. The DNA wave biocomputer, 1994. Available at: <http://www.invertone.com/WEBDOC/DNA-Garjajev-Poponin.pdf>
- Good LB et al. Nonlinear dynamics of seizure prediction in a rodent model of epilepsy. *Nonlinear Dynamics Psychol Life Sci* 2010; 14(4): 411-34.
- Graeber MB and Müller U. Recent developments in the molecular genetics of mitochondrial disorders. *J Neurol Sci* 1998; 153(2): 251-63.
- Guerra de Souza AC et al. Mitochondrial dysfunction and SUMOylation targets in Parkinson's disease. *J Neurochem*. 2016 Mar 2.
- Gurses C et al. Mitochondrial DNA profiling via genomic analysis in mesial temporal lobe epilepsy patients with hippocampal sclerosis. *Gene* 2014; 538(2): 323-7.
- Kang HC et al. Mitochondrial disease and epilepsy. *Brain Dev* 2013; 35(8): 757-61.
- Korsakova et al. Diagnosis of different stages of epileptogenesis by fractal EEG analysis. *Zh Nevrol Psikhiatr Im S S Korsakova* 2011; 111(5): 37-41.
- Khurana DS et al. Mitochondrial dysfunction in epilepsy. *Semin Pediatr Neurol*. 2013 Sep; 20(3): 176-87.
- Lee YM. Mitochondrial diseases. *J Epilepsy Res*. 2012 Mar 30; 2(1): 1-4.
- Litt B and Echaz J. Prediction of epileptic seizures. *Lancet Neurol* 2002; 1(1): 22-30.
- Marchionni M et al. Inherited real risk of Alzheimer's disease: bedside diagnosis and primary prevention. *Front Aging Neuroscience*, 2013. [Pub-Med indexed for Medline] Available at: http://www.frontiersin.org/Aging_Neuroscience/10.3389/fnagi.2013.00013/full
- Marchionni M et al. Quantum Biophysical Semeiotics Bedside Diagnosing Inherited Real Risk of Brain Disorders: Boezio's Sign. *Neurodegenerative Disease Primary Prevention*. *JOQBS*, 2013. Available at: http://www.sisbq.org/uploads/5/6/8/7/5687930/boeziosign_2013.pdf
- Marchionni M et al. The role of 'Modified Mediterranean Diet' and quantum therapy in Alzheimer's disease primary prevention. *JNHA*, Springer, Vol. 18, 1, 2014. [Pub-Med indexed for Medline]
- Molinari F. Mitochondria and neonatal epileptic encephalopathies with suppression burst. *J Bioenerg Biomembr* 2010; 42(6): 467-71.
- Palmieri F. Diseases caused by defects of mitochondrial carriers: a review. *Biochim Biophys Acta* 2008; 1777(7-8): 564-78.
- Papa S et al. Pathogenetic mechanisms in hereditary dysfunctions of complex I of the respiratory chain in neurological diseases. *Biochim Biophys Acta* 2009; 1787(5): 502-17.
- Papetti L et al. Metabolic epilepsy: an update. *Brain Dev* 2013; 35(9): 827-41.
- Raiesdana et al. Complexity evolution in epileptic seizure. *Conf Proc IEEE Eng Med Biol Soc* 2008; 4110-3.
- Rosso OA. Entropy changes in brain function. *Int J Psychophysiol* 2007; 64(1): 75-80.
- Sabesan S et al. Predictability of epileptic seizures: a comparative study using Lyapunov exponent and entropy based measures. *Biomed Sci Instrum* 2003; 39: 129-35.
- Schmiedel J et al. Mitochondrial cytopathies. *J Neurol* 2003; 250(3): 267-77.
- Stagnaro S. *Istangiopatia Congenita Acidotica Enzimometabolica. Una Patologia Mitocondriale Ignorata*. *Gazz Med It Arch Sci Med* 1985; 144: 423.
- Stagnaro S. *Le Costituzioni Semeiotico Biofisiche*. Ed. Travel Factory, Roma, 2004a.
- Stagnaro S. *La Melatonina nella prevenzione primaria dei tumori*. Ed. Travel Factory, Roma, 2004b.



- Stagnaro S. Reale Rischio Semeiotico Biofisico. Ed. Travel Factory, Roma, 2009.
- Stagnaro S and Neri Stagnaro M. Introduzione alla Semeiotica Biofisica. Il Terreno Oncologico. Ed. Travel Factory, Roma, 2004; pg. 194-195.
- Stagnaro S. Introduzione alla Microangiologia Clinica, JOQBS, 2011. Available at:
http://www.sisbq.org/uploads/5/6/8/7/5687930/mc_intro.pdf
- Stagnaro S. Il Manifesto della Medicina Semeiotico Biofisico Quantistica Ristrutturante (MSBQR), JOQBS, 2013. Available at:
<http://www.sisbq.org/uploads/5/6/8/7/5687930/manifestomedicinasbqristrutturante2013.pdf>
- Stagnaro S. Il Reale Rischio Congenito epilettico. JOQBS, 2015. Available at:
http://www.sisbq.org/uploads/5/6/8/7/5687930/rrc_epilessia.pdf
- Stagnaro S. Diagnosi clinica Semeiotico-Biofisico-Quantistica della carenza di ferro mediante valutazione dell'attività midollare dell'eritropoietina endogena: il segno di Baserga. JOQBS, 2015. Available at:
<http://www.sisbq.org/uploads/5/6/8/7/5687930/segnodibaserga.pdf>
- Stagnaro S. E' possibile valutare con un fonendoscopio l'attività del cluster ferro - zolfo nei sistemi biologici? JOQBS, 2015. Available at:
<http://www.sisbq.org/uploads/5/6/8/7/5687930/clusterfes2015.pdf>
- Stagnaro-Neri M and Stagnaro S. Deterministic chaotic biological system: the microcirculatory bed. Theoretical and practical aspects. Gazz. Med It Arch Sc Med 1994; 153: 99.
- Stagnaro-Neri M and Stagnaro S. Deterministic Chaos, Preconditioning and Myocardial Oxygenation evaluated clinically with the aid of Biophysical Semeiotics in the Diagnosis of ischaemic Heart Disease even silent. Acta Med Medit 1997; 13: 109.
- Stagnaro-Neri M. et al. Biophysical Semeiotics: deterministic Chaos and biological Systems. Gazz. Med. It. Arch. Sc. Med 1996; 155, 125.
- Stagnaro S and Caramel S. The Principle of Recursive Genome Function: QBS clinical and experimental evidences. JOQBS, 2011. Available at:
http://www.sisbq.org/uploads/5/6/8/7/5687930/prgf_qbsevidences.pdf
- Stagnaro S and Caramel S. A New Way of Therapy based on Water Memory-Information: the Quantum Biophysical Approach. JOQBS, 2011. Available at:
<http://www.sisbq.org/uploads/5/6/8/7/5687930/qbtherapy.pdf>
- Stagnaro S and Caramel S. Quantum Therapy: A New Way in Osteoporosis Primary Prevention and Treatment. Journal of Pharmacy and Nutrition Sciences, February 2012.
- Stagnaro S and Caramel S. The Role of Mediterranean Diet, CoQ10 and Conjugated-Melatonin in Osteoporosis Primary Prevention and Therapy Current Nutrition & Food, Bentham Science 2012; 8(1): 55-62.
- Stagnaro S and Caramel S. Inherited Real Risk of Coronary Artery Disease: pathophysiology, diagnosis and primary prevention. Epidemiology and Cardiovascular Prevention. Brief Communication. 8th International Congress of Cardiology in the internet. Published: 28 October 2013. FAC Federación Argentina de Cardiología. Available at:
http://fac.org.ar/8cvc/llave/tl054_stagnaro/tl054_stagnaro.php
- Stagnaro S and Caramel S. The Inherited Real Risk of Coronary Artery Disease, Nature PG., EJCN, European Journal Clinical Nutrition, Nature PG, 2013 Apr 24. [PubMed indexed for Medline]
- Stagnaro S and Caramel S. Inherited Real Risk of Type 2 Diabetes Mellitus: bedside diagnosis, pathophysiology and primary prevention. Front Endocrinol (Lausanne), 2013. [PubMed indexed for Medline] Available at:
<http://www.frontiersin.org/Diabetes/10.3389/fendo.2013.00017/full>
- Stagnaro S and Caramel S. The Role of Modified Mediterranean Diet and Quantum Therapy in Oncological Primary Prevention. Bentham PG., Current Nutrition & Food Science 2013; 2212-3881.9: 1. Available at:
<http://eurekaselect.com/106105/article>
- Stagnaro S and Caramel S. The Role of Modified Mediterranean Diet and Quantum Therapy in Type 2 Diabetes Mellitus Primary Prevention. Life Science Global. February 2013, Journal of Pharmacy and Nutrition Sciences 2013: 3.
- Waldbaum S and Patel M. Mitochondria, oxidative stress, and temporal lobe epilepsy. Epilepsy Res 2010; 88(1): 23-45.
- Wang X et al. Research on chaotic behavior of epilepsy electroencephalogram of children based on independent component analysis algorithm. Sheng Wu Yi Xue Gong Cheng Xue Za Zhi 2007; 24(4): 835-41.
- Yambe T et al. Chaosanalysis of electro encephalography and control of seizure attack of epilepsy patients. Biomed Pharmacother 2005; 59(1): 236-238.
- Yu JY, Pearl PL. Metabolic causes of epileptic encephalopathy. Epilepsy Res Treat 2013; 124934.
- Zhang X et al. Association of Mitochondrial Letm1 with Epileptic Seizures. Cereb Cortex 2013; May 3.
- Zhao JY et al. Hearing loss and epilepsy may be associated with the novel mitochondrial tRNASer(UCN) 7472delC mutation in a Chinese family. Yi Chuan 2008; 30(12): 1557-62.
- Zhu G et al. Epileptic seizure detection in EEGs signals using a fast weighted horizontal visibility algorithm. Comput Methods Programs Biomed 2014; 115(2): 64-75.
- Zsurka G and Kunz WS. Mitochondrial dysfunction in neurological disorders with epileptic phenotypes. J Bioenerg Biomembr 2010; 42(6): 443-8.
- Zsurka G and Kunz WS. Mitochondrial dysfunction and seizures: the neuronal energy crisis. Lancet Neurol 2015; 14(9): 956-66.

