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Editorial

An electroencephalographer recalls the history of the Federation on the 70th anniversary of its journal, *Clinical Neurophysiology*

1. Introduction

The history of electroencephalogram (EEG) moves from Richard Caton (1842–1926), a physician practicing in Liverpool, who in 1875 presented his findings about electrical phenomena of the exposed cerebral hemispheres of experimental animals. Later the Polish physiologist Adolf Beck published an investigation of spontaneous electrical activity of the brain of rabbits and dogs, that included rhythmic oscillations altered by light; this was the first observation of brain waves. In 1912, Ukrainian physiologist Vladimir Vladimirovich Pravdich-Neminsky published the first EEG and the evoked potential in mammalians (Haas, 2003). In 1914, Napoleon Cybulski and Jelenska-Macieszyna photographed EEG recordings of experimentally induced seizures.

The modern history of Electroencephalography starts with the research of the German physiologist and psychiatrist Hans Berger (1873–1941) who recorded the first human EEG in 1924 and invented the term *electroencephalogram* (giving the device its name) (Berger, 1929). British scientists Edgar Douglas Adrian and Bryan Harold Cabot Matthews in 1934 first confirmed Berger's findings and further developed the EEG (Adrian and Matthews, 1934). In the following years, there was a *crescendo* of discoveries about the EEG. In 1934, Fisher and Lowenback first demonstrated epileptiform spikes. In 1935, Gibbs, Davis, and Lennox described interictal spike waves and the three cycles/s pattern of epileptic 'absence seizures', which began the field of clinical electroencephalography. Subsequently, in 1936 Gibbs and Jasper reported the interictal spike as the focal signature of epilepsy. The same year, the first EEG laboratory opened in US at Massachusetts General Hospital. Franklin Offner, at Northwestern University, developed a prototype of the EEG that incorporated a piezoelectric ink-writer called 'Crystograph'. In 1947, The American EEG Society was founded and the first International EEG congress was held.

In 1949 the first issue of the official Journal of the International Federation of Clinical Neurophysiology was published. It was the birth of *Electroencephalography & Clinical Neurophysiology*.

An electroencephalographer today can nicely recall the scientific journey of *Electroencephalography & Clinical Neurophysiology*, trying to highlight the insights stemming from the content of the first issues - see the enclosed **Index of Vol I - 1949** in the Editorial from Ulf Ziemann in this issue of *Clinical Neurophysiology* (Ziemann, 2019).

2. Part I – The electroencephalography: o*n waves and rhythms*

2.1. Alpha waves

The history of electroencephalogram was not an easy one since its first steps. Quoting from Jasper and Penfield (Jasper and Penfield, 1949) in a twenty years later tribute to Prof. H Berger: *"his sustained vision which carried him through on exemplary series of rigorously controlled systematic experiments on human subjects finally convinced skeptical scientists throughout the world of the validity and value of records of the electrical activity of the human brain*".

The "*sustained vision*" on alpha rhythm was confirmed by Adrian and Matthews (1934) whose work is summarized in a lively Editorial of Brain (Compston, 2010), from which we quote, in which is reported their "*combined satisfaction*" in demonstrating that the Berger rhythm is not an "artifact *"from pulsation of the vessels, activity of pilomotor muscles, tremor of the head or retinal potentials [. . .]The relationship between the Berger rhythm and vision is clear. Nothing abolishes the activity more easily than a visual stimulus, however uninteresting it may be. More specifically it is the recognition of pattern in the central visual field, or the attempt to perceive it, which interferes with the rhythm. Adrian and Matthews try various perturbations to show that the awareness of shape, not the level of illumination, most effectively stop the rhythms [. . .] responding to questions, mental arithmetic, tying and untying knots, hearing, or registering unexpected touch abolishes the potentials, although subjects may habituate to these intrusions; [. . .] Flicker is able to induce rhythms that can occur at a frequency of up to 25 cycle/s [. . .] Sitting in front of an opal bowl with his head covered by a black velvet curtain viewing a 30 watt bulb rotated by a gramophone motor, initially with his eyes closed and the stable 10 cycles/s Berger rhythm in place, Professor Adrian is exposed to the flickering light and 'responds' with a series of potential waves that are mostly synchronous with the flicker. Ideally, these need to be in the range of 10–20/s: at lower rates, the rhythms are unstable; and if too fast, they may halve or double by comparison with frequency of the visual stimulus*".

The 'Flickering' introduce us to the physiopathology of the complex domain of 'Photosensitive Epilepsies': (i) a variety of stimuli – characteristically related to frequency, pattern of stimulation, central vision, intensity of luminance; (ii) a number of etiologies, and (ii) different clinical manifestations (e.g. myoclonus, consciousness, etc) from papio-papio baboon (Killam et al., 1967) to man (Walter and Walter, 1949; Jeavons and Harding, 1975; Takahashi,2002; Duncan and Panayitopoulos,1996). Being *"evidence of the existence of a functional link between the circuits that*

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trigger the visual sensitivity phenomenon and those that generate the posterior alpha rhythm" (Vaudano et al., 2017).

"What causes the beat of these occipital neurons? Adrian and Matthews have already shown that the rhythmic activity observed from the water beetle eye in darkness breaks up when retinal stimulation is not uniform and of reduced brightness (and with all due modesty the Nobel Laureate points out that his own Berger rhythm is more or less identical to that of the water beetle)".

On a more general perspective we can wonder how "similar" waves or rhythms can be observed in extremely different conditions, in the same species: the alpha of the quiet and conscious Sir Adrian, with the eyes closed, and the alpha of a patient in a comatose or vegetative state with the eyes open. No doubts we will read the answer(s) in the next Centenary Editorial of the Journal.

2.2. Beta waves and the rhythm 'en arceau' (or Mu Rhythm)

Jasper and Penfield (1949) described rapid EEG rhythms*: "the beta rhythm (25 per* sec*) is characteristic of the electrical activity from the central region- is not affected by weak visual stimuli which blocked the alpha rhythm, [. . .] It was blocked by tactile stimulation to the contralateral side of the body [. . .] Blocking of the beta rhythm occurs, however only upon the initiation of movement and upon voluntary termination of a posture which the patient has assumed. It is not sustained during a maintained contraction such as clenching the fist, but blocking occurs at the beginning and cessation of the act. Sustained blocking occurs only with continuous consecutive movement or 'readiness to move' before the movement actually begins. These findings would be consistent with the suggestion that impulses reach the precentral gyrus at the beginning and at termination of a posture that is assumed voluntarily*".

Gastaut (Gastaut, 1952; Gastaut et al., 1952) described an 'arch-shaped' rhythm or "rhythm *en arceau*" (the mu-rhythm or 'wicked' rhythm) that was considered by its topography and reactivity as a '*dédoublement*' of beta activity: *"The somatotopic reactivity was precise: the mid rolandic arceau was blocked by hand mobilization, while a "superior – rolandic" (as recorded on vertex area) was blocked mainly by foot mobilization. Most interesting, thinking of performing a voluntary movement was effective to produce a reactivity of both beta and rhythm 'en arceau'*".

In a third article (Gastaut and Bert,1954) on "EEG changes during cinematographic presentation" it was clarified how the rhythm '*en arceau*': "*It also disappears when the subject identifies himself with an active person represented on the screen. This phenomenon is particularly interesting to study during a sequence of film showing a boxing match. A few seconds and at times, less than a second after the appearance of the boxers all type of rolandic activity disappears in spite of the fact that the subject seems completely relaxed and that there is no noticeable change of posture. The relation between the blocking of the 'arceau' rhythm and the image of boxers in action is unquestionable. In the middle of this particular film strip, the camera is suddenly turned from the ring to the spectators in the hall for a few seconds. In many subjects the rhythm 'en arceau' reappears during this short period and vanishes again as the boxers reappear on the screen. The motor characteristic of this identification is made obvious in a subject showing a bilateral rhythm 'en arceau' which is blocked only on the left hemisphere during the boxing match (it is with the right fist that one hits)*".

It was also demonstrated that mental motor action of the phantom limb in an amputee can block the contralateral rhythm '*en arceau*' (Gastaut et al. 1965).

Fifty years later there is a revival of mu rhythm: quoting from Avanzini et al. (2012):

"Although already mentioned by Gastaut (Gastaut et al., 1952) *the reactivity in mu rhythm to the observation of others' action remained for many years neglected. The discovery of mirror neurons, a set of motor neurons that discharge both during action execution and observation, determined a renewed interest in the cortical motor rhythms not only during action execution but also during action observation. A conceptual link between mu rhythm and the mirror neuron activity was first suggested by* Altschuler et al. (1997) *and later confirmed by other researchers. This proposal was based on the reactivity of both mu rhythm and mirror neurons in response to action observation and execution*".

The observation of motor acts determines a modulation of cortical rhythm analogous to that occurring during motor act execution. In particular the cortical motor system closely follows the velocity of the observed movements. This finding provides strong evidence of the presence in humans of a mechanism (mirror mechanism, Rizzolatti and Sinigaglia, 2016) mapping action observation on action execution motor programs. These data constitute the cornerstone of the modern Brain Computer Interface (BCI) techniques. The aim is to allow humans with motor disabilities to interact with the environment, by modulation of brain waves, i.e. electric signals, to control robotic arms (Babiloni et al., 1999) or even a 'full-body' exoskeleton Benabid et al., 2019).

2.3. The Delta waves and Sleep

M. Brazier (1949) introduces us to a third component of the EEG waves' family mainly occurring during physiological sleep: the Delta waves, at 1–4 c/sec, quite 'slow', as compared to waking alpha and beta rhythm. Quoting from Brazier: "*that slow potentials appear in the record when the subject is asleep was one of the first observations to be established in electroencephalography: and since Berger's original demonstration of this change, it has been plentifully confirmed by subsequent workers. . . The electrical activity of the brain during sleep is not the electrical concomitant of 'resting' cells, or of a quiescent brain, but the signal of activity in certain sub-cortical networks*".

We know actually that the EEG activity for most of normal sleep is characterized by a complex achitecture, physiologically composed by macro- and micro-structure - namely the Cyclic Alternating Pattern (Terzano et al., 1985), reflecting the relationship between other (fast) rhytms and slow wave activity (SWA). SWA corresponds, at a cellular level, to slow oscillations of membrane potentials of cortical neurons (Amzica and Steriade, 1998). The amount of EEG SWA is homeostatically regulated, showing an increment during wakefulness and returning to baseline during sleep (Borbély and Achermann, 2005). Slow wave sleep (SWS) has always been associated with restorative and recovery functions, but what these functions do really represent remains unclear. A hypothesis – the synaptic homeostasis hypothesis (Tononi and Cirelli, 2014) – suggests that a consequence of staying awake is a progressive increase in synaptic strength in many brain circuits. Sleep ensures synaptic homeostasis by promoting synaptic weakening/pruning after the increase of synaptic strength occurred during wakefulness. Recent studies in children with a high amount of paroxysmal activity during slow sleep (Cantalupo et al., 2019), failed to show changes of the slope of the SWA, which is a sensitive measure of cortical synaptic strength; in such children, the impaired synaptic homeostasis in the critical period of development may disrupt – often irreversibly – cognitive functions and behavior (Bölsterli et al., 2011; Rubboli et al., 2019; Halasz and Szucs, 2018).

2.4. A new language

With the end of World War II, electronic devices and human competences became available in EEG and neurophysiological lab-

oratories. The "Electroencephalography", now technically validated and scientifically recognized, became a relevant and trendy investigation in different clinical and neuroscientific domains: psychology, neurology, psychiatry, pharmacology, and in any condition susceptible or suspected to modify the brain function (see Niedermeyer's Electroencephalography). A wealth of new rhythms were observed from scalp electrodes, and carefully analyzed and 'validated' by subdural and intracerebral recordings in patients (mainly epileptic). The introduction of microprocessors and computers also allowed to average EEG signals triggered by an external stimulus giving then the opportunity to record Evoked Potentials to visual, acoustic, somatosensory stimuli (see Desmedt and Cheron, 1980; Rossini et al., 1987). The possibility to record EEG and stimuli on tape opened the door to analyze eventual potentials *preceding* a given event by 'back-averaging' a number of repetitive events; the Bereitschaft potentials preceding a movement as well as the cortical spike preceding a myoclonic jerk were, for the first time, recordable by back-averaging procedures (Shibasaki and Hallett, 2006; Pfurtscheller and Aranibar, 1978).

In the – how useful! – "Glossary of terms most commonly used by clinical electroencephalographers" (Chatrian et al., 1974), we find two variants of '*posterior 4 c/sec rhythm*'; six variants of the '*alpha rhythm*', six variants of '*spikes*', thirteen '*complexes*'. The new EEG findings were discussed for years, usually in lively and friendly moods, at times hotly, even in the cold surroundings of 'Alpine sky meeting' (Miletto, 1964):

"*Why* – claimed an astonished J. Stevens (USA) – *the '14-and-6 positive spikes' are considered so relevant and frequent in the US while are ignored in Europe? 'We never saw them' was the answer of Gastaut and Remond*"*!*

In turn, G. Walter stated of having "*never seen*" the 'rhythm *en arceau*' (the mu rhythm). In the same 'sky meeting' in a discussion on 'spike and waves without epilepsy', the soft spoken, well-mannered gentleman, Prof. Loiseau (Bordeaux) "*rose to protest with vehemence against the use of the term epileptic to describe a paroxysmal tracing*" (Miletto, 1964, page 345).

Unavoidable to reread the Grey Walter (1949) 'Commentaries' on *"Coming to terms with brain waves: the inevitable ambiguity of language is the joy of poets, the despair of scientist; [. . .] We need not to be afraid of naming phenomena we do not understand. It would be ludicrous to use 'alpha rhythm' to mean any activity at about 10c/sec, but equally absurd to have to write 'rhythmic oscillations of potential difference at 8–13c/sec, with an amplitude of 5–100 μv in the parietal occipital region, normally attenuated by opening of the eyes and/or mental alertness'. Perhaps we all know what we mean by alpha rhythm, but do we all mean the same things? And what about the others names, for some of which I must accept responsibility?*"

Indeed G. Walters was responsible for the naming of theta and delta waves.

It has been said: "*The confusion arises not from things but by the names we call them*".

3. Part II - Clinical electroencephalography

3.1. Epilepsy

It is not by chance that the first article of the first number of the first year of our Journal 'Electroenceph. Clin. Neurophysiol' is authored by W. Penfield (1949): 'Epileptic Manifestation of Cortical and subcortical Discharge'. It is a refreshing pleasure to quote verbatim the first sentences:

> *"The validity of the Jacksonian conception of an ictal ganglionic discharge has been verified by the clinical use of the electroencephagraph. But the disturbance of electrical rhythms of the brain during a seizure is only one of the manifestations of an attack. It does not tell the whole story of epilepsy. The clinical picture is also important and we must enquire into the pathological cause.*"

From Penfield there is an additional warning. *"The attempt to classify cases of epilepsy on electroencephalographic evidence alone tend to stop the study of a case before it is complete"*. H. Jasper (1949) a few pages later in the final comment of his article 'Electrical signs of epileptic discharge' furtherly comments: *"The clinical pattern of an epileptic seizure is not closely related to the form of associated EEG disturbance but rather to the functional area of the brain primarily involved and the functional characteristics of the neuronal circuits involved in the path of spread*".

There is an exception to these rules, i.e. an epileptic condition that can be diagnosed 'only' by the electroencephalographic recording of the seizure:

"Thanks to the pioneer work of Gibbs, Davis and Lennox (1935) an expert is now able to recognize a three per second rhythm as characteristic of idiopathic (essential genetic) epilepsy. Bursts of such rhythms accompany the minor lapses of consciousness which have been called petit mal".

How still fascinating the spike-and-wave discharge of 'Petit Mal', an imposing "*cathedral with pinnacles and arches*", so rhythmically repeated with a stereotyped common pattern and yet with so complex correlations, with regard to unitary cell discharges (Creutzfeldt, 1963), to spike morphology (Weir, 1965; Rubboli et al., 1995, 2006), to concomitant DC-shift (Goldring and O'Leary, 1957; Chatrian et al., 1968), and to motor manifestations (Speckmann and Elger, 1983; Stefan et al., 1982; Tassinari et al., 1998).

For years – up to now – and despite the contribution of experimental works (Gloor, 1979; Avanzini et al., 2000; Noebels et al., 2012) the issue is still complex (Loiseau and Cohadon, 1970; Duncan and Panayiotopoulos, 1995; Hirsch et al., 2006) even for the apparently simple "Petit Mal Absence".

3.1.1. Automatism and finger prints

"*The problem of epileptic automatisms is of great importance. . .They may follow any type of generalized convulsive seizure. . . but occur chiefly in seizures which arises in the temporal region, and also as the result of petit mal discharge and of anterior frontal region without major convulsion. Actually in all automatisms there is a paresis of a portion of the highest level of neural integration, and therefore all automatism may be said to be psychoparetic not psychomotor*" (Penfield, 1949).

It is implied in a Jacksonian perspective that the 'paresis' can lead to a 'release' of '*some physiological mechanisms involved in epileptic automatisms*', as discussed, in his Lennox Lecture by Jaspers (1964).

Along these lines, we proposed (Tassinari et al, 2009) that the 'physiological mechanisms' such as those occurring in 'alimentary or locomotor cursive automatisms' may be the stereotypical motor sequences generated by Central Pattern Generators. Central Pattern Generators (Grillner and Wallen, 1985) are functional neuronal aggregates present in spinal cord, pons and mesencephalon (see also Reticular Formation) and under control of cortical – mainly frontal – and subcortical – mainly putamen – structures. When this control becomes impaired the automatic, stereotyped, rhythmic motor sequences – or 'automatisms' – occur.

Significantly, Bancaud and Talairach (1991), in their report of a stereo-EEG investigations in 233 epileptic patients, stated: *"It would be very unusual to have two absolutely identical seizures in similar patients or even in the same patient. However, at least in the temporal lobe epilepsies, we have to admit – in a significant number of patients – an anatomo-electro-clinical profile strangely similar*

and extremely homogeneous as if a seizure event, in each subject, could be programmed as a fingerprint".

3.1.2. The Montreal-Marseille axis

Gastaut, with evident pleasure, often reminded us that he considered W. Penfield as one (the other being W. Lennox) of his teachers. The axis Montreal-Marseille was essentially lying on few basic common ideas:

1. There is an Idiopathic Epilepsy "*which would be due to an abnormality of cerebral physiology with a genetic background*". The prototype of such condition is the epilepsy with Petit Mal absences with the 3 c/sec. spike and waves (vide supra).
2. The others forms of Epilepsies should be defined on clinical ground first, then with the investigations relevant to define ictal and interictal EEG patterns, the etiologies and the involved brain areas. These information will constitute the backbone for the classification of seizures and epilepsies, 'personalized' according to the finalities of the various classifications and the specific interests and knowledges (medical or surgical therapy; genetic investigations; triggering mechanisms etc) (Gastaut and Broughton,1972; Gastaut and Tassinari, 1975).

In Gastaut's own word *"my approach (to Epilepsy) was to be based on the three disciplines in which I had been trained: clinical neurology, electroencephalography and neuropathology*". Gastaut used all his knowledges to produce clinical neurophysiology. In 1952 he wrote: *"It now seems necessary to go beyond the static conditions of EEG recording, as performed with eyes closed, to try and study the bioelectrical modifications in conditions as close as possible to those of life itself, i.e. with the eyes opened, in front of diverse and changing situation*" (in this way he discovered the lambda waves and the rhythm '*en arceau*'; vide supra).

Gastaut was very curious on the limit of impatience at times: late on the night, on his way home, he would appear in the EEG lab with his wife Yvette, both elegantly dressed, asking: *"Did you record the seizures? the enuresis? the apnoeas?*" If so he would stay, looking at the 'polygraphic records'.

Polygraphic recording became indeed one of the legacies of the Marseille School (Broughton edr., 1982) and of its disciples. The polygraphic recordings in different domains of neurosciences further document and expand what has been suspected or collected on clinical ground (is it a myoclonus or a spasm? has the patient sleep apnea and of which type? is there ictal bradycardia or tachycardia? etc) or by previous EEG recordings. Consequently, various parameters are associated with the EEG recording, to simultaneously evaluate muscular activity, vegetative functions, respiration, eye movements or degree of performance/responsiveness, etc; tailoring each polygraphic study to the specific clinical problem that the researcher/clinician is investigating.

The need of establishing and maintaining – as much as possible – a continuous link with the clinical aspects was one of the efforts of Gastaut, primarily a clinician, as Secretary, President, and Past President of the Federation.

An important albeit difficult balance, though, and a not new problem. In the 1962 Editorial of the journal, pag. 604, we read a complaint: too few 'clinical' papers [..]? The answer was "*To those who say that the Journal is insufficiently clinical it is only possible to replay that the remedy is in their hands [...]*".

Over the years, other neurophysiological techniques have been developed and implemented in the clinical evaluation, joining electroencephalography in the diagnostic armamentarium of the clinician/researcher. Appropriately, in a more general perspective *"to foster and disseminate information on all aspects of Neurophysiology, we have changed [...] the name of the Journal to "Clinical Neurophysiology" to represent all fields of human physiology and pathophysiology*" (Celesia and Rossini, 1999).

3.2. Brain Stem Reticular Formation and Behaviors

The reactivity of the above-described rhythms requires a functional interplay to connect the different rhythmogenic areas and networks. This function is that of the 'Brain Stem Reticular Formation', as described by Moruzzi and Magoun in Vol. I n.1 of the EEG Journal, 1949 (Moruzzi and Magoun, 1949).

The Reticular Formation (RF) is a neuronal network in the central part of neuroaxis, elongated from inferior bulbar pontine region to posterior hypothalamus. As by Magoun (1958) we can think of the RF as a transactional center acting as an integrator and modulator of other systems in relation also to its own internal excitability, in turn depending of peripheral afferences, humoral and metabolic conditions and signals and on reciprocal interplay in the cortex.

Quoting from Moruzzi and Magoun (1949) the RF stimulation *"evokes changes in the EEG consisting in the abolition of synchronized discharges and introduction of low voltage fast activity in its place. The effect of Reticular Stimulation is strikingly like Berger's alpha wave blockade or any arousal reaction... The EEG modifications reflect the transition from sleep to wakefulness or from relaxation to alertness*". It became thus possible to correlate (a) the EEG rhythms reactivity with the processes of attention, habituation and plasticity, and of sleep and arousals: that is the basis of behavior (Jouvet, 1963); and concomitantly (b) to correlate the clinical manifestations with the motor and autonomic events.

In this context, it became rewarding to have chronic experimentations, allowing to compare the behaviors in animal and in man in wakefulness and sleep in a more appropriate ethological approach (Jouvet, 1978; Hess, 1944; Moruzzi, 1969).

At times, the clinical conditions could offer the initial cue for experimental investigations: the eye movements during the dreaming of the students in the Dement and Kleitman (1957) sleep laboratory were the first clinical observations leading subsequently to experimental definition of a 'new' stage of sleep, paradoxical or REM (Hobson et al., 1974; Jouvet and Michel, 1960).

Understandably disorders of the RF, "*the modulator or transactional system*" attuning vigilance, muscular and vegetative activities can occur during sleep-to-wakefulness transition.

It is matter – during slow sleep – of the parasomnias (e.g. pavor nocturnus, enuresis, somnambulism), rightly considered as 'arousal disorders' (Broughton, 1968), or it is matter of REM-related sleep events: violent behaviors, sleep paralysis, cataplectic attacks, and narcoleptic episodes, examples of brain-body 'dissociated' behavioral disorders (Plazzi et al., 2011; Schenck et al., 1997).

4. Unending conclusions

If Hans Berger were with us now, trying to evaluate the scientific accomplishments stemming from his *Elektrenkephalogramm*, would he be satisfied? It is certainly so, but not completely in some respect. Quoting from his last paper (Berger, 1938): *"My preferred interest was the psychophysiology [...] and the connection between cerebral processes and the psychic manifestations*". In this respect he himself considered the Electroencephalogram somewhat disappointing. We can speculate, 70 years later, how the mirror neuron system (Rizzolatti and Sinigaglia, 2016) can offer a partial answer to Berger psychophysiology; even if, in the last decades, a rich scientific production has focused on 'Event Related potentials' and to those brain waves (e.g. the P300) which are mainly produced by a cognitive/psychological process. In recent years neuroscientists started to explore 'brain connectivity' as the cornerstone for better

understanding neuropsychiatric diseases and normal brain functions. Within this frame advanced mathematical analysis of EEG signals (e.g. via graph theory) are progressively opening new avenues for a better interpretation of the working brain, of its dynamics as well as of its network organization in the healthy and disease conditions (Rossini et al., 2019). However, Berger went on, quoting Mosso (1883): *"As far as we apply instruments to evaluate the human brain functions, we should hope to understand the physical basis of consciousness. And even if do not reach satisfactory results we are on the right track"* (as quoted by Fischgold et al., 1963).

Along this track, neuroscientists from all over the world went in a seventy year old journey as by testimonial evidence of our International Journal*: Clinical Neurophysiology.*

On perusal of our Journal I got the feeling that the people on the Mosso track became progressively a family, a family of friends in a common *Bergerian* pursuit: how *"cerebral processes became psychic manifestations?"* Neuroscientists, as novel untiring Hermes, are striving for data collection and interpretation, in an unremitting hermeneutic endeavor, *"a continuous tension between the will of listening and the will of conjecture"* (Ricoeur, 1965).

Declaration of Competing Interest

None.

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