CORTICAL ACTIVITY VARIATIONS DURING VARIOUS MOTOR TASKS

MASTER THESIS

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ACRONYMS

MRCP movement-related cortical potential

FES functional electrical stimulation

fMRI functional magnetic resonance imaging

BCI brain-computer interface

CNS central nervous system

VOL voluntary

FESVOL voluntary movement performed in conjunction with FES

EEG electroencephalography

ERD event-related desynchronization

ERS event-related synchronization

ERP event-related potential

MEG magnetoencephalography

LTP long-term potentiation

M1 primary motor cortex

ECG electrocardiograph

CNS central nervous system

AP action potential

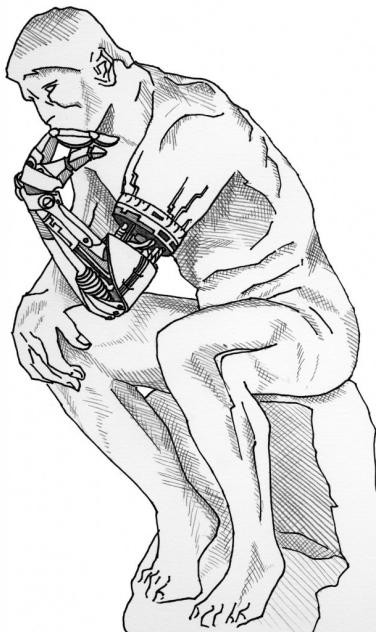
FET functional electrical therapy

1

INTRODUCTION

The merger of systems neurophysiology and engineering has resulted in approaches to link brain activity with man-made devices to replace lost sensory and motor function. The excitement in this field is based not only on the prospect of helping a wide range of patients with neural disorders, but also on the certainty that this new technology will make it possible to gain scientific insight into the way populations of neurons interact in the complex, distributed systems that generate behavior [1].

Capturing motor intention and executing the desired movement form the basis of brain-computer interface (BCI) prosthetics, which need to decode intention in order to restore motor ability or communication to impaired individuals. Motor impairment after stroke is common following damage to areas of the brain normally involved in planning and executing motor commands. Due to limited regeneration of damaged tissue in adults, the real improvement in motor function observed after stroke is a consequence of reorganization of the surviving elements of the motor network [2]. The main way of improving lost motor functions is neurorehabilitation [3, 4, 5]. Overall, approach is effective and the benefit of strategies aimed at helping patients adapt to impairment is well proven [6]. A better understanding of the underlying mechanisms of recovery (or deterioration) of function after a central nervous system (CNS) lesion, as well as those leading to maladaptive or unfavourable outcomes, would be essential for directing specific and effective rehabilitative strategies as well as avoiding potentially harmful interventions [7].



Biological basis of post-stroke motor function recovery, particularly one occurring after following a rehabilitation therapy, has long remained elusive. Success of a treatment depends on the ability to drive functionally relevant reorganization in surviving brain regions and networks. This varies dramatically across patients. Neuroimaging techniques are used to assess how treatments interact with functioning parts of the anatomy. They reveal active mechanisms and allow targeted application of therapies based on neuroscientific principles. functional electrical stimulation (FES) is used in the rehabilitation therapy of patients after stroke to improve their motor abilities. It uses electrical currents to activate muscle nerves to produce either isometric or concentric contractions of the treated muscles [8]. It appears to facilitate recovery in an additive or interactive way. Results from studies where therapeutic FES was applied in acute and in chronic hemiplegia, suggest better recovery of function compared with conven-

tional treatment [9]. Neuroimaging studies demonstrated that passive movements result in cortical reorganization, meaning mere external treatment caused changes in functional brain activations to resemble the ones elicited by active movements. Applying electrical stimulation with voluntary movement provides an intensive traffic of neural information towards the brain, which occurs in a predictable manner, and that this may promote neural plasticity. However, the functional brain correlates of therapeutic FES have yet to be determined. Having a good understanding of how therapeutical FES may interact with the central nervous system may therefore be crucial to improve and optimize the treatment.

In this thesis I assessed cortical activation of healthy individuals performing simple motor tasks. Using multichannel surface electroencephalography (EEG) I analyzed alpha and beta oscillatory activity and movement-related cortical potential (MRCP) associated with three grasp tasks: voluntary (VOL) self-paced movement, movement produced using only FES and voluntary movement performed in conjunction with FES (FESVOL), in which a movement is initiated using FES and perform with a combination of FES and VOL. A recent study has shown that there are differences in brain activity with these types different movement initiation, and that they are detectable using functional magnetic resonance imaging (fMRI) [10]. This hypothesis follows from the lack of the "attention-to-move" in FESVOL and FES and in the idea that the central processing of peripheral input would be reflected differently in the modulation of cortical alpha and beta oscillatory activity and MRCPs. A follow-up study had hypothesized that these differences are measurable using EEG as well and showed promising results [11]. However, this study suffered from several oversights in the experimental procedure. As these results could provide very important insights in the working of our brains, I tried to replicate that research with corrected and improved experimental procedure. True purpose of this research was to contribute to the understanding of the neural consequences of the applied therapeutic FES to promote reaching and grasping. An important question that requires further research is whether patients with interrupted efferent and afferent pathways will have the same alpha and beta pattern during hand movement as healthy subjects.

This thesis is presented in five chapters. This was the introductory chapter, serving to inform the reader of the goal of the experiment, the history of similar experiments, introduce terms used throughout the thesis and formulate research questions that are to be answered by the thesis. Second chapter explains the theoretical concepts behind the research: basics of the electrical acitivity of the brain, cortical plasticity, EEG and FES. In the third chapter, the experiment is described, as are the methods used for data acquisition and signal processing.

Results of the experiment are listed in the fourth chapter, and discussed in the fifth.

2

THEORETICAL FRAMEWORK

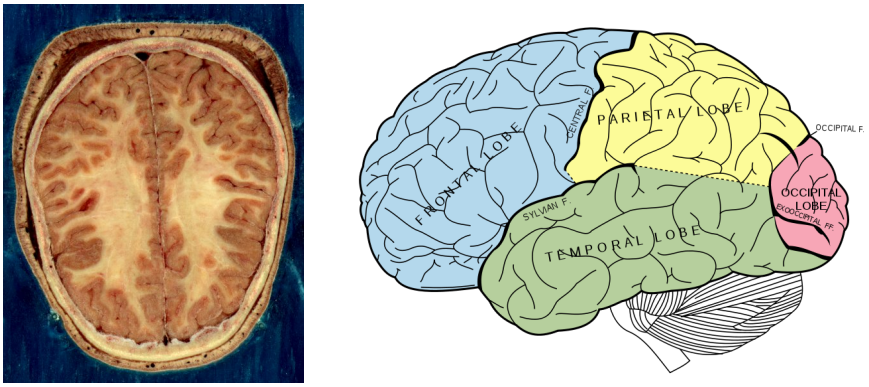
2.1 HUMAN BRAIN ACTIVITY

The CNS has a wide array of functions: receiving sensory input, storing memories, coordinating motor plans, maintaining posture, and generating consciousness and higher thought. The nervous system accomplishes this diversity of functions with one key feature: it can change and adapt. In this way, characteristics can be tuned to the task at hand and new properties can be acquired. This ability of the nervous system to change is perplexing as the adult nervous system generates relatively few new cells. The brain is the center of the human nervous system. It has the same general structure as the brains of other mammals. Estimates put the number of neurons in the human brain in the range from 80 to 120 billion [12, 13]. The cerebral hemispheres form the largest part of the human brain and are situated above most other brain structures. They are covered by cerebral cortex whose surface is folded, such that more than two-thirds of it is buried in the deep grooves (sulci) and wrinkles (gyri). The cerebral cortex is nearly symmetrical, with left and right hemispheres that are approximate mirror images of each other. Anatomists conventionally divide each hemisphere into four "lobes", the frontal lobe, parietal lobe, occipital lobe, and temporal lobe. Underneath the cerebrum lies the brainstem, resembling a stalk on which the cerebrum is attached. At the rear of the brain, beneath the cerebrum and behind the brainstem, is the cerebellum, a structure with a horizontally furrowed surface that makes it look different from any other brain area.

According to a recent popular account of what makes us unique, we have brains that are bigger than expected for an ape, a neocortex three times bigger than predicted for our body size, neocortex areas and the cerebellum that are larger than expected – and the list goes on [14]

The functions of the brain depend on the ability of neurons to transmit electrochemical signals to other cells, and their ability to respond appropriately to electrochemical signals received from other cells. As a side effect of the electrochemical processes used by neurons for signaling, brain tissue generates electric fields when it is active. When large numbers of neurons show synchronized activity, the electric fields that they generate can be large enough to detect outside the skull, using EEG [15]. These recordings, along with recordings made from electrodes implanted inside the brains of animals such as rats, show that the brain of a living animal is constantly active, even during sleep [16]. Each part of the brain shows a mixture of rhythmic and nonrhythmic activity, which may vary according to behavioral state. In mammals, the cerebral cortex tends to show large slow delta waves during sleep, faster alpha waves when the animal is awake but inattentive, and chaotic-looking irregular activity when the animal is

actively engaged in a task. Relating these population-level patterns to the computational functions of individual neurons is a major focus of current research in neurophysiology.



(a) Bisection of the head of an adult man

(b) The four lobes of the cerebral cortex

Figure 1: The human brain

Spontaneous activity is brain activity in the absence of an explicit task, such as sensory input or motor output, and hence also referred to as resting-state activity. It is opposed to induced activity, i. e. brain activity that is induced by sensory stimuli or motor responses. The term ongoing brain activity is used in EEG and magnetoencephalography (MEG) for those signal components that are not associated with the processing of a stimulus or the occurrence of specific other events, such as moving a body part, i. e. events that do not form evoked potentials/evoked fields, or induced activity. Spontaneous activity is usually considered to be noise if one is interested in stimulus processing. Spontaneous activity may be informative regarding the current mental state of the person (e. g. wakefulness, alertness) and is often used in sleep research. Certain types of oscillatory activity, such as alpha waves, are part of spontaneous activity. Statistical analysis of power fluctuations of alpha activity reveals a bimodal distribution, i. e. a high- and low-amplitude mode, and hence shows that resting-state activity does not just reflect a noise process. In case of fMRI, spontaneous fluctuations in the Blood-oxygen-level dependent (BOLD) signal reveal correlation patterns that are linked to resting states networks, such as the default network. The temporal evolution of resting state networks is correlated with fluctuations of oscillatory EEG activity in different frequency bands. Ongoing brain activity may also have an important role in perception, as it may interact with activity related to incoming stimuli. Indeed, EEG studies suggest that visual perception is dependent on both the phase and amplitude of cortical oscillations. For instance, the amplitude and phase of alpha activity at the moment of visual stimulation predicts whether a weak stimulus will be perceived by the subject [17]. Oscillations have been

commonly reported in the motor system. Pfurtscheller and colleagues found a reduction in alpha (8–12 Hz) and beta (13–30 Hz) oscillations in EEG activity when subjects made a movement [18]. As this is the brain activity of interest in this thesis, it is covered in detail in the latter parts of this chapter starting on page 15.

From a philosophical point of view, what makes the brain special in comparison to other organs is that it forms the physical structure that generates the mind. Understanding the relationship between the brain and the mind is a great challenge. It is very difficult to imagine how mental entities such as thoughts and emotions could be implemented by physical entities such as neurons and synapses, or by any other type of mechanism. The mechanisms by which brain activity gives rise to consciousness and thought have been very challenging to understand: despite rapid scientific progress, much about how the brain works remains a mystery. The operations of individual brain cells are now understood in considerable detail, but the way they cooperate in ensembles of millions has been very difficult to decipher [19]. If we want to use the signals from the brain to control man-made devices it is important to understand at least some ways in which the brain exerts its control on the rest of the body. These devices could range from artificial limbs controlled in by thoughts alone, vastly superior visual and auditory prosthesis than the ones we have now to various implant straight from science fiction.

One of the most influential early contributions to the field of computational neuroscience was a 1959. paper titled *What the frog's eye tells the frog's brain*. The paper examined the visual responses of neurons in the retina and optic tectum of frogs, and came to the conclusion that some neurons in the tectum of the frog are wired to combine elementary responses in a way that makes them function as "bug perceivers" [20]. A few years later David Hubel and Torsten Wiesel discovered cells in the primary visual cortex of monkeys that become active when sharp edges move across specific points in the field of view—a discovery that eventually brought them a Nobel Prize. Theorists have worked to understand these response patterns by constructing mathematical models of neurons and neural networks, which can be simulated using computers. Some useful models are abstract, focusing on the conceptual structure of neural algorithms rather than the details of how they are implemented in the brain; other models attempt to incorporate data about the biophysical properties of real neurons. David Marr's work focused on the interactions between neurons, suggesting computational approaches to the study of how functional groups of neurons within the hippocampus and neocortex interact, store, process, and transmit information. No model on any level is yet considered to be a fully valid description of brain function, though.

2.2 ELECTROENCEPHALOGRAPHY

2.2.1 *History*

EEG is a noninvasive technique for the recording of electrical activity along the scalp produced by the firing of neurons within the brain [21]. Neurons, or nerve cells, are electrically active cells that are responsible for carrying out the brain's functions. Neurons create action potentials, discrete electrical signals that travel down axons and cause the release of chemical neurotransmitters at the synapse, which is an area of near contact between two neurons. The neurotransmitter causes an electric current within the dendrite or of the post-synaptic neuron. This neuron then synapses on other neurons, and so on. The activity of a single cortical neuron cannot be measured on the scalp due to thick layers of tissue (fluids, bones, and skin) which attenuate the electrical signal when it propagates toward the electrode. However, the joint activity of millions of cortical neurons, at a depth down to several millimeters, produces an electrical field which is sufficiently strong to be measured on the scalp [22].

In 1929. Hans Berger recorded human EEG in the duration of one to three minutes on photographic paper, and it included the description of the alpha rhythm as the major component of the EEG signals. Subsequent research revealed a connection between the EEG and the

Richard Caton (1842–1926), a Liverpool-based physician, was the first person to present his findings about electrical phenomena of the exposed cerebral hemispheres of rabbits and monkeys in the British Medical Journal in 1875 [23]

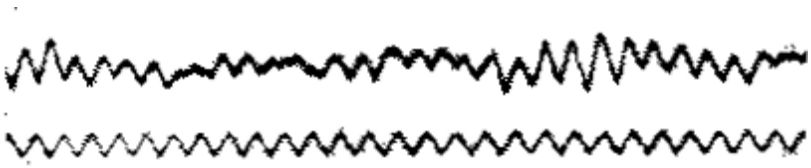


Figure 2: The first human EEG recording obtained by Hans Berger and published in 1929. The upper tracing is EEG, and the lower is a 10 Hz timing signal.[24]

physiological states, such as sleep and wakefulness. Berger placed the electrodes on the front and back of the head as a measure of global cortical activity. In 1958. a committee recommended a specific system of electrode placement for use in all laboratories under standard conditions [25]. Their recommendation was the system now known as the International 10-20 system. This system ensures that the naming of electrodes is consistent across laboratories.

2.2.2 *Method*

In modern conventional scalp EEG, the recording is obtained by placing electrodes on the scalp with a conductive gel or paste, usually after preparing the scalp area by light abrasion to reduce impedance due to dead skin cells. Some systems use caps or nets into which elec-

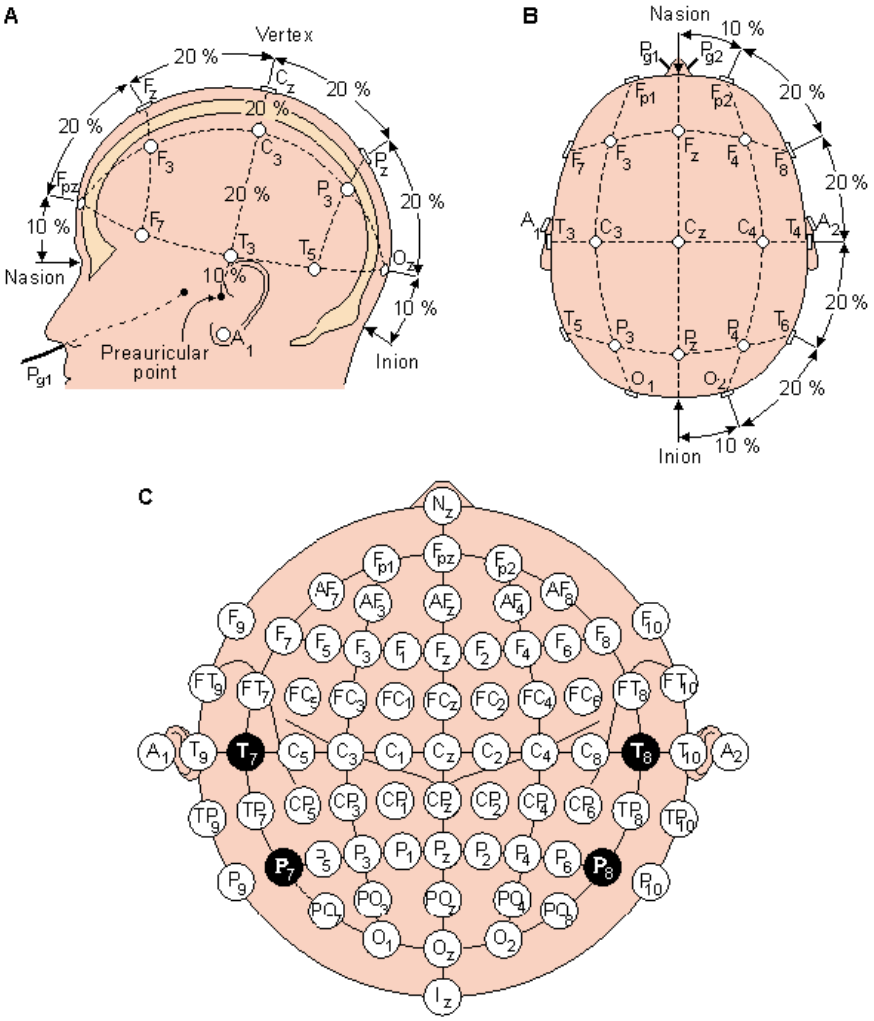


Figure 3: The original 10-20 system included only 19 electrodes (panels A and B). Later on, extensions were proposed so that now you can place over 70 electrodes in standard positions (panel C). This extension also renamed four electrodes (marked in black in the figure); the original names were: T3, T5, T4, and T6 for T7, P7, T8, and P8, respectively.

trodes are embedded; this is particularly common when high-density arrays of electrodes are needed. Each electrode is connected to one input of a differential amplifier. Since a typical adult human EEG signal is about 10 ⁻V to 100 ⁻V in amplitude when measured from the scalp [26], these amplifiers amplify the voltage between the active electrode and the reference typically 1,000–100,000 times, or 60–100 dB of voltage gain. Most EEG systems are digital, and the amplified signal is digitized via an analog-to-digital converter, after being passed through an anti-aliasing filter. Analog-to-digital sampling typically occurs at 256–512 Hz in clinical scalp EEG; sampling rates of up to 20 kHz are used in some research applications. The digital EEG signal is stored electronically and can be filtered for display. Typical settings for the high-pass filter and a low-pass filter are $0.5 - 1$ Hz and 35–70 Hz, respectively. The high-pass filter typically filters out slow artifact, such as electrogalvanic signals and movement artifact, whereas the low-pass filter filters out high-frequency artifacts, such as electromyographic signals. An additional notch filter at 50 Hz is typically used to remove artifact caused by electrical power lines.

Since an EEG voltage signal represents a difference between the voltages at two electrodes, the electrodes may be set up and connected in one of several ways. The representation of the EEG channels is referred to as a montage. These are:

- BIPOLAR MONTAGE. Each channel represents the difference between two electrodes. The entire montage consists of a series of these channels. For example, the channel "F3-C3" represents the difference in voltage between the F3 electrode and the C3 electrode.

- REFERENTIAL MONTAGE. Each channel represents the difference between a certain electrode and a designated reference electrode. There is no standard position for this reference; it is, however, at a different position than the "recording" electrodes. Midline positions are often used because they do not amplify the signal in one hemisphere vs. the other. Another popular reference is "linked ears," which is a physical or mathematical average of electrodes attached to both earlobes or mastoids (A1 and A2).

- AVERAGE REFERENCE MONTAGE. The outputs of all of the amplifiers are summed and averaged, and this averaged signal is used as the common reference for each channel.

2.2.3 *Advantages and disadvantages*

Several other methods to study brain function exist, including functional magnetic resonance imaging (fMRI), positron emission tomography, MEG, Nuclear magnetic resonance spectroscopy, Electrocorticog-

raphy, and Single-photon emission computed tomography. Neither of these techniques is better than the others, each has multiple advantages and disadvantages over others. Some of the advantages of EEG are:

- Hardware costs are significantly lower than those of all other techniques.
- EEG sensors can be used in more places than fMRI, SPECT, PET, MRS, or MEG, as these techniques require bulky and immobile equipment.
- EEG has higher temporal resolution - milliseconds, rather than seconds - it can, in fact, take as many as 2000 samples per second (20000 in some applications). Only MEG rivals these speeds.
- EEG is silent, which allows for better study of the responses to auditory stimuli
- EEG does not aggravate claustrophobia, unlike fMRI, PET, MRS, SPECT, and sometimes MEG [27].

And the disadvantages:

- Significantly lower spatial resolution. fMRI, for example, can directly display areas of the brain that are active, while EEG requires intense interpretation just to hypothesize what areas are activated by a particular response [28].
- EEG determines neural activity that occurs below the upper layers of the brain (the cortex) very poorly.
- Signal-to-noise ratio is very poor, so sophisticated data analysis and relatively large numbers of recordings are needed to extract useful information from .EEG.

It is probably only through the integration of different neuroimaging techniques that it will be possible to overcome the pitfalls of each methodology, in the study of normal brain function [29]

2.2.4 *Noise reduction*

The reasons for the poor signal-to-noise ratio are numerous and can be either internal or external by nature. Some of these artifacts can be useful in various applications. The EOG signals, for instance, can be used to detect [30] and track eye-movements, which are very important in polysomnography, and is also in conventional EEG for assessing possible changes in alertness, drowsiness or sleep. Modern EEG acquisition commonly includes a one-channel electrocardiograph (ECG) from the extremities because ECG artifacts are quite common and can be mistaken for spike activity. This also allows the EEG to identify cardiac arrhythmias that are an important differential diagnosis to syncope or other episodic/attack disorders. Glossokinetic artifacts

are caused by the potential difference between the base and the tip of the tongue. Minor tongue movements can contaminate the EEG, especially in parkinsonian and tremor disorders.

In addition to artifacts generated by the body, many artifacts originate from outside the body. Movement by the patient, or even just settling of the electrodes, may cause electrode pops, spikes originating from a momentary change in the impedance of a given electrode. Poor grounding of the EEG electrodes can cause significant 50 Hz artifact, depending on the local power system's frequency. In clinical applications, a source of possible interference can be the presence of an IV drip; such devices can cause rhythmic, fast, low-voltage bursts, which may be confused for spikes.

Recently, independent component analysis techniques have been used to correct or remove EEG contaminates [30, 31, 32]. These techniques attempt to "unmix" the EEG signals into some number of underlying components. There are many source separation algorithms, often assuming various behaviors or natures of EEG. Regardless, the principle behind any particular method usually allow "remixing" only those components that would result in "clean" EEG by nullifying (zeroing) the weight of unwanted components. Fully automated artifact rejection methods, which use ICA, have also been developed [33].

2.2.5 *Signal analysis*

Frequency is one of the most important criterions for assessing abnormality in clinical EEG and for understanding functional behaviour in cognitive research. With billions of oscillating communities of neurons as its source, the human EEG potentials are manifested as aperiodic unpredictable oscillations with intermittent bursts of oscillations having spectral peaks in certain observed bands: $0.1 - 3.5$ Hz (delta, δ), $4 - 7.5$ Hz (theta, ϑ), $8 - 13$ Hz (alpha, α), $14 - 30$ Hz (beta,β) and > 30 Hz (gamma,γ). Activity that is either less than 0.5 Hz or greater than 20 Hz is often assumed to be of limited clinical utility. EEG has been used for many purposes besides the conventional uses of clinical diagnosis and conventional cognitive neuroscience. Long-term EEG recordings in epilepsy patients are used for seizure prediction. Neurofeedback, which displays electroencephalography in realtime to illustrate brain activity, often with a goal of controlling central nervous system activity, remains an important extension, and in its most advanced form is also attempted as the basis of BCIs.

2.3 NEUROPLASTICITY

Neuroplasticity using the broadest definition is the ability of neurons (or the nervous system) to rearrange their anatomical and functional connectivity and properties in response to environmental input.

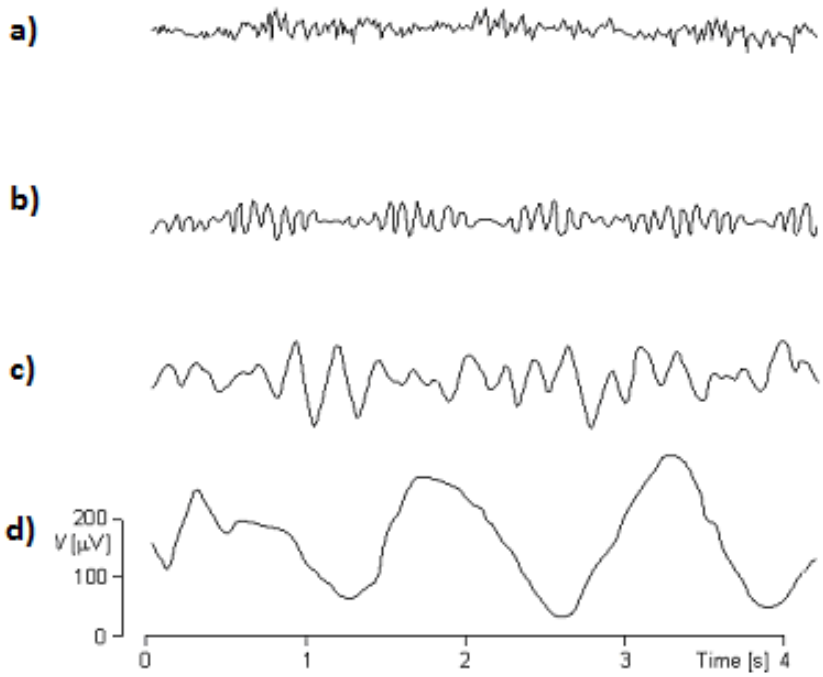


Figure 4: EEG rhythms: a) beta; b) alpha; c) theta; d) delta

This broad definition encompasses functional, structural, physiological and molecular changes. The most interesting form of neuroplasticity is neuroplasticity that obeys Hebbian rules as first described by Daniel Hebb in 1949.:

> When an axon of cell A is near enough to excite cell B and repeatedly or persistently takes part in firing it, some growth process or metabolic change takes place in one or both cells such that A's efficiency, as one of the cells firing B, is increased.

This description has often been simplified as: "neurons that fire together, wire together"

Hebbian principles form the mathematical basis of neural network models and provide a principle that governs neuroplasticity, allowing synapses to retain a memory of previous activity.

For many years, the CNS has been viewed as a rigid structure with little capacity for modification and adaptation. In the last couple of decades, however, there has been a paradigm shift characterized by the understanding of the CNS as a plastic organ, capable of adaptation or modification when confronted with environmental challenges or lesions [34]. The first clues for the molecular basis of how a nervous system can display neuroplasticity and adapt its motor behaviour was found in the invertebrate sea-slug, *Aplysia californica*, by a group of researchers in the sixties [35]: changes in synaptic properties were shown to occur after the *Aplysia californica* had acquired a memory. This led to the discovery of long-term potentiation (LTP) in the mammalian hippocampus in 1973 by Bliss & Lømo, which provided a

molecular mechanism for neuroplasticity which obeys Hebbian principles [36]. LTP was first described as the long-lasting increase in synaptic efficacy after tetanic stimulation of the presynaptic neuron. Long term depression (LTD) is the corrollary of LTP with reduction of synaptic efficacy after lower frequency repetitive stimulation.

The previous forms of neuroplasticity suggest a change in the properties of synapses between neurons. However, researchers in the past decade have provided evidence for the unmasking of *silent synapses* [37] and new synapse formation [38] associated with LTP-induction, indicating structural neuroplasticity after neuronal stimulation. Dendritic spines and presynaptic terminals are extremely dynamic in animals, and changes have been shown to be associated with experience and associative learning in a number of brain regions. Another form of structural plasticity is neurogenesis. While it has been accepted for most of the 20th century that no new neurons are formed in the adult brain, this has been shown to be false. It is now widely accepted that neurogenesis does occur in the adult hippocampus and the olfactory bulb [39]. There is some limited evidence that neurogenesis also occurs in other brain regions, although how widespread this phenomenon is and whether it participates in learning and memory remains controversial [40]. The study of neuroplasticity in humans was initially limited to the study of cultured human neurons or slices from surgical excisions in patients with epilepsy. However the invetion of transcranial magnetic stimulation spurred newer different types of non-invasive stimulation which allowed the study of neuroplasticity in humans.

Learning refers to the process by which a relatively lasting change in potential behavior, and occurs as a result of practice or experience. Neurophysiologically it involves the constant adaptation of the central nervous system to incoming information in order to optimize behavioural outcome. This requires the dynamic, plastic reorganization of neural connectivity in cortical networks. Plasticity in the primary motor cortex (M1) (Figure 5) has been shown to be functionally important as it plays an important role in forming new or adapting existing motor skills, as exemplified by recent works in experimental animals including primates and humans [41]. Motor cortical representations can reorganize rapidly in response to different pathological forms of damage, and this capacity has drawn great attention and interest especially in restorative neurology [42, 43]. This is likely to be a phenomenon resulting from the interaction of multiple brain regions rather than isolated neuroplasticity occuring in synapses in only one brain region.

The study of how diseases of the nervous system affect motor learning and plasticity provides some clues to the structures and processes that support these phenomena. For instance, cerebrovascular insults to the primary motor cortex and corticospinal tract are asso-

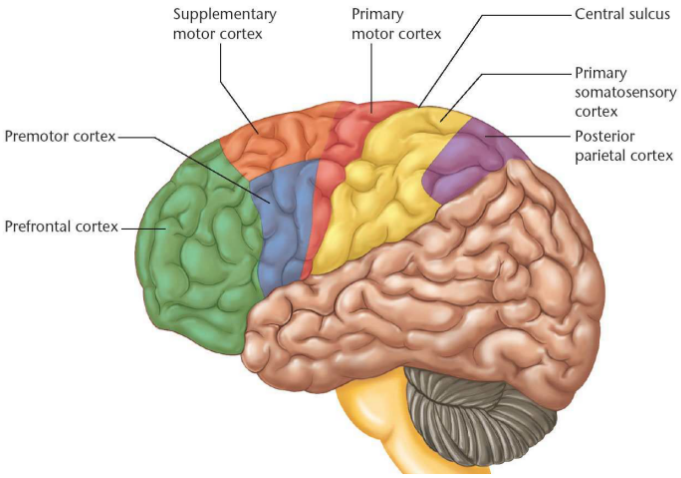


Figure 5: Regions of the brain

ciated with the reorganisation of brain regions occurring over many months and years [6, 44, 45]. This reorganisation process is believed to involve the resolution of oedema after the insult and neuroplastic changes [43], with the former predominanting in the acute and subacute phase and the latter predominating in the subacute and chronic phase. Functional imaging studies have shown that initially there is increased activation of undamaged secondary motor areas after the stroke, and focusing of these widespread activation patterns to fewer areas during functional recovery. Additionally, it has also been shown that certain activation patterns are associated with poorer outcome: activation of contralateral motor cortices are associated with poorer outcome. It has been proposed that there is a hierarchy of functional architecture with the function of the damaged primary motor cortex being taken over by the ipsilesional premotor cortex preferentially, and then the contralesional premotor cortex [46, 47]. The cortical reorganisation after stroke is likely to require shaping to get results and it is widely held that physiotherapy and rehabilitation in stroke provide this by encouraging motor learning [48]. The best rehabilitation strategies, therefore, should enhance cortical plasticity.

2.4 EVENT-RELATED SYNCHRONIZATION AND EVENT-RELATED DESYNCHRONIZATION

2.4.1 *History*

When Hans Berger described the human EEG in the 1920s [24], a pivotal finding was the demonstration of prominent oscillations in the frequency range between 8 and 12 Hz, which he called alpha wave rhythm. He also described for the first time the so-called "alpha blockade", i.e., the suppression of the ongoing alpha activity when the sub-

ject opens his eyes. In 1970s Gert Pfurtscheller and colleagues introduced the term event-related desynchronization (ERD) for this kind of frequency specific changes of ongoing EEG activity. Later, an increase in power of certain frequency bands after the application of certain stimuli was discovered and named event-related synchronization (ERS) [49]. Based on these findings, induced changes of oscillations have been reported for diverse physiological manipulations and processing of sensory information.

2.4.2 *Alpha band*

Task-induced frequency specific changes in the alpha band ($7-13\,\mathrm{Hz}$) are recorded in a variety of motor and cognitive tasks and are typically observed over frontal, parietal and motor areas. The traditional view is that the alpha rhythm is associated with "cortical idling" and its decrease in amplitude indicates cortical activation [50]. However, recent studies have shown that alpha ERD/ERS also depends on the level of consciousness [51], task performance [52] and IQ score [53]. A desynchronization localized to the auditory cortex following auditory stimuli was reported in MEG recordings [54]. Moreover, the alpha band rhythms demonstrate a relatively widespread desynchronization in perceptual, judgement and memory tasks [55]. In motor tasks, alpha ERD is recorded over sensorimotor areas before and during self-paced or externally triggered movements and motor imagery. It starts around 2 s prior to movement onset, being initially stronger over contralateral rolandic areas and it gradually becomes symmetric just before movement execution [56]. Interestingly, the time course and the topography of alpha ERD does not depend on the duration or the type of movement, a property which indicates a rather unspecific preparatory state of sensorimotor areas. In motor tasks, the amplitude decrease of alpha oscillations is often followed by a short lasting increase, which according to the traditional view of "cortical idling", denotes a deactivated cortical network. Contrary to this view, recent studies have provided strong evidence that the enhancement of alpha oscillation results from a top-down mechanism, which is related to active inhibition and timing of cortical processing [57]. In this line of argument, the gradual build-up of alpha ERD reflects the cessation of inhibition in certain task-relevant cortical areas in order for active information processing to take place.

2.4.3 *Beta band*

Similarly to the alpha rhythm, the amplitude of beta-band ($13-30\,\mathrm{Hz}$) is modulated during motor tasks. A decrease in amplitude (i.e. ERD) is normally observed during self-generated [58] or externally paced [59] movements and movement imagery [60]. Same as the alpha ERD,

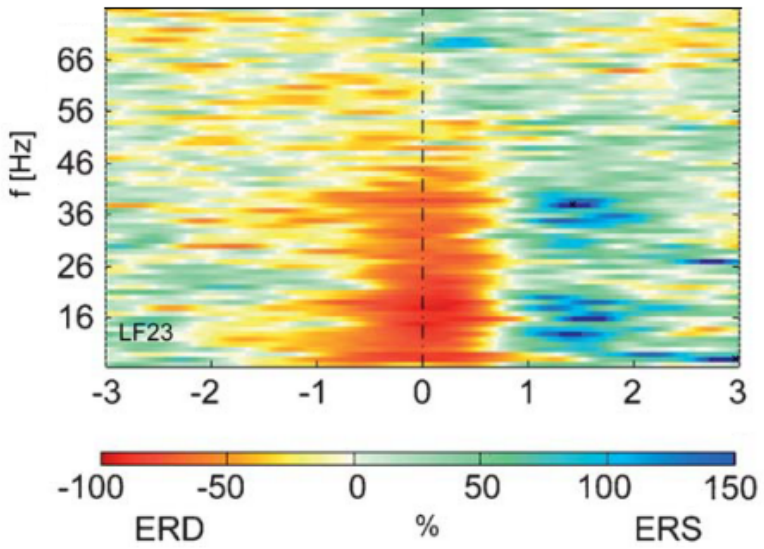


Figure 6: Event-related synchronization and desynchronization - spectral representation

beta ERD starts around 1.5 s prior to the movement in motor areas contralaterally to the responding hand and it gradually becomes bilateral during movement execution. Beta ERD is typically followed by an increase (synchronization), often termed post-movement beta ERS or beta rebound. It usually peaks around 1 s after movement onset and, unlike beta ERD, it depends on movement parameters [58] and the type of the effector [61]. Regarding the functional significance of beta oscillations, it has been proposed that they play an antikinetic role [62] and that they reflect the preservation of the current motor state [63]. However, the interpretation of beta ERS is still debated. Initially it was believed that it simply reflects a deactivated motor network ("cortical idling") [64]. In contrast, other studies suggest that beta ERS is an active inhibitory process which requires sensory feedback [65] and signifies the re-establishment of the previous postural state. It is possible though that these views are complementary rather than contradictory.

2.4.4 *Analysis*

It is important to note that, unlike phase-locked event-related potential (ERP)s, these types of changes are time-locked to the event. That is why ERD cannot be extracted by a simple linear method, such as averaging, but may be detected by frequency analysis. This means that ERD and ERS represent frequency specific changes of the ongoing EEG activity and may consist, of either decreases or of increases of power in given frequency bands. This happens due to a decrease or increase in synchrony of the underlying neuronal populations, re-

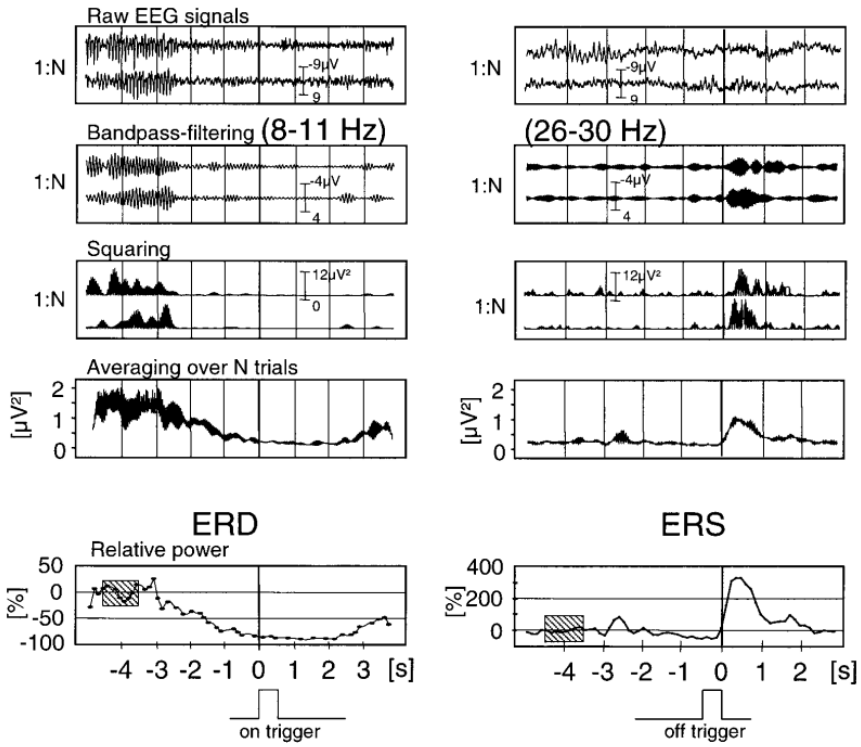


Figure 7: Principle of ERD (left panel) and ERS (right panel) processing. A decrease of band power indicates ERD and an increase of band power ERS. Note the different triggering with ERD and ERS processing[50]

spectively. Classically, induced ERD and ERS are quantified by the following procedure: the most reactive frequency bands are chosen, and then signals, band-pass filtered within those bands, are squared before averaging. Motor-related ERD is most prominent over the contralateral sensory-motor (SM) areas during motor preparation and extends bilaterally with the onset of movement [66]. The ERD and ERS must be observed in relation to the baseline activity measured some seconds before the event. It is now well established that even just the imagination of movement produces an event-related desynchronization (ERD) over the sensorimotor areas. The main difference between the imagination and execution of movement is that in the former case execution would be blocked at some cortico-spinal level [67].

2.5 EVENT-RELATED POTENTIALS

An ERP is any measured brain response that is directly the result of a thought or perception. More formally, it is any stereotyped electrophysiological response to an internal or external stimulus. Mental operations such as those involved in perception, selective attention, lan-

guage processing and memory, proceed over time ranges in the order of tens of milliseconds. As the EEG reflects thousands of simultaneously ongoing brain processes, the brain response to a single stimulus or event of interest is not usually visible in the EEG recording of a single trial; to see the brain response to the stimulus, the experimenter must conduct many trials and average the results together, causing random brain activity to be averaged out and the relevant ERP to remain. The assumption is that the event-related activity, or signal of interest, has a more or less fixed time delay to the stimulus, while the spontaneous background EEG fluctuations is random relative to the time when the stimulus occurred. Averaging across the time-locked epochs highlights the underlying ERP by averaging out the random background EEG activity (similar to additive white noise), thus improving the signal-to-noise ratio. These electrical signals reflect only the activity which is consistently associated with the stimulus processing in a time-locked manner. The ERP thus reflects, with high temporal resolution, the patterns of neuronal activity evoked by a stimulus.

Though some ERP components are referred to with acronyms (e.g., contingent negative variation - CNV, error-related negativity - ERN, early left anterior negativity - ELAN, closure positive shift - CPS), most components are referred to by a letter indicating polarity, followed by a number indicating either the latency in milliseconds or the component's ordinal position in the waveform. Thus, for instance, a negative-going peak that is the first substantial peak in the waveform and often occurs about 100 ms after a stimulus is presented is often called the N100 (indicating its latency) or N1 (indicating that it is the first peak and is negative); it is often followed by a positive peak usually called the P200 or P2. The stated latencies for ERP components are often quite variable; for example, the P300 component may exhibit a peak anywhere between 250 ms − 700 ms.

Physicians and neurologists will sometimes use a flashing visual checkerboard stimulus to test for any damage or trauma in the visual system. In a healthy person, this stimulus will elicit a strong response over the primary visual cortex located in the occipital lobe in the back of the brain. Experimental psychologists and neuroscientists have discovered many different stimuli that elicit reliable ERPs from participants. The timing of these responses is thought to provide a measure of the timing of the brain's communication or time of information processing [68].

2.5.1 *Movement-related Cortical Potentials*

Using the averaging technique, [Kornhuber and Deecke 69] observed brain potentials related to the initiation of voluntary hand movements in the scalp EEG. They recorded EEG and electromyogram (EMG) si-

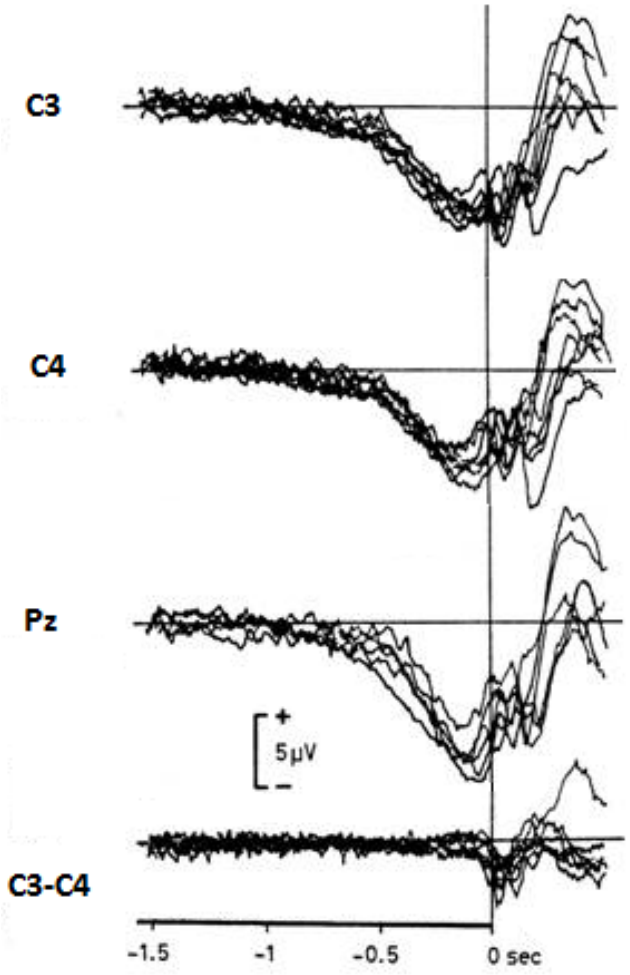


Figure 8: Bereitschaftspotential

multaneously while the subjects were repeating movements at a self-paced rate, and stored all the data on magnetic tape. Then they made an off-line averaging of the EEG segment prior to the EMG onset by playing the tape backward. By using this chronologically reversed averaging technique, they successfully identified two components, one each before and after the EMG onset. Those were the *Bereitschaftspotential* (BP) or readiness potential (RP), and *Reafferente Potentiale*. Later they found two more components just before the movement onset: pre-motion positivity (PMP) and motor potential (MP) [70].

Figure 8 shows that BP starts about 2 s before the movement onset. It is maximal at the midline centro-parietal area, and symmetrically and widely distributed over the scalp regardless of the site of movement. The origin of it has been attributed to bilateral activation of premotor areas and/or the supplementary motor area (Figure 5). The onset of BP with respect to the movement onset significantly differs among different conditions of movement and among subjects. For ex-

ample, in the experimental setting in which the subject is requested to repeat the same movement at a self-paced rate of once every 5 s or longer, the BP commonly starts much earlier as compared to the movement executed in more natural conditions, because, in such experimental conditions, the subject has a much longer time to prepare for the movement. BP suddenly increases its gradient about 400 ms before the movement onset. Based on the clearly different scalp distribution of the late steeper slope from that of the early slow shift it was designated as late BP or Negative Slope (NS'). Late BP was distinguished from the early BP based on abrupt increase of the gradient at the central electrode corresponding to the movement for each individual subject, instead of arbitrarily setting the time such as 500 ms before the movement onset for the distinction of the two slopes. It is maximal over the contralateral central area (approximately C1 or C2 of the International 10–20 System) for the hand movement and at the midline (approximately Cz) for the foot movement [71] and is believed to originate from M1 activation. For the study of BP in individual subjects, therefore, it is important to record EEG from multiple electrodes.

Artifacts due to head-, eye-, lid-, mouth-movements and respiration have to be eliminated before averaging because such artifacts may be of a magnitude which makes it difficult to render them negligible even after hundreds of sweeps. That is why, although BP reflects cortical activity, it is not practical as a BCI input signal because it is discernable only after averaging many trials, unlike ERD.

2.6 FUNCTIONAL ELECTRICAL STIMULATION

Information in nerve cells is coded and transmitted as series of electrical impulses called action potential (AP)s, which represent brief changes in cell electric potential of about 80 mV. APs can be artificially generated by inducing electric charge into the nerve cell or nerve axon. The intensity of the signal transmitted is directly proportional to the frequency of APs that occur in the axon per unit of time. When APs are generated using electrical stimulation and are used to produce a body function, it is referred to as FES. During FES, for every AP that propagates towards the end of the axon that is innervating a muscle (orthodromic propagation) one AP will propagate backwards towards the cell body of the motoneuron (antidromic propagation). FES is typically concerned with orthodromic propagation as they generate muscle contractions in order to produce the desirable body function [72]. Figure 9 illustrates the direct stimulation of a motoneuron which then innervates the specific muscle. In the case when the APs are generated by the central nervous system (instead of FES), the cell body receives AP driven inputs from dendrites, it summates the excitatory and inhibitory APs, processes them and

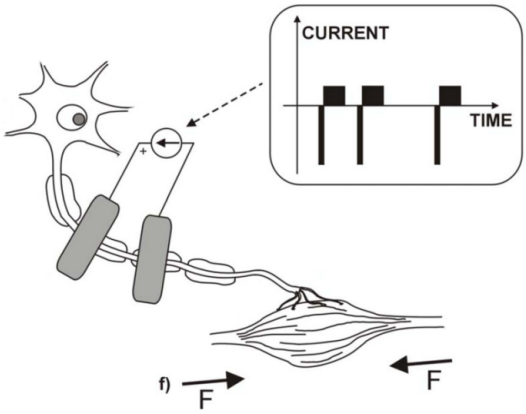


Figure 9: Direct motor neuron stimulation

decides whether or not to generate an output AP. Following stroke or spinal cord injury the motoneurons do not receive appropriate input from the central nervous system therefore inhibiting muscle function. FES replaces this functionality by artificially generating required APs to elicit a desired muscle/limb function.

Since the early 1960s electrical stimulation has been used to artificially generate body functions such as walking, grasping, and hearing. Today FES is used for restoring voluntary motor function following short-term use of FES as a therapy, as well. Systems using FES can be external or internal. Surface FES systems apply self-adhesive or non-adhesive electrodes placed on the skin surface just above the muscle that needs to be stimulated. Implanted FES systems are intended for more permanent applications, i.e., orthoses that are used to substitute a function at all times. Most, if not all components of the implanted FES systems are internal to the body where the stimulation electrodes are always implanted. Common examples include cochlear implants and bladder management systems. Percutaneous FES systems are those which have electrodes implanted in the body while the rest of the system is external to the body.

Surface FES systems are non-invasive, electrodes are easy to apply, are generally less expensive and safer. However, since the stimulus signal must travel through skin, considerably higher-intensity signals are required due to the higher impedance of skin and dispersion of the signal when compared to subcutaneous or implanted stimulation electrodes. Another limitation of surface stimulation is the targeting of deeper nerves. One of the most common uses of external FES systems is therapy of patients after stroke or spinal cord injury with the intent to improve their motor abilities [73, 3]. A combination of intensive voluntary activation of proximal muscles and patterned multichannel electrical stimulation of distal muscles providing grasp and release functions in the paretic hand is called functional electrical therapy (FET) [74]. These patients are often unable to functionally use one arm and/or hand. Most of clinical studies agree that active re-

habilitation is better than passive and that early treatment leads to better recovery. The essential difference between FET and other electrical stimulation methods is that while electrical stimulation assists the opening, closing, and releasing functions, in parallel, a hemiplegic subject can concentrate on manipulation, that is, on shoulder and elbow movements. This added ability to grasp and release objects motivates a hemiplegic subject to exercise in a functional manner, i.e. to practice typical movements that were part of his or her normal daily activities before the cerebrovascular accident.

3 METHODS

3.1 INSTRUMENTATION

The biorecording technology used was EEG over the motor cortex. The EEG was recorded with an Electro-Cap of an elastic spandex-type fabric with recessed, pure tin electrodes attached to the fabric in the custom method of electrode placement. The electrodes chosen for this experiment were, as follows: C3, Cz, C4, PC3, PCz, PC4, PC1, PC2, PC5, PC6, FC2, FC6, FC5, FC1 (Figure 10). EOG was recorded using the same cap and the electrode Fp1. Bipolar EMG was recorded on the extensor carpi radialis longus muscle [75] with Ambu Neuroline 720 electrodes. Both EEG and EMG electrodes were connected to two 8-channel PsychLab EEG8 amplifiers (set up with a 24 dB high pass Bessel filter with a cut-off frequency set to 0.01 Hz and a low pass filter with a cut-off frequency set to 100 Hz and a T notch filter set to 50 Hz). These amplifiers were connected to a National Instruments A/D converter connected to a standard USB slot of a laptop computer. Data were digitized with a sampling rate of 500 Hz. Data were referenced to the linked right and left ear lobes with a ground slightly above nasion. This configuration was selected based on classification results from other studies which indicated that the most important electrode locations for differentiation between different motor imagery tasks are the electrode positions C3, Cz, and C4.

Neuromuscular electrical stimulation was used to effect hand extension movements in the FES and FESVOL trials. The stimuli were delivered by a commercial FES stimulator. Stimuli were delivered through self-adhesive round surface stimulation electrodes. A cathode was positioned over the motor point of the extensors (extensor carpi radialis longus) and an anode was placed on the forearm near the wrist. The stimulation pattern was triggered by a push-button switch. Each button press initiated a stimulation pattern that produced a 1 s finger extension. The amplitude of stimulation was determined individually for each participant before the experiment. The stimulation intensity was set to a level that produced clear hand extension movement but not high enough to produce pain. While the amplitude of stimulation varied from one subject to another, the range of stimulation current intensity ranged from $10 - 15$ mA.

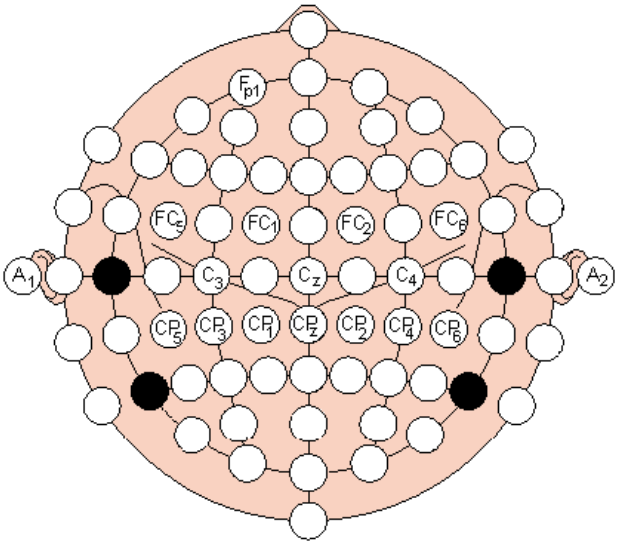


Figure 10: Electrode locations

3.2 SUBJECTS

Five healthy volunteers (2 female, 3 male; mean age: 24.4 ± 1.14 years; range $23 - 26$ years) with no history of neuromuscular disorder participated in the experiment. All of the subjects are right-handed. There was no selection criteria or screening of subjects. They participated voluntary without receiving any fee for their participation. All subjects were students at University of Belgrade and had no notable experience with FES or EEG recording.

3.3 EXPERIMENTAL PROTOCOL

The subjects sat in a chair with their arms resting comfortably on the table in front of them. The complete experiment lasted about 90 minutes per subject including electrode placement, subject preparation and recording. They performed three different motor tasks (VOL, FES and FESVOL), one per session. The movement performed was the same for every session; subjects were resting with hands laying flat on the table and then extended their right hand in wrist and returned back to the starting position, as seen on Figure 11.

- During the FES session, the movement was completely initiated by the electrical stimulation of extensor carpi radialis longus muscle. A person sitting behind the subject was operating the stimulator so that the subject was unaware of the timing of the stimulation. Duration of the stimulus was 1 s, and the interstimulus interval was random (mean 10 s), so the subjects couldn't get used to the pace and as a precaution to avoid any slow-changing measurement artifacts.

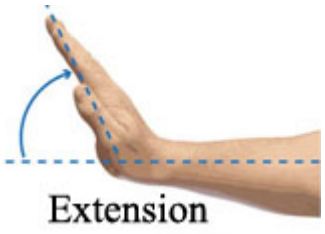


Figure 11: Movement performed

- During the FESVOL session, the movement was initiated by the stimulator (same as FES), but performed in part by the subjects themselves. That is, the start of the stimulation acted as a trigger for the subjects to perform the hand extension (and subsequent flexion) themselves.
- The VOL session consisted of self-paced voluntary extensions of the hand. Subjects were instructed to perform the movement as similar as possible to the movement from the first and second sessions. The interval between the movements remained at around 10 s.

Each session lasted about 20 minutes (~ 50 trials) separated by a 5-minute break. Intentionally, the experiments were conducted in a room with heavy traffic where other students work on their projects with the intent of replicating a real life situation.

3.4 DATA PROCESSING

Recorded EEG data were analyzed in the time and time-frequency domains to find useful features that could discriminate between the three types of movement performed. Offline analyses from 14 EEG channels were performed in MATLAB to find relevant subject-dependent activation patterns using different signal processing tools.

3.4.1 *EMG and EOG Processing*

EMG sequence processing consisted of baseline removal and signal squaring to enable easire detection of movements. Two digital filters were used, a band-pass filter with cut-off frequencies set to $5 - 100$ Hz, and a notch filter with a 50 Hz center frequency to eliminate the noise originating from the power lines. This signal was used to the detect the onset of movement (or start of stimulation) using a simple thresholding algorithm.

EOG signal was filtered using a band-pass filter to retain only the frequencies $0.05 - 30$ Hz. Each trial was then visually inspected and EEG trials which were contaminated by EOG were eleminated from

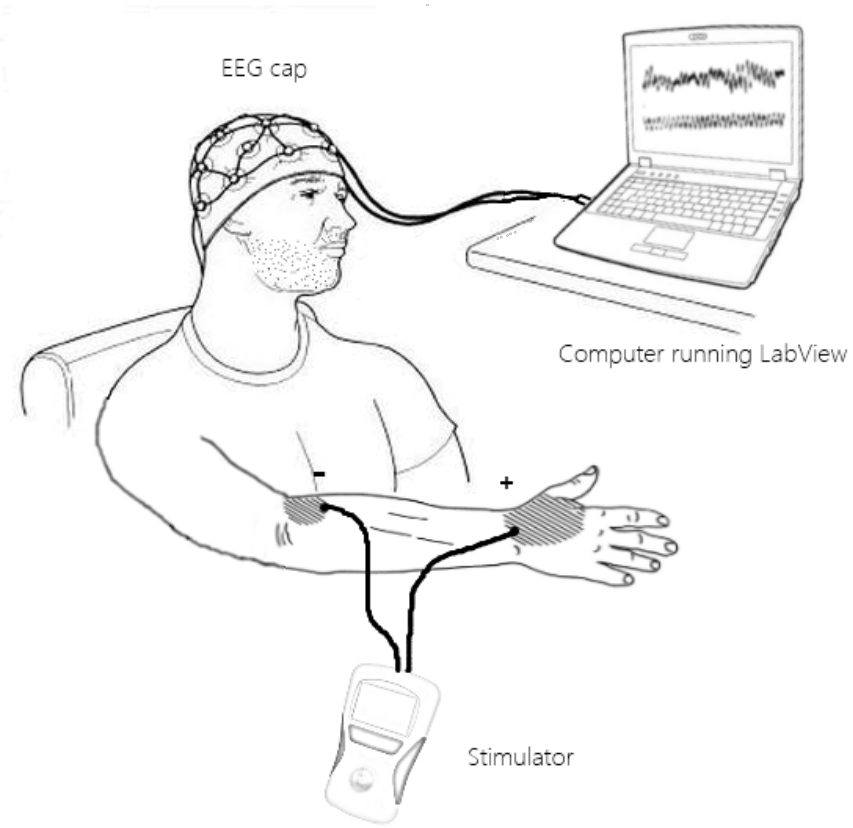


Figure 12: Setup for the experiment

further analysis. The number of artifact-free trials per session and subject ranged from 20 to 40.

3.4.2 *Time-frequency Analysis*

Time-frequency analysis is an effective method for the visualization of event-related changes in oscillatory brain activity [76]. Time-frequency maps are useful for the selection of frequency bands and electrode locations with the most significant band power increase or decrease during motor tasks. Considering that these bands differ from person to person it was natural to perform this step before any other. The data was analyzed using the MATLAB's built-in *spectrogram* function (hamming window size: 1000 samples, number of overlapping samples: 900). For each EEG channel, a time-frequency map was calculated for frequencies between 5 and 25 Hz and for a 10 s epoch (time-locked to the onset of movement, determined as the onset of EMG activity) which extended from 5 s prior to 5 s after movement onset. Then, spectrograms for each subject were averaged over all of the trials in a session.

3.4.3 *Temporal Analysis*

3.4.3.1 *ERD/ERS*

Time-frequency maps computed in the previous step were visually inspected to determine which frequency range expressed most prominent desynchronization. This was performed for each subject individually. The results, although different for each subject, were within the expected range (mean: $9 - 11.3$ Hz) . All EEG channels were filtered to retain only those frequency components, squared and filtered using a moving-average filter with the window length of 51 samples (102 ms). Then, each subject's EEG recordings were cut into 13 s epochs (5 s before and 8 s after movement onset) and averaged over all trials in a session. ERS was computed in the same way as ERD, only the original signals were filtered to retain frequencies $18 - 25$ Hz. Since they are defined as the proportional power decrease (ERD) or power increase (ERS) in a given frequency band in relation to a reference interval several seconds before the task was performed, I present and analyze this baseline power as well as the power during the movement.

3.4.3.2 *Movement-related Cortical Potentials*

In order to analyze the occurence of MRCPs, the EEG channels were first low-pass filtered with a cut-off frequency of 5 Hz and then cut up into 10 s in the same way as before (5 s before and after movement) except this time the mean baseline activity ($5 - 2$ s before movement

onset) was removed from each trial. These 10 s-epochs were then averaged over all of the trials.

4 RESULTS

4.1 TIME-FREQUENCY MAPS

Spectrogram maps for one of the subjects for all three experiments are presented here. A bipolar signal referenced to the electrode CP4 is shown. FES signal is presented on Figure 13, FESVOL on Figure 14, and VOL on Figure 15. The spectrograms of each subjects were visually inspected to determine the μ-band range of frequencies. These results are presented in Table 1. It is important to mention that the signal form the electrode PC5 was severely covered by noise and was therefore discarded from the analysis.

Table 1: Detected mu-rhythms from spectrograms

Subject	Age [yrs]	μ frequency range [Hz]
1	26	9.5 – 12
2	23	9 – 11
3	24	8 – 10
4	25	8.5 – 11.5
5	24	10 – 12

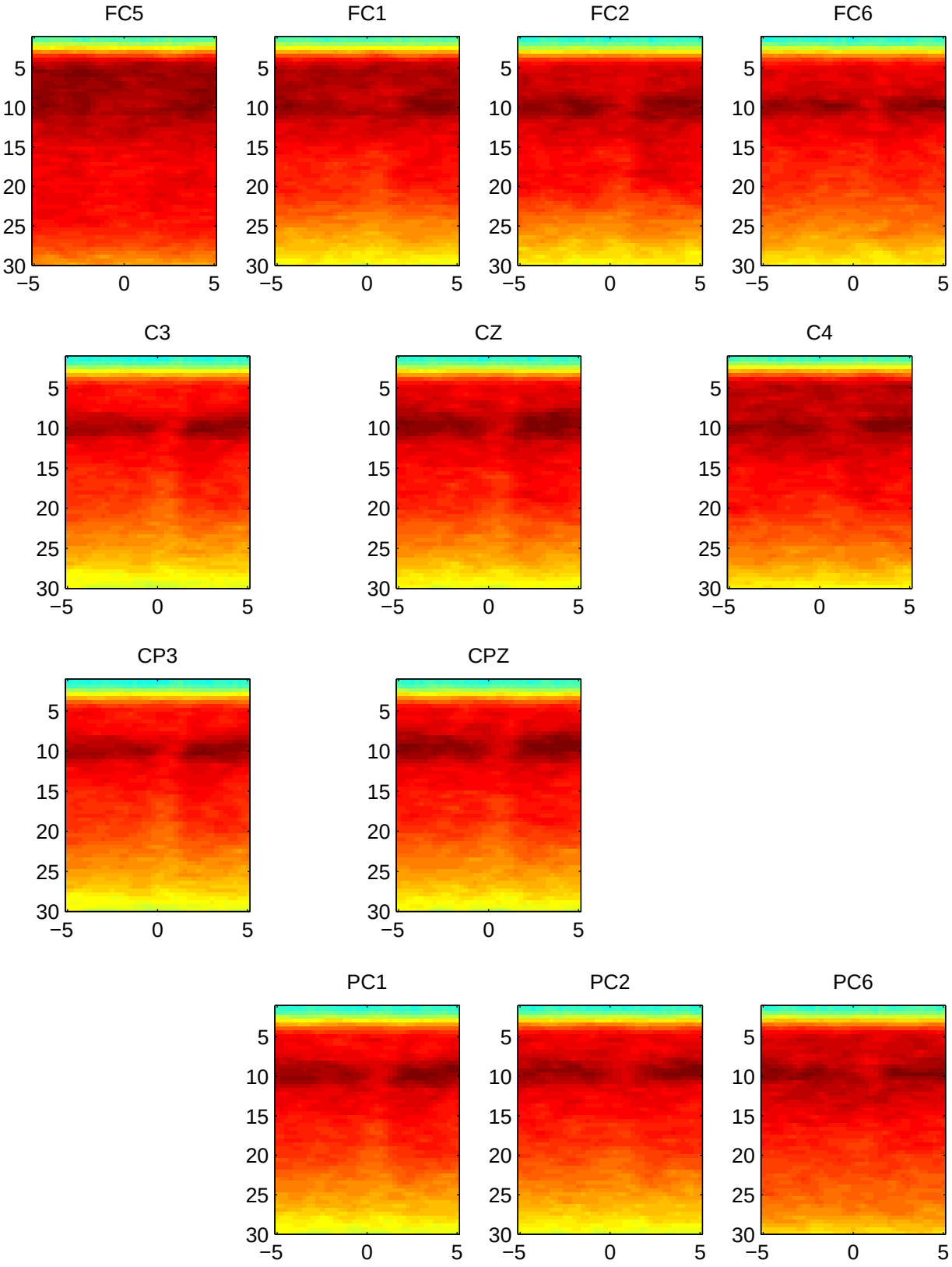


Figure 13: Bipolar spectrogram of subject 4 - FES; x-axes are in seconds, with 0 being the start of movement, y-axis are in Hz

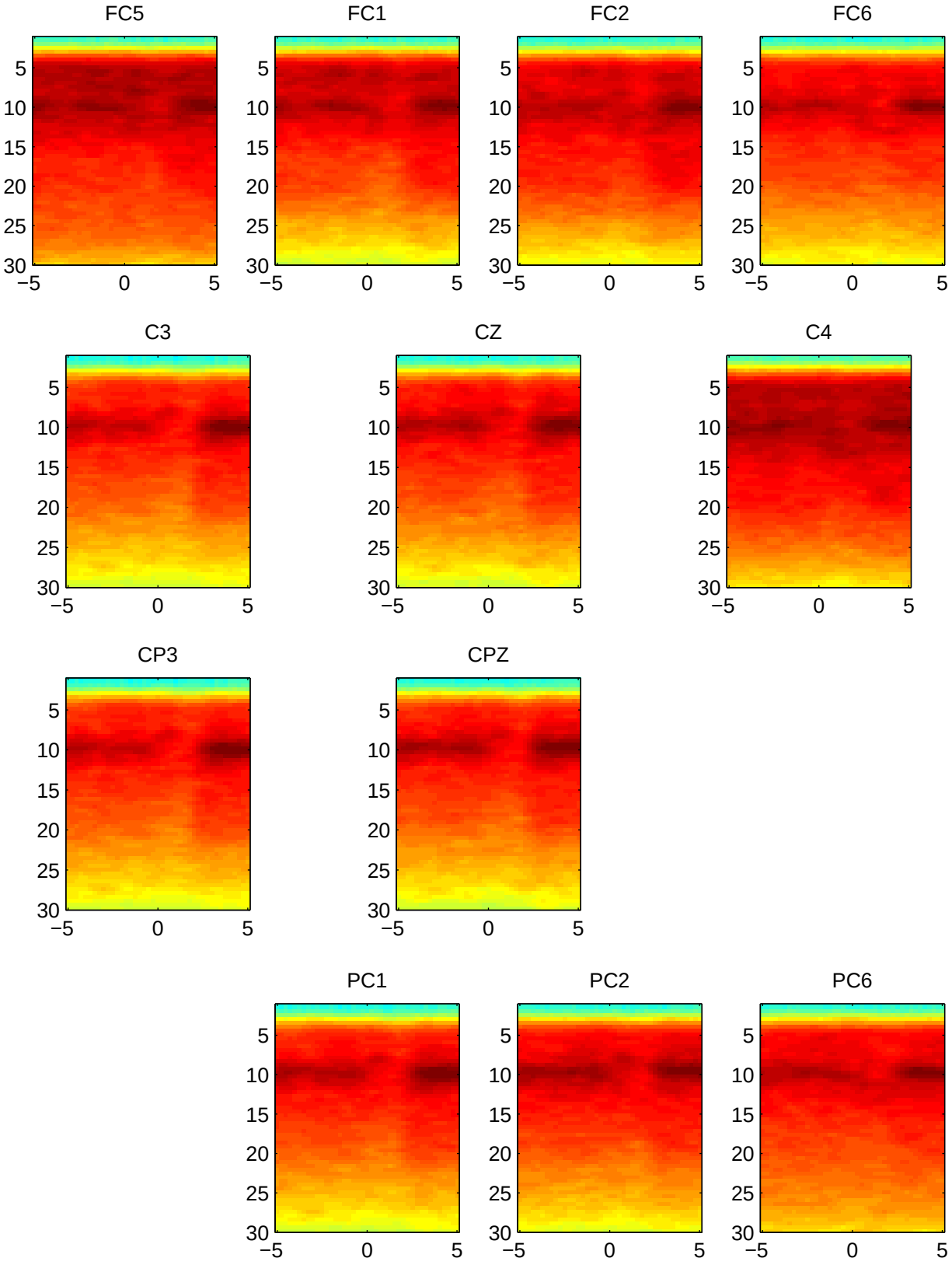


Figure 14: Bipolar spectrogram of subject 4 - FESVOL; x-axes are in seconds, with 0 being the start of movement, y-axis are in Hz

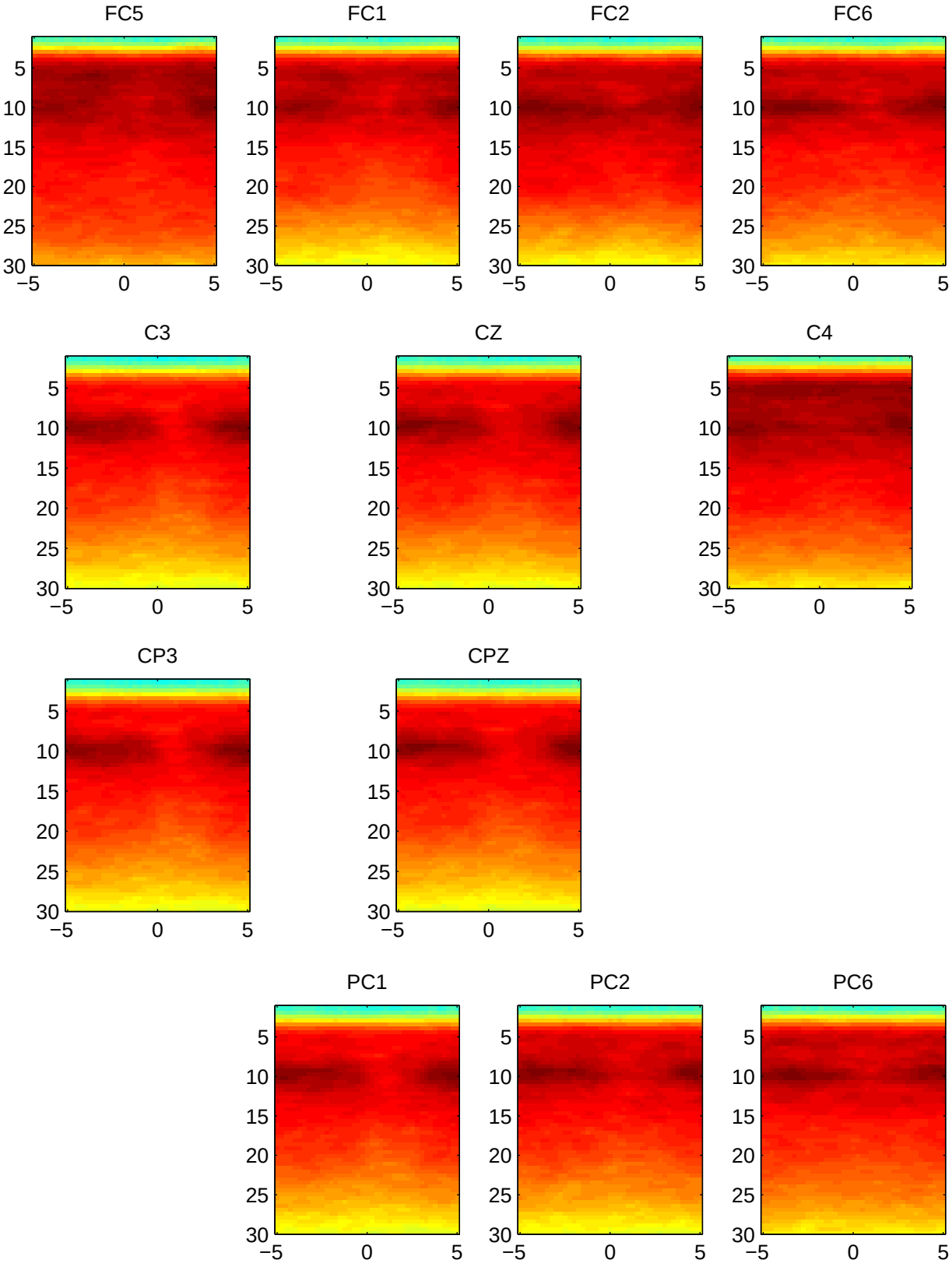


Figure 15: Bipolar spectrogram of subject 4 - VOL; x-axes are in seconds, with 0 being the start of movement, y-axis are in Hz

4.2 MOTOR-RELATED CORTICAL POTENTIALS

Figure 16 shows a timeseries of all three experiments on the same plot. The monopolar recording was referenced to the linked right and left ear lobes. The bipolar measurement shows no remarkable differences to the monopolar recording, and it is not depicted. First, the results are presented as timeseries of averaged trials for one subject. Four representative lectrodes were chosen (C_3, C_4, CP_3, CP_4) and mean and variance of the averaged signals of all trials over time were examined in different time periods before the onset of movement. The examined periods are the ones in which the greatest difference between experiments is supposed to be seen, and are: 2 s before movement onset till movement onset, 1s before movement onset till movement onset, and 1.5 s before movement onset till movement onset. These results are presented in the form of a bar graph in Figure 17.

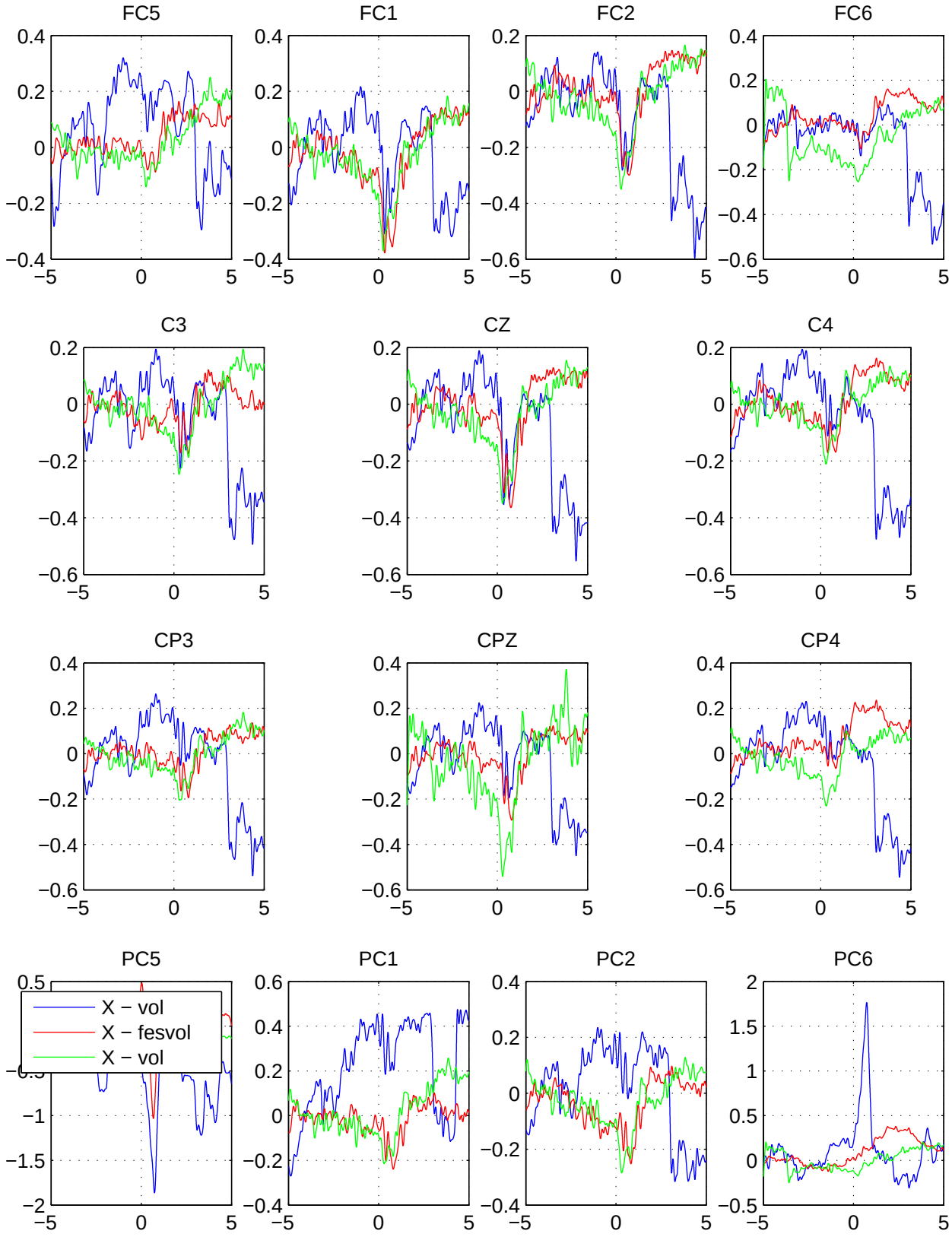


Figure 16: Monopolar MRCPs of subject 3; x-axes are in seconds, with 0 being the start of movement, y-axis are in mV

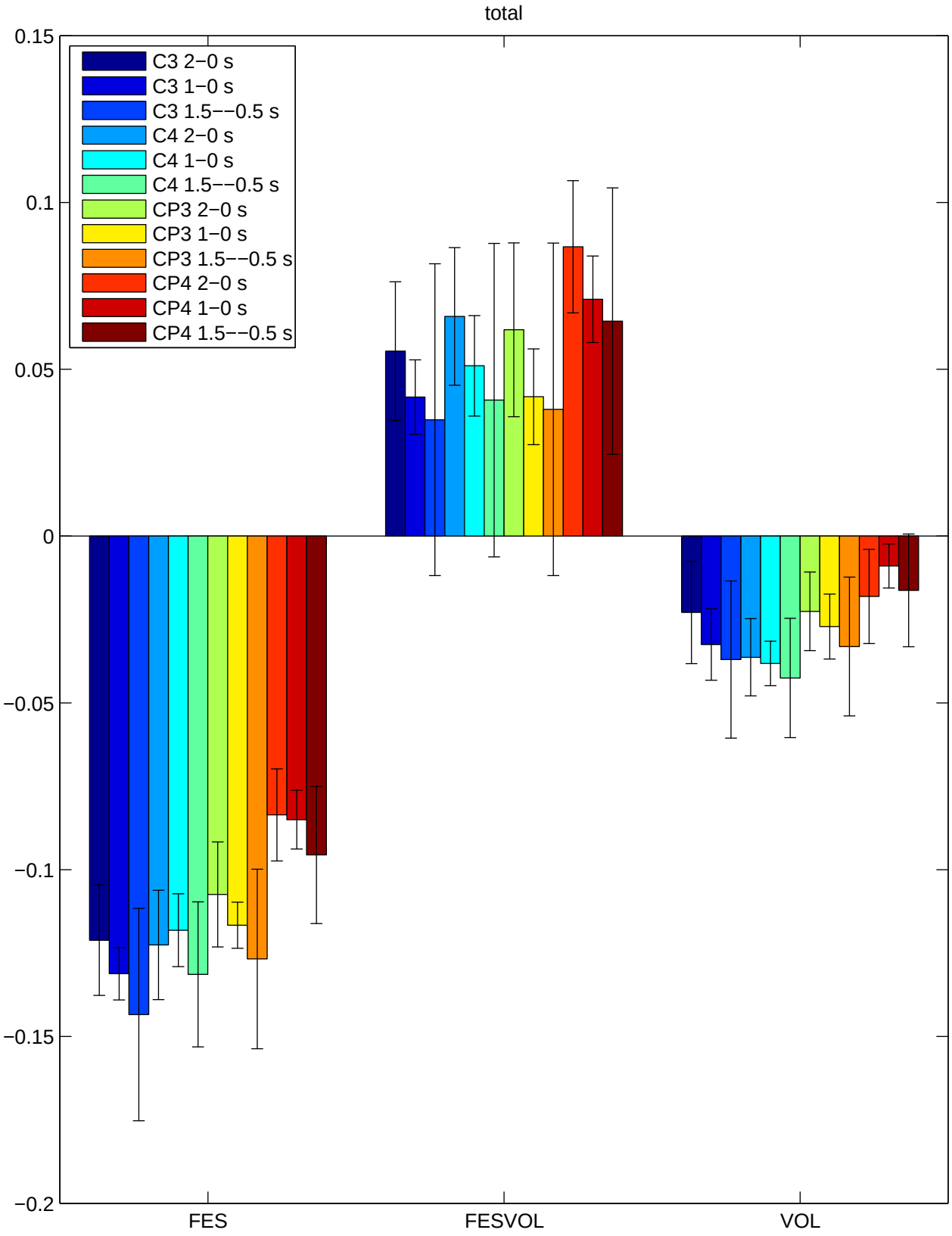


Figure 17: Monopolar MRCP features of all subjects; y-axis is in mV

4.3 α-RHYTHM EVENT-RELATED DESYNCHRONIZATION/SYNCHRONIZATION

A signal filtered to the alpha band and averaged over all of the trials of one subject is presented on Figure 18. A bipolar recording of the same subject, referenced to the CP4 electrode and computed in the same way as the monopolar recording can be seen on Figure 19. Again, the same four representative electrodes wre chosen (C3, C4, CP3, CP4) and their signals analyzed.

First, the baseline amplitude of the signal were considered in two periods of time: 2 s prior to movement onset till movement onset, and 0.5 s before movement onset till movement onset. These results can be seen on Figure 20 and Figure 21.

Amplitude of the signals during the movement was analyzed in the same way in the following ranges: from movement onset for 2 s, and 1 s post-movement onset for 1 s. These bar graphs are presented on Figure 22 and Figure 23.

Finally, post-movement ERS was analyzed and the results can be seen on Figure 24 and Figure 25. Time ranges used in the computation of the bar graphs are: 3 s after movement onset to 5 s after movement onset, 4 s after movement onset to 6 s after movement onset.

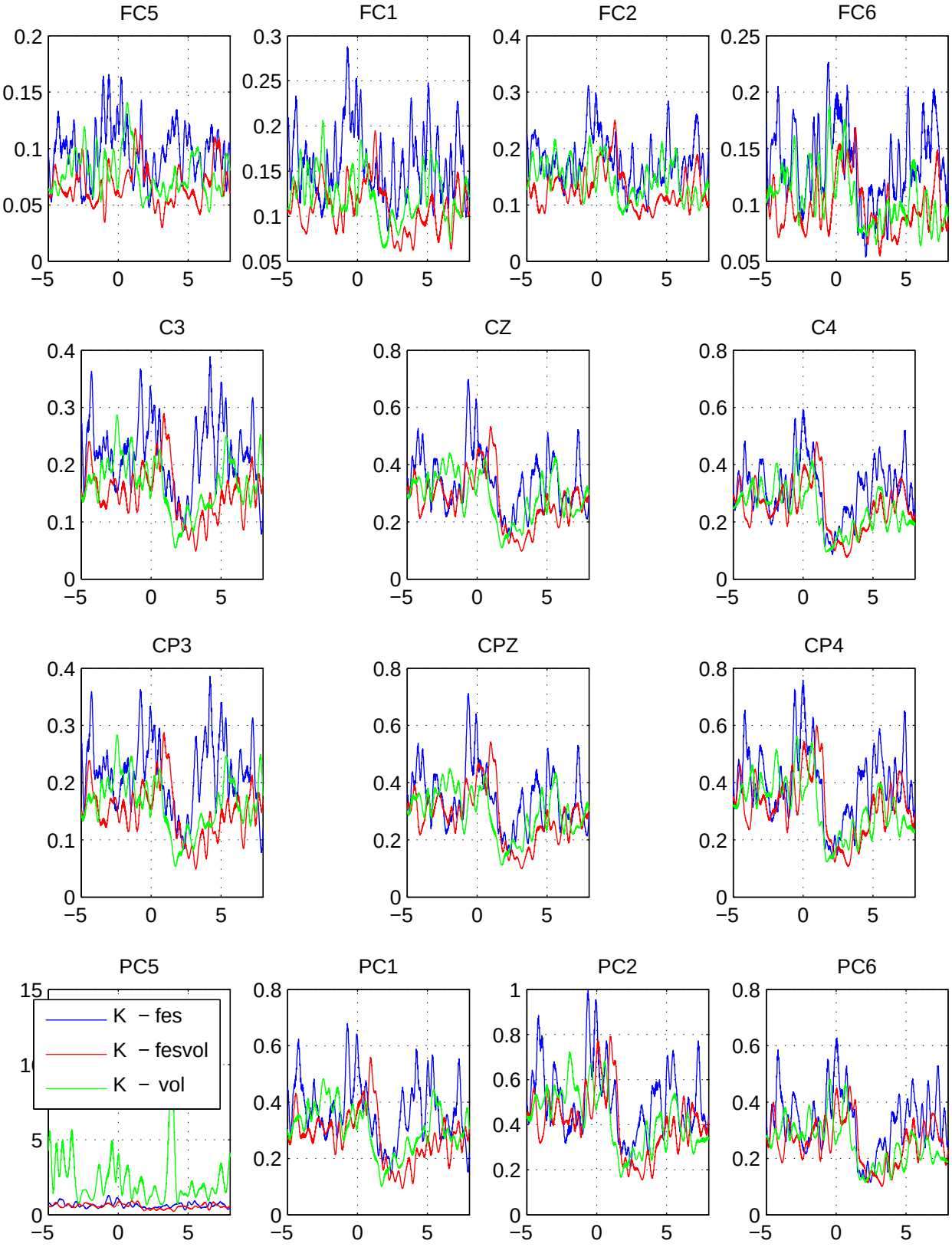


Figure 18: Monopolar alpha ERD/ERS of subject 2; x-axes are in seconds, with 0 being the start of movement, y-axis are in mV

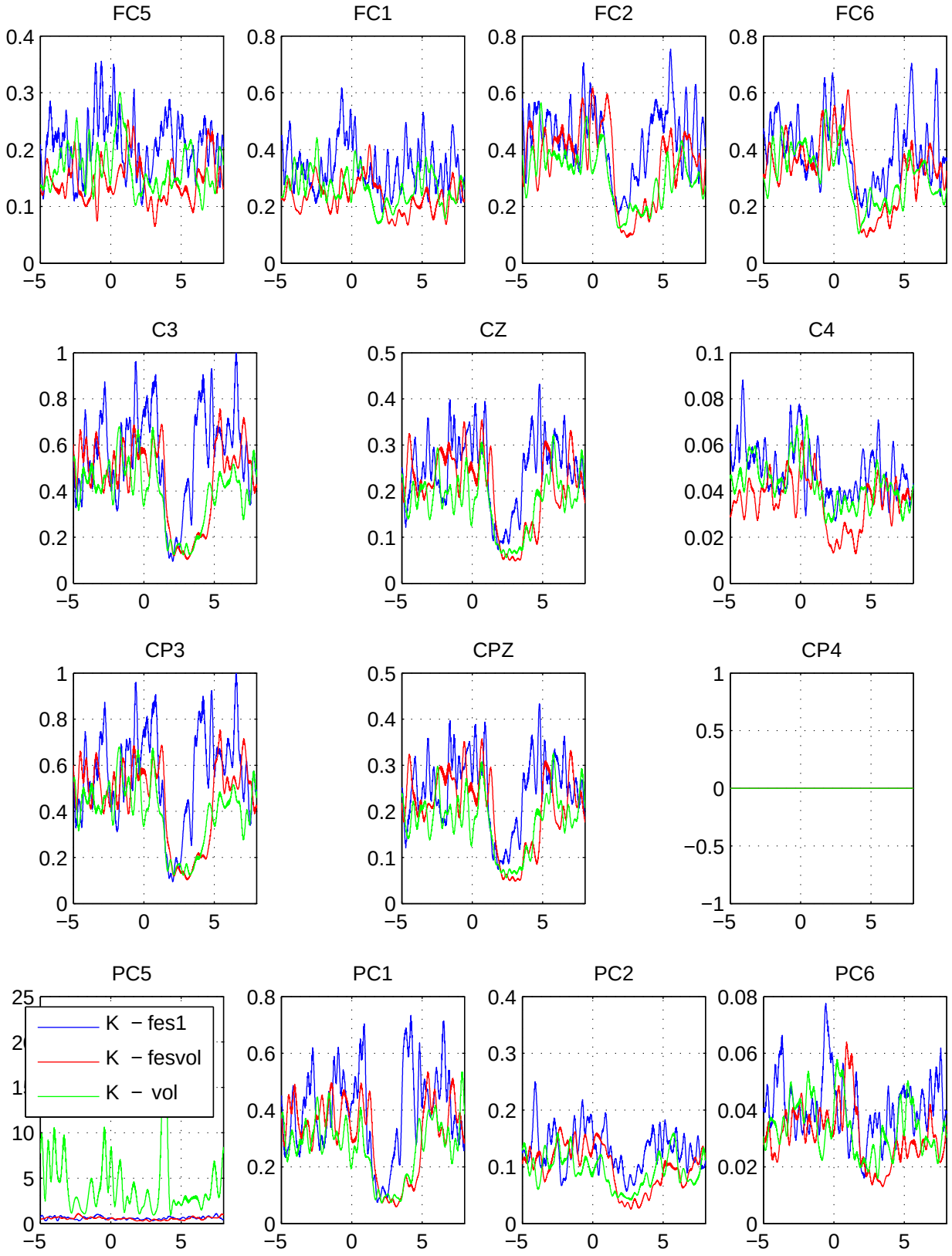


Figure 19: Bipolar alpha ERD/ERS of subject 2; x-axes are in seconds, with 0 being the start of movement, y-axis are in mV

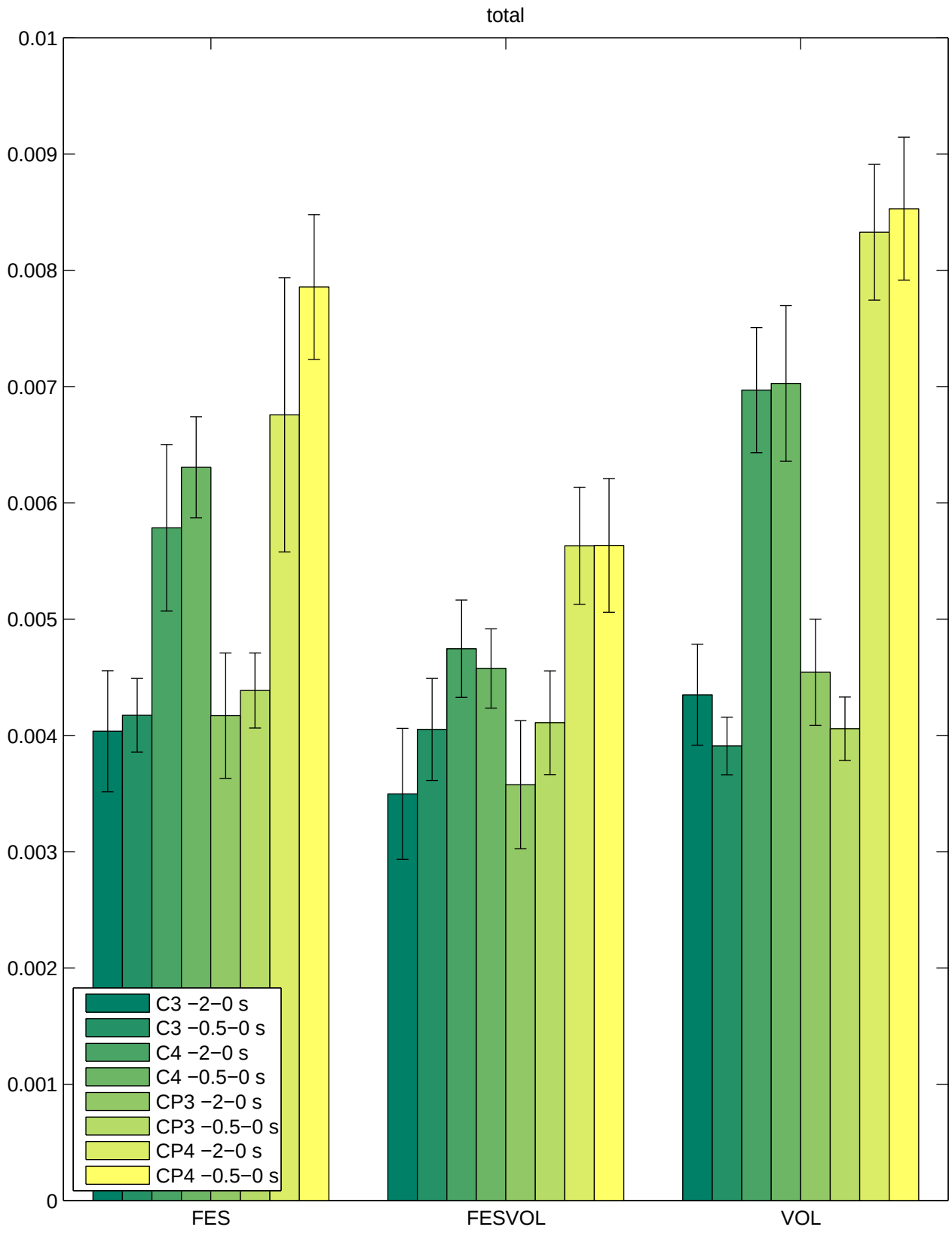


Figure 20: Monopolar baseline alpha power of all trials; y-axis is in mV

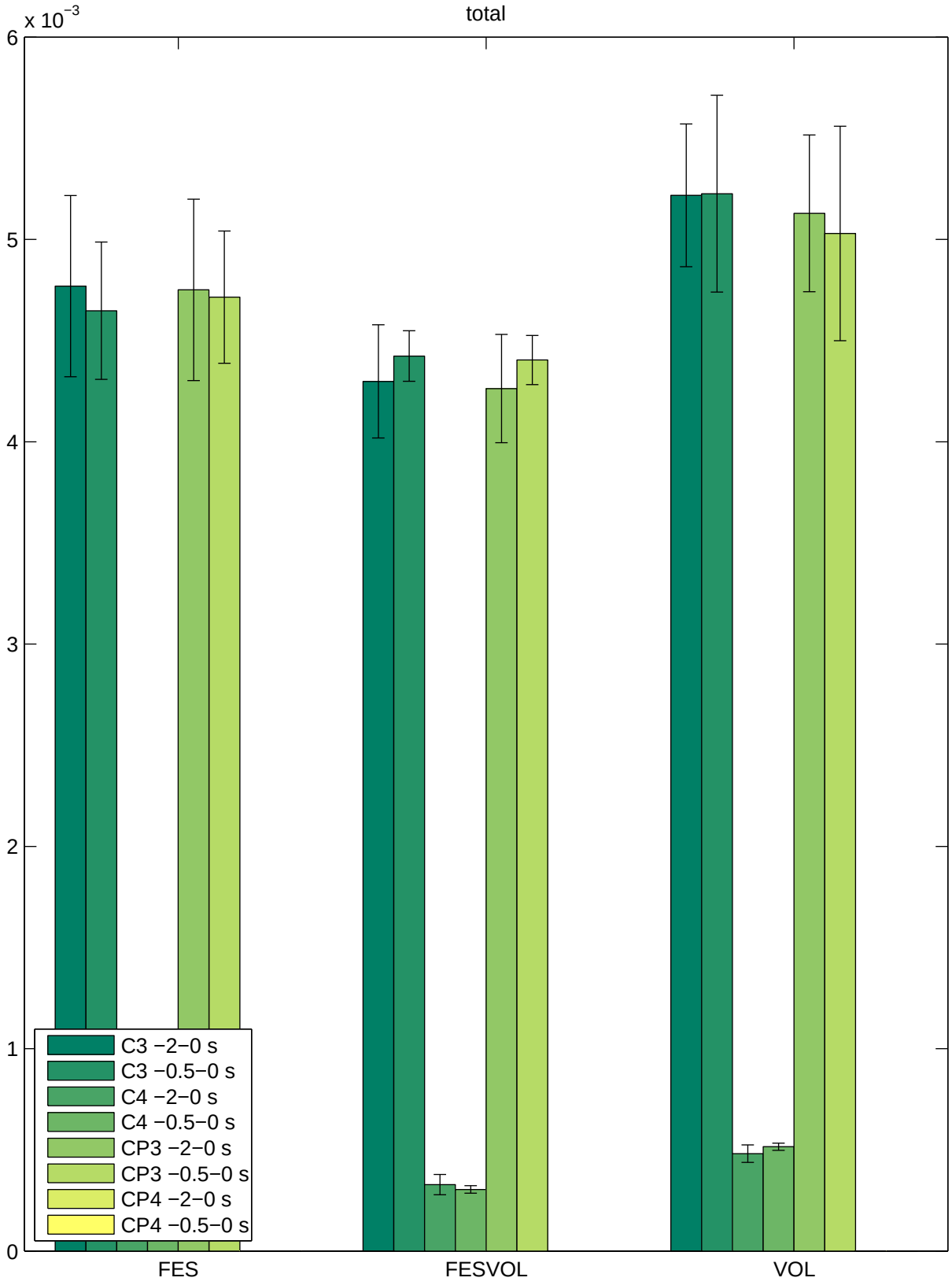


Figure 21: Bipolar baseline alpha power of all trials; y-axis is in mV

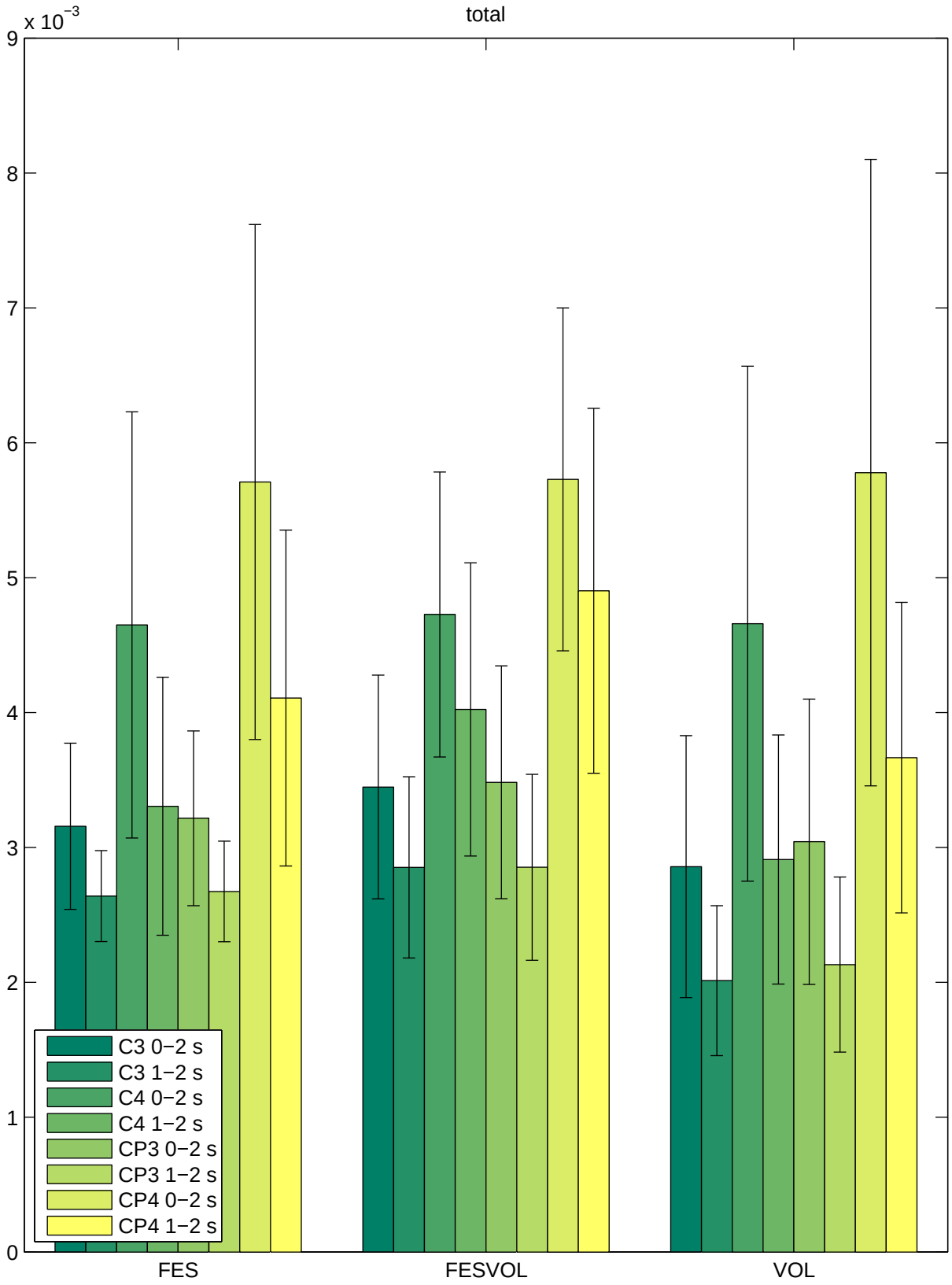


Figure 22: Monopolar alpha power after movement onset of all trials; y-axis is in mV

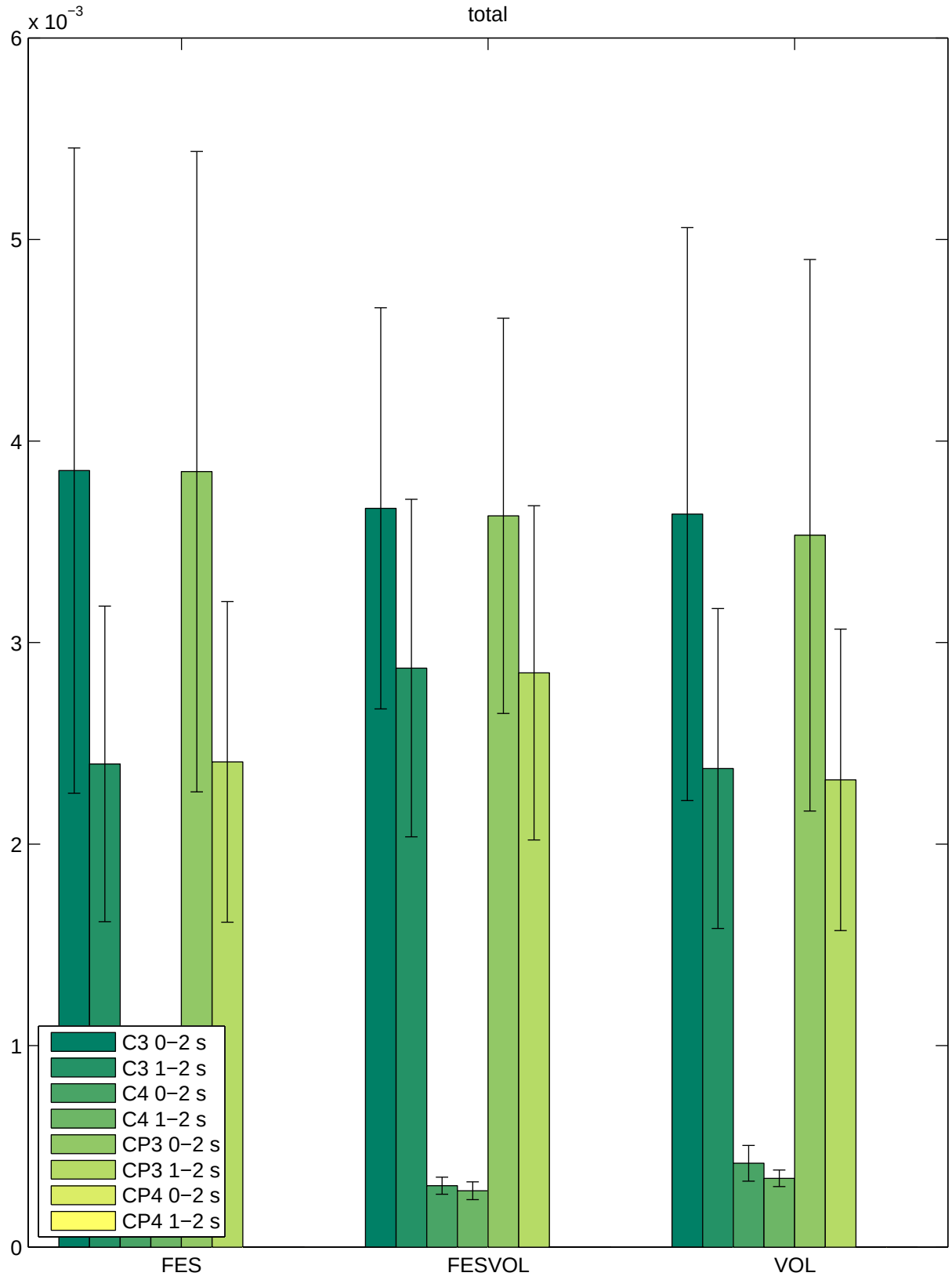


Figure 23: Bipolar alpha power after movement onset of all subjects; y-axis is in mV

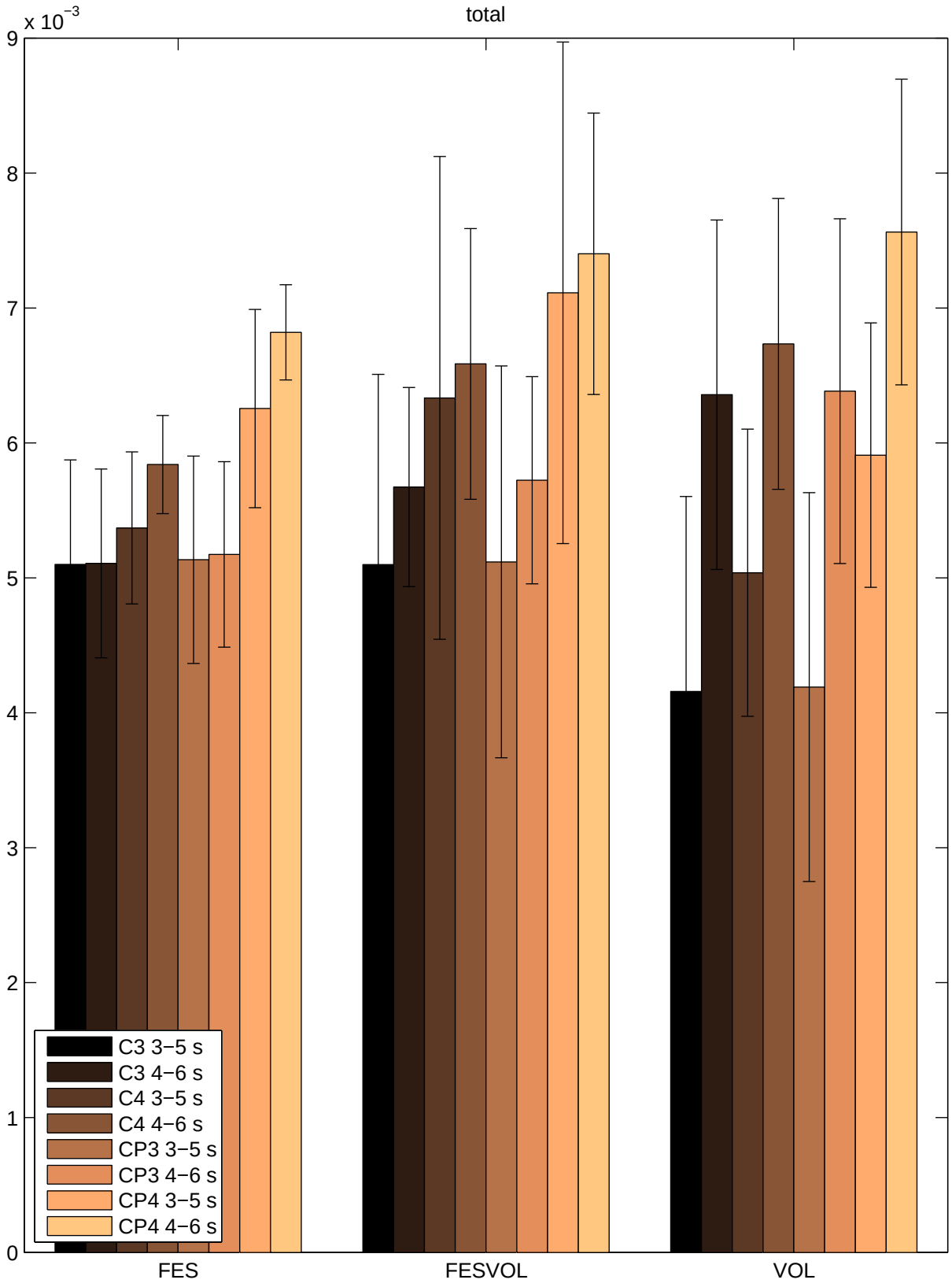


Figure 24: Monopolar alpha power after movement end of all trials; y-axis is in mV

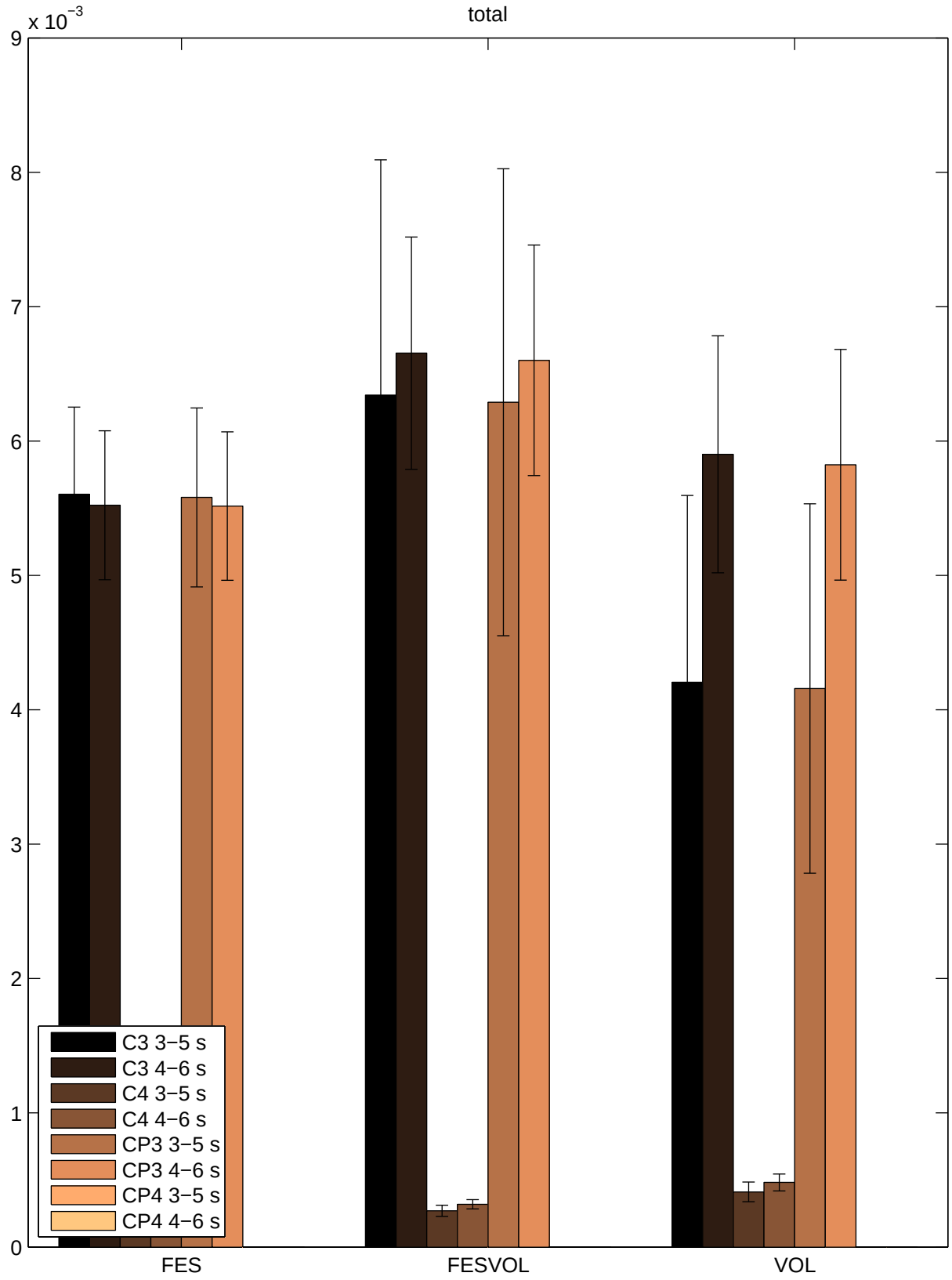


Figure 25: Bipolar alpha power after movement end of all subjects; y-axis is in mV

4.4 β-RHYTHM EVENT-RELATED SYNCHRONIZATION

A signal filtered to the beta band and averaged over all of the trials of one subject is presented on Figure 26. A bipolar recording of the same subject, referenced to the CP_4 electrode and computed in the same way as the monopolar recording can be seen on Figure 27. Again, the same four representative electrodes wre chosen (C_3, C_4, CP_3, CP_4) and their signals analyzed.

First, the baseline amplitude of the signal were considered in two periods of time: 2 s prior to movement onset till movement onset, and 0.5 s before movement onset till movement onset. These results can be seen on Figure 28 and Figure 29.

Finally, post-movement ERS was analyzed and the results can be seen on Figure 30 and Figure 31. Time ranges used in the computation of the bar graphs are: 3 s after movement onset to 5 s after movement end, 4 s movement end to 6 s after movement end.

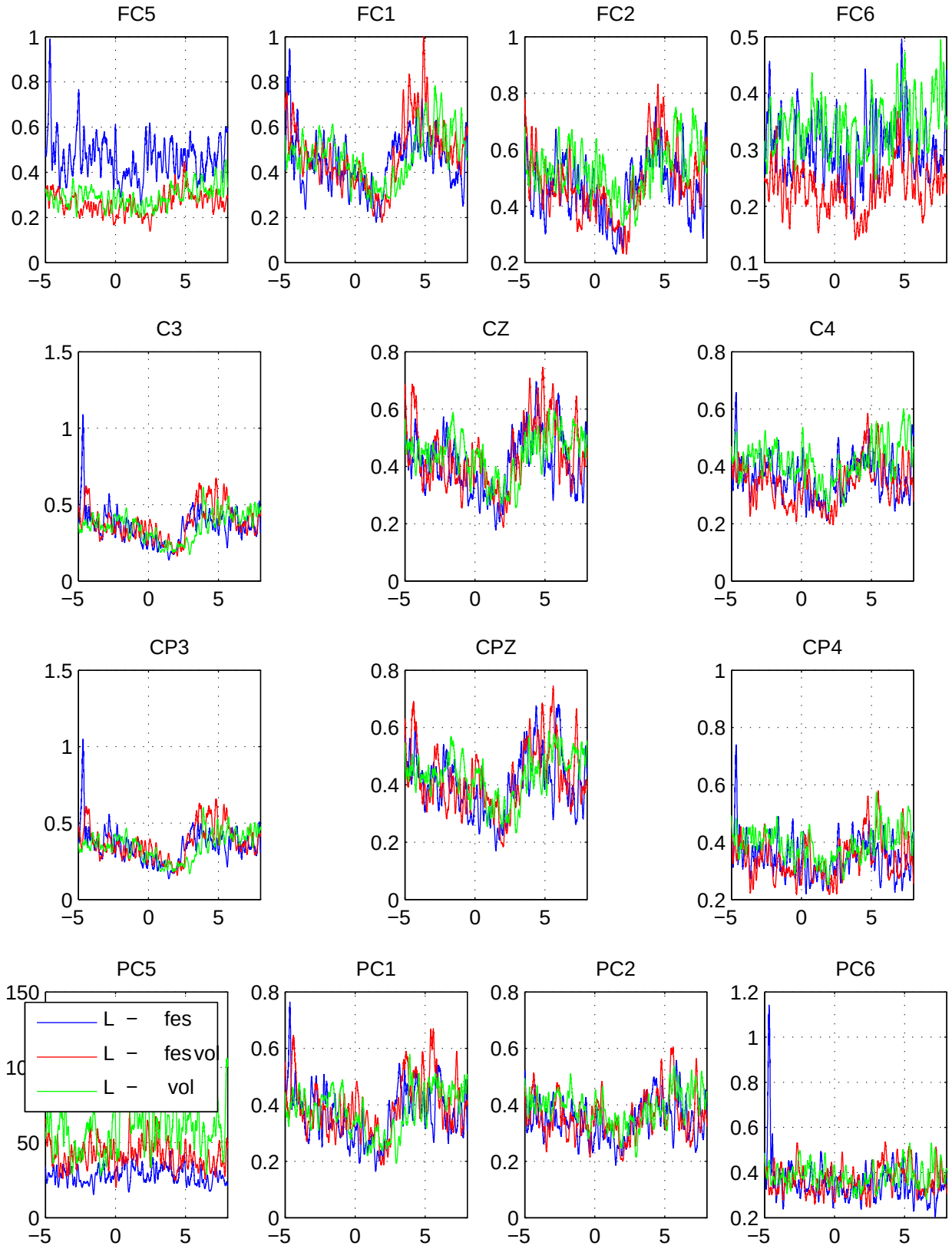


Figure 26: Monopolar beta ERD/ERS time-locked to the beginning of the movements - subject 4; x-axes are in seconds, with 0 being the start of movement, y-axis are in mV

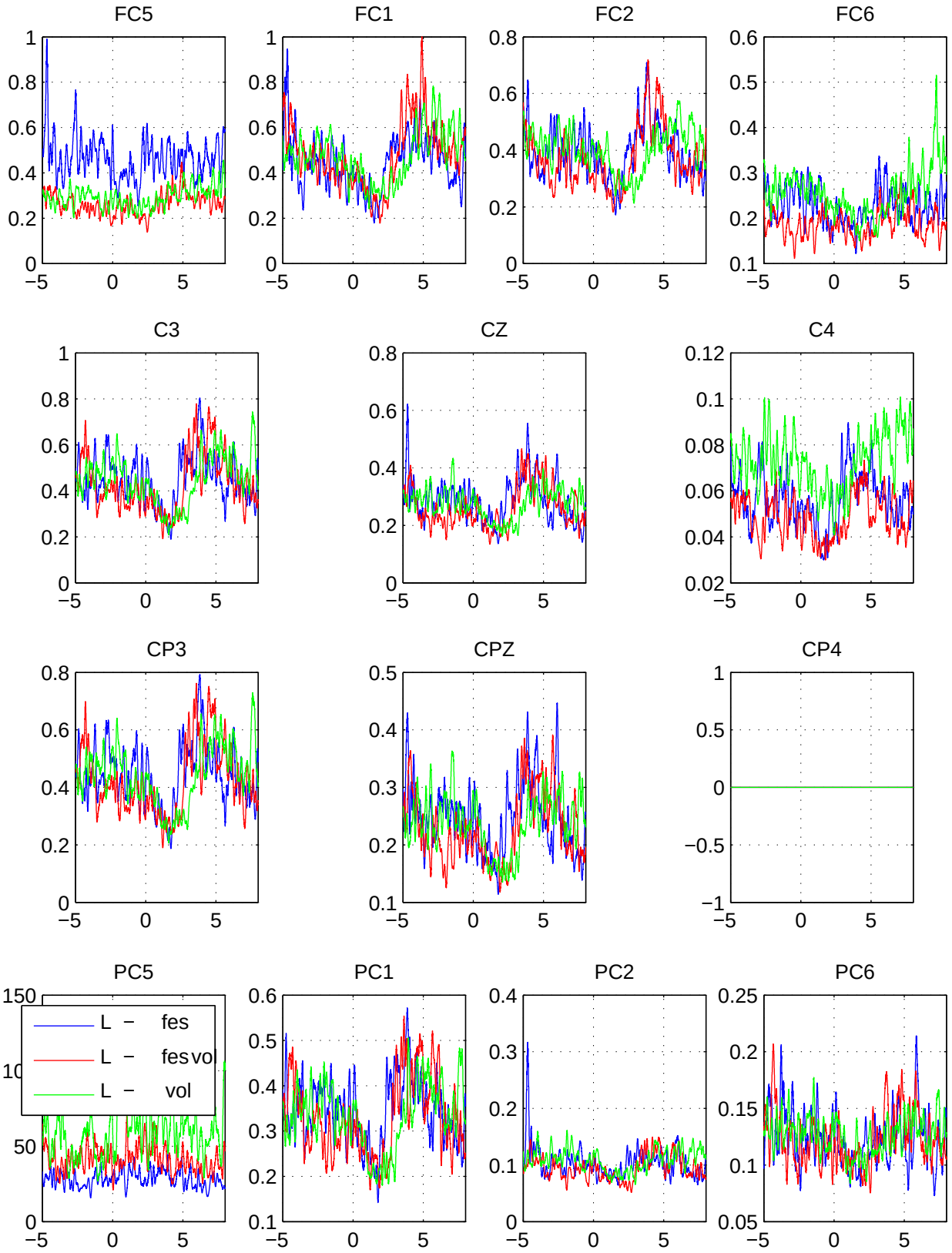


Figure 27: Bipolar beta ERD/ERS time-locked to the beginning of the movement - subject 4; x-axes are in seconds, with 0 being the start of movement, y-axis are in mV

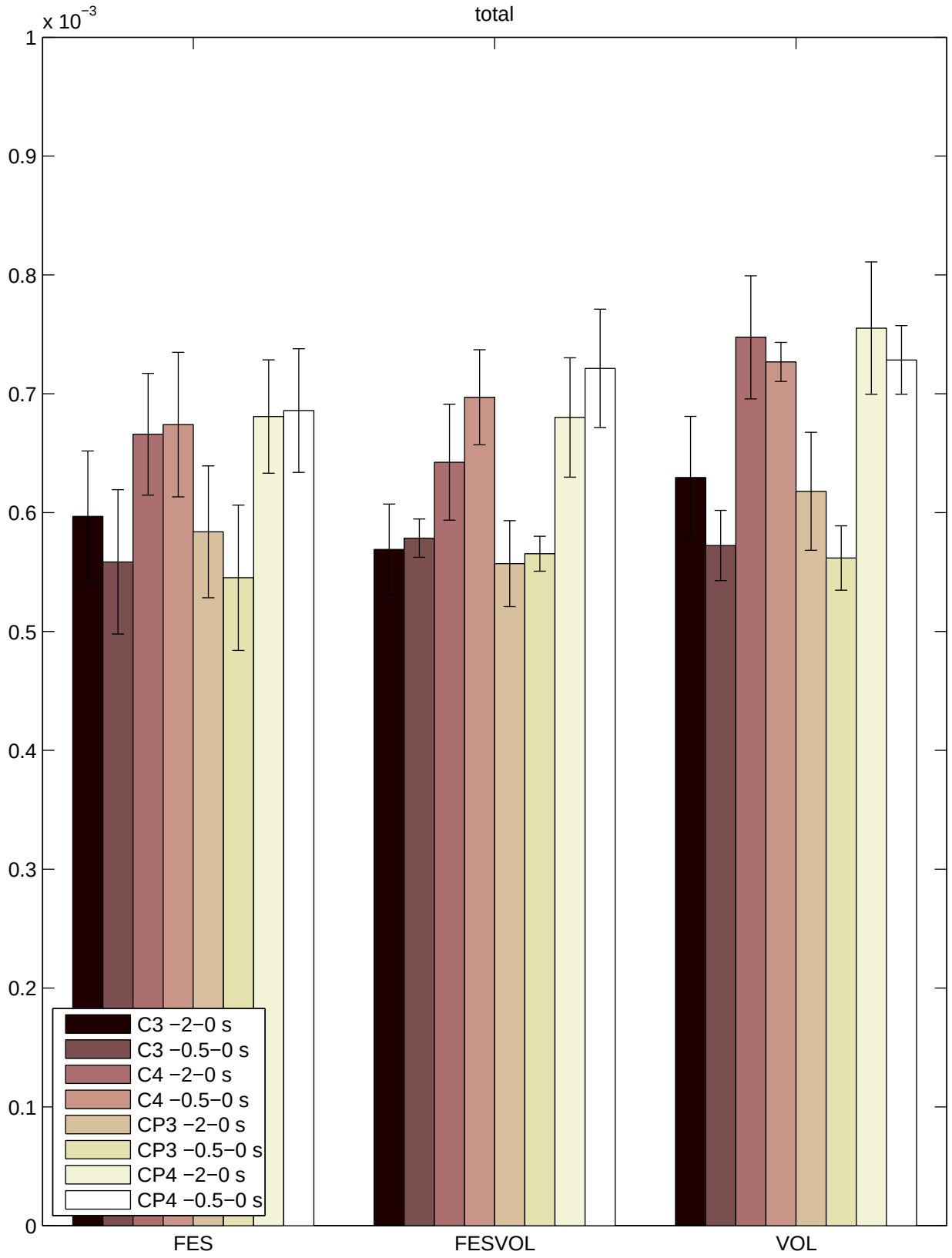


Figure 28: Monopolar baseline power in the beta band before movement onset - all subjects

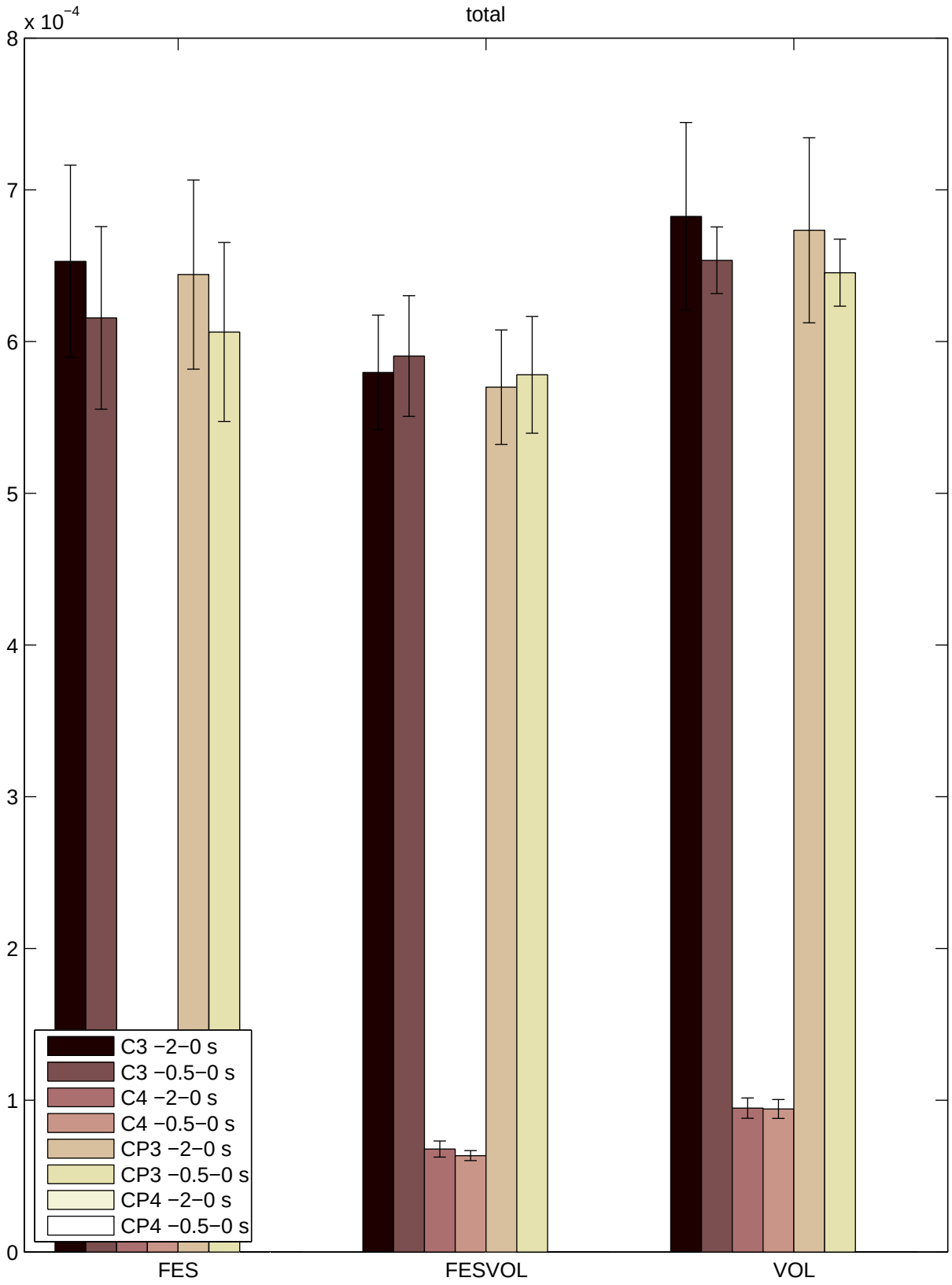


Figure 29: Bipolar baseline power in the beta band before movement onset - all subjects

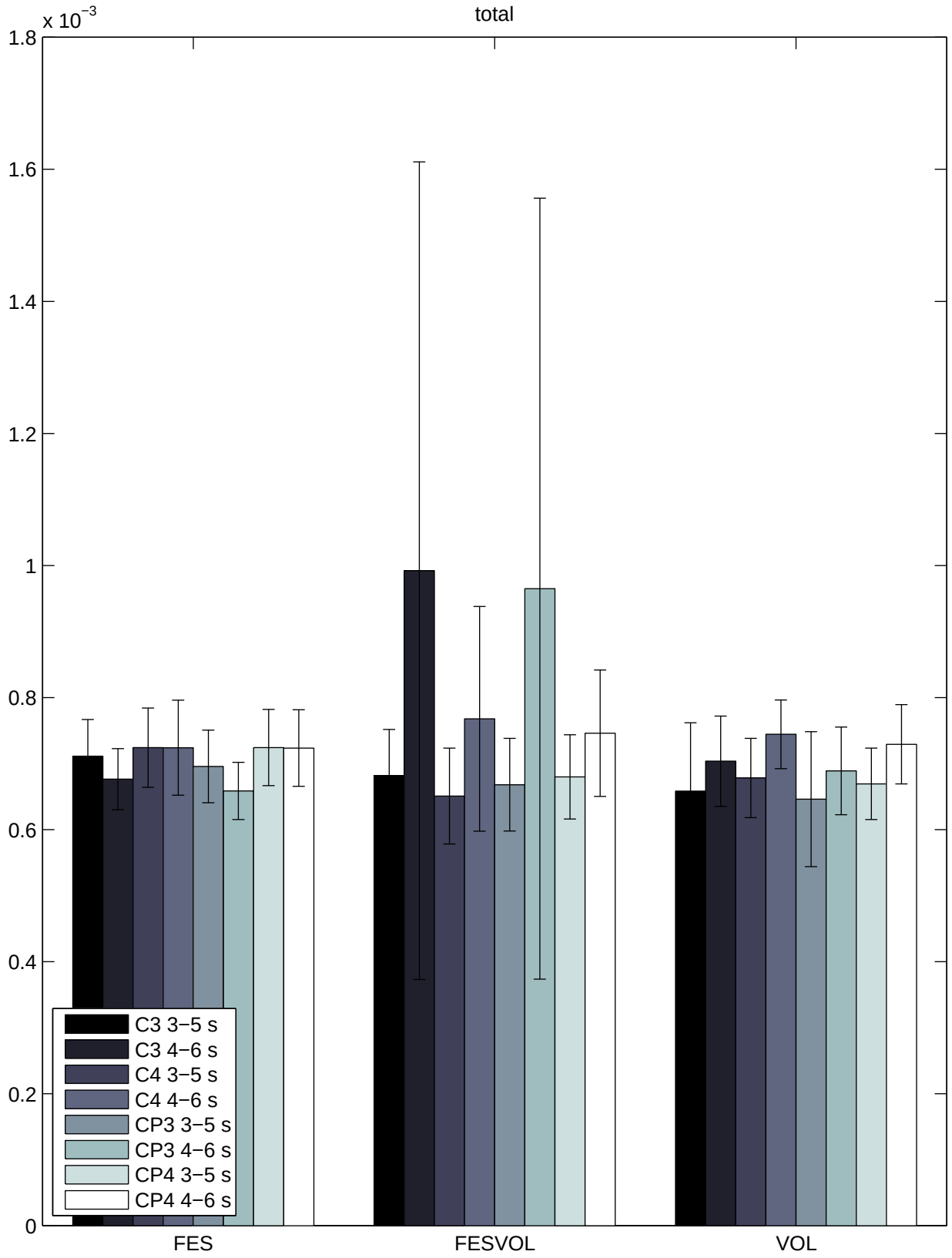


Figure 30: Monopolar beta power after movement end (time-locked to the start of the movement) - all subjects

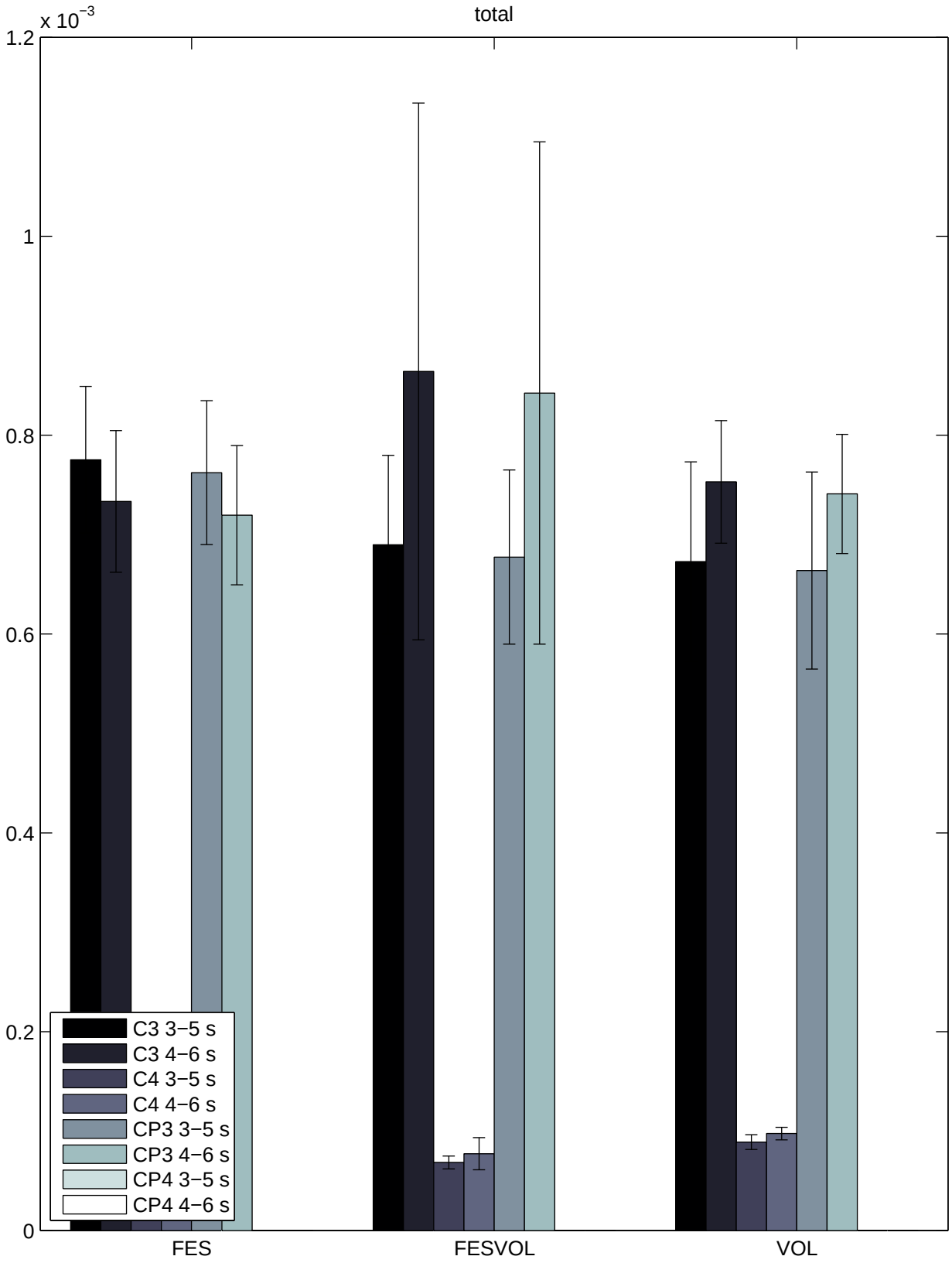


Figure 31: Bipolar beta power after movement end (time-locked to the start of the movement) - all subjects

5 DISCUSSION

5.1 TIME-FREQUENCY MAPS

These 2-dimensional maps weren't analyzed using any algorithm, but is important to note that there are differences visible to the naked eye between the three tasks. These differences support the hypothesis stated in the beggining of this thesis. It is clearly visible that VOL trials have a milder drop in amplitude starting about a second before the movement onset, whereas the other two experiments have a sharper drop following the movement onset, and no visible change in the signal amplitude before the movement onset. The question that remains is how to measure and quantify these differences.

5.2 MOTOR-RELATED CORTICAL POTENTIALS

In the signals of all the subjects, the morphology of MRCPs for the VOL experiment is completely different than for the FES and FES-VOL experiments. VOL MRCPs of these subjects exhibit all of the properties that are expected and known in literature [71, 77, 34]. FES and FESVOL signals of one subject are too contaminated with noise to compare with VOL. FES and FESVOL timeseries of other subjects vary in shape, but all have some defining traits, most notably the (expected) lack of characteristic activity before the movement onset and the sudden drop in amplitude immediately after the movement onset. The only difference between these two experiments is the bigger increase in positivity of the FESVOL signal in 3 subjects compared to the FES signal, resembling the morphology of the late (after movement onset) VOL signal. The features that were selected and presented in the bar graph on Figure 17 show a visible diference between the features of the three experiments, but, when the individual subjects' signals are concerned, the signals vary greatly. It is not known whether these variations are something characteristic to a person or random in nature.

I believe that there is a statistical way to better differentiate between, at least, VOL and the other two types of movement, but, in this thesis, only the displayed features were examined.

5.3 α- AND β-RHYTHM EVENT-RELATED DESYNCHRONIZATION/SYNCHRONIZATION

Looking at baseline features of both monopolar and bipolar recordings in the alpha band, it can be clearly seen that FESVOL signals have the lowest baseline value of the three experiments. This is most probably due to the increased states of preparation and anticipation during these trials because the subjects are waiting for a cue to perform their task. This decreased baseline value indicates that these parts of the brain are activated more than in other trials, which could prove important in the rehabilitation of patients after stroke.

Visual inspection of the signals for the three types of movement show the expected results. VOL trials have the characteristic slow baseline drop in the seconds preceding the movement, and a sharp drop once the movement starts. Once the movement ends, it takes some time for the VOL signal to return to the baseline values, and this increase is gradual. FES trials show a sudden decrease in amplitude once the movement starts, and a sudden increase in amplitude as soon as the movement is completed. FESVOL, as expected presents a combination of these two signals, starting suddenly like FES, and the gradually reverting to baseline, akin to VOL trials. From the selected features of the signals it can be noted that VOL trials have the lowest amplitude during the movement, although not by a large margin. This can be explained by a greater amount of cortical activation when a movement has a voluntary component.

One of the features which were not analyzed in this thesis is the gradual decrease of the VOL movement amplitude before the movement onset, and it seems that this period carries a significant amount of information when a differentiation of the three experiments is required.

ERS of the experiments shows a great variability between subjects, but is important to note that in all of the subjects, one experiment usually yields a higher amplitude of after-movement activity than others. It remains to be seen if this is subject-specific, random, or has some other underlying cause.

Beta signals in the seconds before movement onset and immediately after exhibit the same characteristics as the alpha signals.

5.4 GENERAL DISCUSSION

An issue that I had during this experiment is that one of the channels had to be completely discarded from the analysis because of the noise present in the recording which was the result of a faulty amplifier. A lot of the trials had to be discarded because of the constant resetting and malfunctioning of the equipment.

The issues aside, all of the results are in accordance to the current research and theory. The difference between the results of the original study [11] and this one stem from the differences in the experimental protocol, namely the fact that in this study the subjects didn't start the stimulation by themselves. Because of that, it can be said with greater certainty that were was no preparation before the movement. The differences between the three motor tasks is not that big on first glance, but I am certain that it can be greatly improved by carefully choosing the features and combining them. This matter requires further research. There are no notable differences between monopolar and bipolar signals in any of the experiments and selected features. The results suggest higher cortical activation when electrical stimulation is coupled with voluntary movements which was the original goal of the experiment. This has great potential to improve current practices used in rehabilitation therapy, and, if confirmed by consecutive studies, improves our understanding of the inner processes involved in the movement planning and execution.

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