EEG: Origin and Measurement

2

Fernando Lopes da Silva

1
Introduction to the Electrophysiology of the Brain

The existence of the electrical activity of the brain (i.e. the electroencephalogram or EEG) was discovered more than a century ago by Caton. After the demonstration that the EEG could be recorded from the human scalp by Berger in the 1920s, it made a slow start before it became accepted as a method of analysis of brain functions in health and disease. It is interesting to note that this acceptance came only after the demonstration by Adrian and Mathews (1934) that the EEG, namely the alpha rhythm, was likely generated in the occipital lobes in man, and was not artefactual. However, the neuronal sources of the alpha rhythm remained undefined until the 1970s, when we demonstrated, in dog, that the alpha rhythm is generated by a dipole layer cantered at layers IV and V of the visual cortex (Lopes da Silva and Storm van Leeuwen 1977). It may be not surprising that the mechanisms of generation and the functional significance of the EEG remained controversial for a relatively long time considering the complexity of the underlying systems of neuronal generators on the one hand and the rather involved transfer of signals from the cortical surface to the scalp due to the topological and electrical properties of the volume conductor (brain, cerebrospinal fluid, skull, scalp) on the other.

The EEG consists of the summed electrical activities of populations of neurons, with a modest contribution from glial cells. The neurons are excitable cells with characteristic intrinsic electrical properties, and their activity produces electrical and magnetic fields. These fields may be recorded by means of electrodes at a short distance from the sources (the local EEG or local field potentials, LFPs), or from the cortical surface (the electrocorticogram or ECoG), or at longer distances, even from the scalp (i.e. the EEG, in the most common sense). The associated MEG is usually recorded via sensors that are highly sensitive to changes in the very weak neuronal magnetic fields, which are placed at short distances around the scalp.

F. Lopes da Silva

Centre of NeuroSciences, Swammerdam Institute for Life Sciences, University of Amsterdam, Kruislaan 320, 1098, SM Amsterdam, The Netherlands
e-mail: silva@science.uva.nl

2
# Origin of EEG and MEG I: Cellular Sources

Neurons generate time-varying electrical currents when activated. These are ionic currents generated at the level of cellular membranes; in other words, they consist of transmembrane currents. We can distinguish two main forms of neuronal activation (Lopes da Silva and van Rotterdam 2005; Lopes da Silva 2002; Nunez (1995)): the fast depolarisation of the neuronal membranes, which results in the action potential mediated by the sodium and potassium voltage-dependent ionic conductances gNa and gK (DR), and the slower changes in membrane potential due to synaptic activation, as mediated by several neurotransmitter systems. The action potential consists of a rapid change in membrane potential such that the intracellular potential suddenly jumps from negative to positive, and quickly (in 1 or 2 ms) returns to the resting intracellular negativity. In this way, an impulse is generated that has the remarkable property of propagating along axons and dendrites without any loss of amplitude. Regarding the slower postsynaptic potentials, two main kinds have to be distinguished: the excitatory (EPSPs) and the inhibitory (IPSPs) potentials, which depend on the kind of neurotransmitter and corresponding receptor and their interactions with specific ionic channels and/or intracellular second messengers.

Generally speaking, at the level of a synapse in the case of the EPSP, the transmembrane current is carried by positive ions inwards (e.g. Na^+). In the case of the IPSP, it is carried by negative ions inwards (e.g. Cl^-) or positive ions (e.g. K^+) outwards. Thus, the positive electric current is directed to the extracellular medium in the case of an EPSP and is directed from the inside of the neuron to the outside in the case of an IPSP (Fig. 1).

As a consequence of these currents, an active sink is generated in the extracellular medium at the level of an excitatory synapse, whereas in the case of an inhibitory synapse an active source occurs. The flows of these compensating extracellularly currents depend on the electrical properties of the local tissue. Glial cells occupy an important part of the space between neurons and are coupled to one another by gap junctions. The conductivity of the latter is very sensitive to changes in pH and extracellular K^+ and Ca^{2+}, and can therefore be modulated under various physiological and pathological conditions (Huang et al. 2005). Furthermore, the volume of the extracellular space may change under various physiological and pathological conditions, which will also be reflected in changes in tissue conductivity.

Since there is no accumulation of charge anywhere in the medium, the transmembrane currents that flow in or out of the neuron at the active synaptic sites are compensated by currents that flow in the opposite direction elsewhere along the neuronal membrane. Consequently, in the case of an EPSP, besides the active sink at the level of the synapse, there are distributed passive sources along the soma-dendritic membrane. The opposite occurs in the case of an IPSP: besides the active source at the level of the synapse, distributed passive sinks are formed along the soma-dendritic membrane.

Therefore, we can state that synaptic activity at a given site of the soma-dendritic membrane of a neuron causes a sink–source configuration in the extracellular medium around the neurons. In the context of the present discussion, we have to take into consideration the geometry of the neuronal sources of electrical activity. Indeed, the neurons that mainly

Post-synaptic extra-cellular potentials
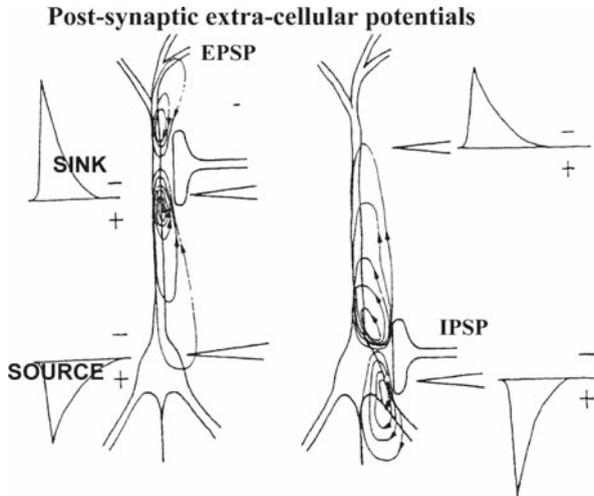
Fig. 1 Scheme of a cortical pyramidal cell showing the patterns of current flow caused by two modes of synaptic activation at an excitatory (*E*) and an inhibitory (*I*) synapse. Typically the apical dendrites of these cells are oriented toward the cortical surface. *EPSP*: current flow caused by the activation of an excitatory synapse at the level of the apical dendrite creates a *current sink* in the extracellular medium next to the synapse. The extracellularly recorded EPSP is drawn on the *left*. It has a negative polarity at the level of the synapse. At the soma there is a distributed passive *current source* resulting in an extracellular potential of positive polarity. *IPSP*: current flow caused by activation of an inhibitory synapse at the level of the soma creates an extracellular source at the level of the soma and a passive sink at the basal and apical dendrites. The IPSP recorded extracellularly at the level of the soma and of the apical dendrites is shown. Note that both cases show a dipolar source–sink configuration. (Adapted from Niedermeyer 2005; Lopes da Silva and van Rotterdam 2005)

contribute to the MEG or the EEG are those that form "open fields" according to the classic description of Lorente de Nó (1947), i.e. the pyramidal neurons of the cortex, since the latter are arranged in palisades with the apical dendrites aligned perpendicularly to the cortical surface. Pyramidal neurons, when activated with a certain degree of synchrony, generate coherent electric/magnetic fields. In this way, these neurons are akin to "current dipoles", the activity of which can be detected by electrodes placed at relatively small distances.

In quantitative terms, our current level of knowledge about the neuronal sources of EEG/MEG signals has benefited a great deal from recent model studies combining in vitro recordings and computational simulations, such as those proposed by the group of Yoshio Okada. These authors adapted the detailed compartmental models of Traub et al. (1994) and Traub and Miles (1991) and applied them to hippocampal slices kept in vitro, and also to the neocortex. Measured electric and magnetic activities were compared with the theoretical results of a computer model. The study of Murakami et al. (2006) in the neocortex is particularly relevant to the present discussion because it has yielded some results that may help to interpret EEG and MEG recordings from the scalp. These authors made a computer model, based on that proposed by Mainen and Sejnowski (1996), of the four main types of cortical neurons, taking into account their realistic shapes. Each neuron

is described as a 3D compartmental model, where each compartment has its typical geometric dimensions, passive electrical properties (membrane capacitance and resistance, intracellular resistance), and five voltage-dependent ionic conductances; the quantitative values of these variables were taken from the literature. For example, the maximal sodium conductance gNa was assumed to be 40 pS μm^{-2} based on the measurements of Stuart and Sakman (1994), but several values were used in a trial and error way to reproduce experimental results. Neuronal activity was obtained by stimulating each neuron with an intracellular current injected at the soma. The intracellular current is represented by a vector quantity Q.

According to this model, the overall magnitude of Q for the activity of one pyramidal neuron of layers V and II/III is on the order of 0.29–0.90 pA, a value that is of the same order of magnitude as that estimated for hippocampal pyramidal neurons (Okada et al. 1997). Murakami et al. (2006) point out that, assuming a Q of 0.2 pA per cortical pyramidal neuron, a population of 50,000 synchronously active cells would generate a field with a magnitude of 10 nA, which corresponds precisely to the value measurable from the human cortex using the MEG according to Hämäläinen et al. (1993). According to the latter, the average value of the volume current density of the cerebral cortex is 175 nA/mm^2 (or nA mm/mm^3) for normal background activity. Assuming a cortical thickness of 3 mm, the average value of the corresponding surface current density is 525 nA/mm, and the average value of the dipole moment $m_i(t)$ associated with a neuronal population i of surface s_i is $M = s_i \times 525$ (nA mm). We will return to these concepts when discussing volume conduction and source estimation.

3
Main Types of Rhythmical EEG/MEG Activities: Phenomenology and Functional Significance

We do not consider all of the different types of rhythmical activities that can be recorded from the brain here, only some prominent activities that are frequently the object of neurocognitive studies, namely sleep rhythms, activities in the alpha frequency range and beta/gamma rhythms. A comprehensive, erudite and thoughtful analysis of these and other brain rhythms can be found in Buzsáki's (2006) monography *Rhythms of the Brain*.

3.1
Sleep EEG Phenomena

In the neurophysiology of sleep, two classic EEG phenomena have been established: the *spindles* or waves between 7 and 14 Hz, also called sleep or *sigma spindles*, which appear at sleep onset, and the *delta waves* (1–4 Hz), which are paradigmatic of deeper stages of sleep. Steriade and his group in Quebec (see Sect. 2) described another very slow oscillation (0.6–1 Hz) in animals that is able to modulate the occurrence of different typical EEG sleep events, such as delta waves, sleep spindles and even short, high-frequency bursts.

This very slow oscillation has now also been demonstrated in the human EEG and in the MEG, as indicated above.

The sleep spindles are generated in the thalamocortical circuits and result from the interplay between intrinsic membrane properties of the thalamocortical relay neurons (TCR) and of the GABAergic neurons of the reticular nucleus and the properties of the circuits to which these neurons belong. It is clear that the spindles are a collective property of the neuronal populations. Experimental evidence has demonstrated that the sleep spindle oscillations are generated in the thalamus since they can be recorded in this brain area after decortication and high brain stem transection. However, the very slow rhythm (0.6–1 Hz) is generated intracortically, since it survives thalamic lesions but it is disrupted by intracortical lesions. Interestingly, we may note that the rhythmicity of the very slow oscillation appears to be reflected in that of the typical K-complexes of human EEG during non-REM sleep (Amzica and Steriade 1997).

One question is: how are these oscillations controlled by modulating systems? It is well known that sleep spindles are under brain stem control. It is a well-known neurophysiological phenomenon that electrical stimulation of the brain stem can block thalamocortical oscillations, causing "EEG desynchronisation", as shown in classic studies by Moruzzi and Magoun (1949). This desynchronisation is caused mainly by the activation of cholinergic inputs arising from the mesopontine cholinergic nuclei, namely the pedunculopontine tegmental (PPT) and the laterodorsal tegmental (LTD) areas. Indeed, both the reticular nucleus and the TCR neurons receive cholinergic muscarinic synapses. Cholinergic activation of the reticular nucleus neurons elicits hyperpolarisation with an increase in K^+ conductance, which is mediated by an increase in a muscarinic-activated potassium current, and in contrast it causes depolarisation of TCR neurons. Furthermore, the reticular nucleus receives inputs from the basal forebrain that may be GABAergic and can also exert a strong inhibition on the reticular neurons, leading to the subsequent suppression of spindle oscillations. In addition, monoaminergic inputs from the brain stem, namely those arising at the mesopontine junction (i.e. from the noradrenergic neurons of the locus coeruleus and the serotoninergic neurons of the dorsal raphe nuclei) also modulate the rhythmic activities of the forebrain. These neuronal systems have only a weak thalamic projection but they have a diffuse projection to the cortex. Metabotropic glutamate receptors also appear to exert a modulating influence on the activation of thalamic circuits by descending corticothalamic systems.

Because this point is often misunderstood, we should emphasise that slow-wave sleep, characterised by typical EEG delta activity, does not correspond to a state where cortical neurons are inactive. On the contrary: in this sleep state cortical neurons can display mean rates of firing similar to those that they show during wakefulness and/or REM sleep. Regarding the neuronal firing patterns, the main difference between delta sleep on the one hand and wakefulness and REM sleep on the other is that, in the former, the neurons tend to display rather long bursts of spikes with relatively prolonged interburst periods of silence, whereas in the latter the firing pattern is more continuous. The functional meaning of these peculiar firing patterns of delta sleep has not yet been unravelled.

In general terms we can state that EEG signals co-vary strongly with different levels of arousal and consciousness. The changes in EEG with increasing levels of anaesthesia are typical examples of this property.
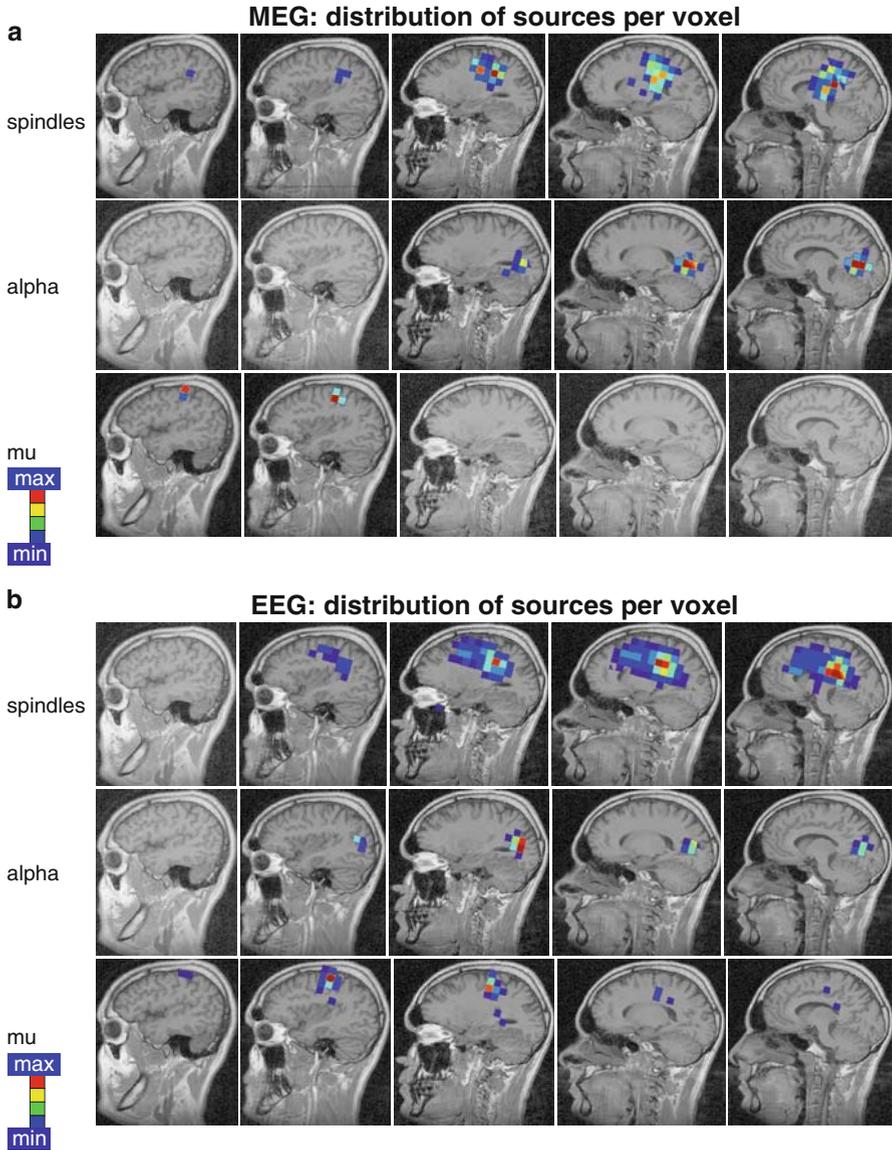
3.2
Alpha Rhythms of Neocortex and Thalamus

Alpha rhythms recorded from the occipital areas occur in relaxed awake animals and show a typical reactivity to closure of the eyes. Background illumination can result in decreased alpha rhythm amplitude (Paskewitz et al. 1973; Cram et al. 1977), while investigations of the suspected relationship between heart rate and alpha have not led to any firm conclusions (Stenett 1966; Surwillo 1965, 1967).

Although the frequency range of alpha rhythms overlaps that of sleep spindles, these two types of phenomena differ in a number of aspects. Namely, the behavioural states at which these types of oscillations occur are quite different, and their distributions over the thalamus and cortex also differ considerably, as exemplified by Fig. 2. The basic mechanisms responsible for alpha oscillations at the cellular level have not been described in detail. The reason for this is the inherent difficulty of studying a phenomenon that—by definition—occurs in the state of relaxed wakefulness, under conditions where measurement of the underlying membrane currents is not a simple task, since this cannot be done under anaesthesia. To overcome this difficulty, some researchers have assumed that spindles occurring under barbiturate anaesthesia are analogous to alpha rhythms. However, this analogy was challenged on experimental grounds because a comparative investigation of alpha rhythms obtained during restful wakefulness upon closure of the eyes, and spindles induced by barbiturates, recorded from the same sites over the visual cortex and lateral geniculate nuclei in dog, presented differences in frequency, spindle duration, topographic distribution and amount of coherence among different cortical and thalamic sites. Investigations combining multiple electrode arrays placed on the cortical surface, intracortical depth profiles and intrathalamic recordings from several thalamic nuclei unravelled a number of elementary properties of alpha rhythms (Lopes da Silva 1991):

- In the visual cortex, alpha waves are generated by a current dipole layer centred at the level of the somata and basal dendrites of the pyramidal neurons of layers IV and V
- The coherence between alpha waves recorded from neighbouring cortical sites is greater than any thalamocortical coherence
- The influence of alpha signals recorded from the pulvinar on cortical rhythms can be conspicuously large, depending on the cortical area, but intracortical factors play a significant role in establishing cortical domains of alpha activity

These experimental findings led to the conclusion that, in addition to the influence of some thalamic nuclei (mainly the pulvinar) on the generation of alpha rhythms in the visual cortex, there are systems of surface-parallel intracortical connections that are responsible for the propagation of alpha rhythms over the cortex. These oscillations appear to be generated in small patches of cortex that behave as epicentres, from which they propagate at relatively slow velocities, about 0.3 cm s^{-1}. This type of spatial propagation has been confirmed, in general terms, by experimental and model studies. A comprehensive study of alpha rhythms in the visual cortex of the cat (Rougeul-Buser and Buser 1997; Buser and Rougeul-Buser 2005) showed characteristics corresponding closely to those of alpha rhythms in man and in dog. It was found that this rhythmic

a

MEG: distribution of sources per voxel

spindles

alpha

mu

max

min

b

EEG: distribution of sources per voxel

spindles

alpha

mu

max

min

Fig. 2 a–b Dipole density plots of the MEG and EEG sleep spindles, alpha and mu rhythms of one subject. Voxels containing a relative high amount of dipoles are shown in *red*. *Blue* voxels contain relatively few dipoles. Voxels comprising less than 10% of the maximal amount of dipoles present in the red voxels are omitted for clarity. **a** MEG data: the "hot spots" for the MEG spindles are located in the centroposterior areas; **b** EEG data: the plots demonstrate that there is no overlap of the alpha and mu clusters. Furthermore, the "hot spot" of the alpha rhythm is located more superficially than those of the spindles, whereas the spindle cluster is more widespread than that of the alpha rhythm. The EEG sleep spindle dipoles spread to more frontal areas than the MEG data. (Adapted from Manshanden et al. 2002)

activity was localised to a limited part of the primary visual cortex area 18 and the border between 17 and 18. In this context, more insight into the sources of alpha rhythms in man was obtained using EEG and MEG recordings integrated with anatomical information obtained from magnetic resonance images (MRI), as shown in Fig. 2. Different sources of alpha rhythms were found to be mainly concentrated in the region around the calcarine fissure, with most sources occurring within 2 cm from the midline. In addition to the alpha rhythms of the visual cortex, rhythmic activities with about the same frequency range (in man: 8–13 Hz; in cat: 12–15 Hz) have been shown to occur in other cortical areas, namely in the somatosensory cortex (SI areas 1, 2 and 3). These activities are known as "rolandic *mu rhythms*", or "wicket rhythms" (named after the appearance of the records on the scalp in man), and have a typical reactivity, since they appear when the subject is at rest and are blocked by movement. The mu rhythm is particularly pronounced in the hand area of the somatosensory cortex, and it reacts typically to the movement of closing the fists. In the cat, there is no significant coherence between the mu rhythm of the SI cortex and the alpha rhythm of the visual cortex, which supports the general idea that these two types of rhythms are independent. Furthermore, mu rhythms of the SI area also differ from the alpha rhythms of the visual cortex recorded in the same animal, in that the former have systematically higher frequencies than the latter, the difference being about 2 Hz. Mu rhythms were also recorded in thalamic nuclei, namely in the ventroposterior lateral nucleus. The mu rhythm has also been identified in MEG recordings over the Rolandic sulcus, particularly over the somatomotor hand area. In addition, another spontaneous MEG activity, the so-called *tau rhythm*, was detected over the auditory cortex. This rhythmic activity was reduced by sound stimuli. This MEG tau rhythm, which was first described by the group of Hari (Hari et al. 1997; Lehtelä et al. 1997), is apparently similar to an EEG rhythm that was found using epidural electrodes over the midtemporal region by Niedermeyer (2005), who called it "third rhythm" or "independent temporal alphoid rhythm".

The cellular mechanisms responsible for the generation of alpha rhythms have recently been unveiled using in vitro preparations of thalamic nuclei. Hughes et al. (2004) showed that in the lateral geniculate nucleus, oscillations in the alpha frequency range can be generated by the pharmacological activation of the metabotropic glutamate receptor (mGluR) mGluR1a. These oscillations display similarities with thalamic alpha rhythms recorded in the intact animal. Hughes and Crunelli (2005) discovered that the occurrence of these oscillations depends on the activity of a subset of thalamocortical (TC) neurons termed high-threshold (HT) bursting cells, which are interconnected via gap junctions. These in vitro thalamic alpha rhythms can slow down until the theta frequency range when the TC neuron population is less depolarised.

3.3
Beta/Gamma Activity of the Neocortex

The identification and characterisation of high-frequency rhythms in the neocortex has concentrated mainly on two neocortical areas, the visual cortex and the somatomotor cortex. Here we examine here some of the properties of the *beta/gamma* rhythmic activities

for these two areas, although beta/gamma rhythmic activities have also been recorded in olfactory brain areas, particularly by Freeman (2005).

Commonly, the EEG of the *visual cortex* is associated with the alpha rhythm, with its typical reactivity upon closing and opening the eyes, as described above. However, other types of rhythmic activities can be present in the same cortical areas, namely within the beta frequency range. In the dog, we showed that the EEG spectral density was character-ised by peaks within the beta/gamma frequency range while the animal was looking atten-tively at a visual stimulus (Lopes da Silva et al. 1970). Similarly, Freeman and van Dijk (1987) found in the visual cortex of a rhesus monkey that fast EEG rhythms (spectral peak of 30 ± 3.7 Hz) occurred during a conditioned task in response to a visual stimulus. A related finding is the discovery by the group of Charles Gray and Wolf Singer (Gray et al. 1989) and by Eckhorn et al. (1988) of oscillations within the beta/gamma frequency range (most commonly between 30 and 60 Hz) in the firing of individual neurons of the visual cortex in response to moving light bars. It was demonstrated using auto- and cross-correlation analyses that neurons tended to fire in synchrony, in an oscillatory mode, within cortical patches that could extend up to distances of about 7 mm. The oscillations in neuronal firing rate were correlated with those of the LFPs. The cortical oscillations are modulated by the activation of the mesencephalic reticular formation (MRF), but the stimulation of the MRF alone does not change the pattern of firing of the cortical neurons (Munk et al. 1996). However, MRF stimulation increases the amplitude and coherence of both the LFP and multiunit responses when applied jointly with a visual stimulus.

In the *somatomotor cortex*, beta/gamma oscillations of both neuronal firing and LFPs were also described in the awake cat by the group of Buser and Rougeul-Buser (2005), Bouyer et al. (1987) particularly when the animal was in a state of enhanced vigilance while watching an unreachable mouse. Also, fast oscillations were found in the somatomo-tor cortex in monkey during a state of enhanced attention (Rougeul et al. 1979). Oscillations of 25–35 Hz occurred in the sensorimotor cortex of awake, behaving monkeys in both LFPs and single-/multiunit recordings. They were particularly apparent during the perfor-mance of motor tasks that required fine finger movements and focussed attention. These oscillations were coherent over cortical patches extending up to at least 14 mm that included the cortical representation of the arm. Synchronous oscillations straddling the central sul-cus were also found, so they may reflect the integration of sensory and motor processes. The LFP reversed polarity at about 800 μm under the cortical surface, indicating that the source of the LFP is in the superficial cortical layers. It is noteworthy that at least some of the cortical beta/gamma rhythmic activities appear to depend on projecting dopaminergic fibres arising in the ventral tegmental area, but the extent to which the beta rhythms of the somatomotor cortex are related to thalamic or other subcortical activities is not yet clear.

With respect to the origin of beta/gamma rhythmic activity, several experimental facts have led to the interpretation that these rhythmic activities are primarily generated in the cortex itself. These include the fact that oscillations in the beta/gamma frequency range were easily recorded from different cortical sites but not from simultaneously obtained recordings from thalamic electrodes; the observation that in the visual cortex there are neurons that show oscillatory firing rates with a phase difference of about a quarter cycle, which indicates that a local recurrent feedback circuit may be responsible for the oscilla-tions; the finding of intrinsic oscillations in cortical neurons from layer IV of the frontal

cortex of guinea pig in vitro. Nevertheless, it is possible that thalamic neuronal networks also contribute to the cortical beta/gamma rhythmic activity, since oscillatory (about 40 Hz) behaviour has been observed in neurons of the intralaminar centrolateral nucleus, which projects widely to the cerebral cortex, by Steriade et al. (1996). The question cannot be phrased as a simple alternative between a cortical or a thalamic rhythmic process, both considered to be exclusive mechanisms. As we have discussed in relation to other rhythmic activities of the mammalian brain, *both network- and membrane-intrinsic properties cooperate* in shaping the behaviour of the population, including its rhythmic properties and its ability to synchronise the neuronal elements. Recently, new observations made in vitro have shed light on the sources of these fast cortical rhythms. In an in vitro model of the cortex, the group of Whittington and Traub (Roopun et al. 2006) showed concurrent but independently generated gamma (30–70 Hz) rhythms in layer II/III and beta2 (20–30 Hz) rhythms in layer V somatosensory cortex. The beta2 rhythm occurred robustly in layer V intrinsically bursting (IB) neurons in the form of bursts admixed with spikelets, and single action potentials. It was blocked by reducing gap junction conductance with carbenoxolone, and was unaffected by the blockade of synaptic transmission sufficient to ablate the layer II/III gamma rhythm. It could also be seen in the absence of synaptic transmission with axonal excitability enhanced with 4-aminopyridine, suggesting a nonsynaptic rhythm mediated by axonal excitation. A network model based on the hypothesis of electrical coupling via axons, is consistent with this hypothesis. The frequency of this network beta2 rhythm appears to depend on the magnitude of the M current, a non-inactivating potassium current found in many neuronal cell types that can be modulated by a large array of receptor types, including muscarinic cholinergic receptors, in IB interneurons. These findings suggest the possibility that a normally occurring cortical network oscillation involved in motor control could be generated largely or entirely by nonsynaptic mechanisms. According to these authors, higher beta2 frequency oscillations occur mainly during the anticipatory period leading up to a movement in response to a sensory cue. Indeed, it was found that layer V pyramidal neurons and motor cortex LFPs displayed coherence at beta2 frequencies with hand and forearm EMG in monkeys performing a precision grip task (Baker et al. 1999). This group showed that the generation of beta2 in layer V stands in contrast to that of gamma rhythms in layers II/III, which may underlie corticocortical synchronisation. There are probably a variety of rhythmic activities in the beta/gamma range with different behavioural correlates, as discussed below with respect to event-related (de)synchronisation of EEG/MEG phenomena.

In this context it is interesting to consider changes in EEG/MEG phenomena, particularly in the beta and gamma frequency ranges, that are event-related and reflect a decrease or an increase in the synchrony of the underlying neuronal populations. The former is called event-related desynchronisation (ERD), and the latter event-related synchronisation (ERS) (Pfurtscheller and Lopes da Silva 1999). In relation to a hand movement, both the 10–12 Hz mu rhythm and the beta (around 20 Hz) display ERD, but with a different distribution over the scalp, although both activities are localised around the central sulcus. The mu rhythm ERD exhibits its maximum magnitude more posteriorly than the beta activity, indicating that it is generated mainly in the post-Rolandic somatosensory cortex, whereas the low beta activity is preferentially generated in the pre-rolandic motor area. In addition,

after a voluntary movement, the central region exhibits a localised beta ERS that becomes evident in the first second after cessation of the movement, at a time where the rolandic mu rhythm still presents a desynchronised pattern. The exact frequency of this rebound beta ERS can vary considerably with the subject and type of movement. This beta ERS is observed not only after a real movement but also after an imagined movement. Furthermore. ERS in the gamma frequency band (around 36–40 Hz) can also be found over the central regions, preceding the execution of a movement, in contrast with the beta ERS, which has its maximum after the termination of the movement (Fig. 3).

Our understanding of the significance of ERS of the *beta* frequency range, which typically occurs after a movement, has been greatly enhanced by the observation that when this form of ERS occurs, the excitability of the corticospinal pathways decreases, as revealed by means of transcranial magnetic stimulation. This supports the hypothesis that the postmovement beta ERS corresponds to a deactivated state of the motor cortex. In contrast, the ERS in the *gamma* frequency band appears to reflect a state of active information processing.

From this perspective, it is important to note the likely connection between gamma oscillations and synaptic plasticity. As Buzsáki (2006) colourfully writes, the gamma oscillation may be considered the "buzz" that provides the central timing mechanism that is essential for modulating synaptic strength, and in this way it may stabilise the formation of neuronal assemblies in the cortex.
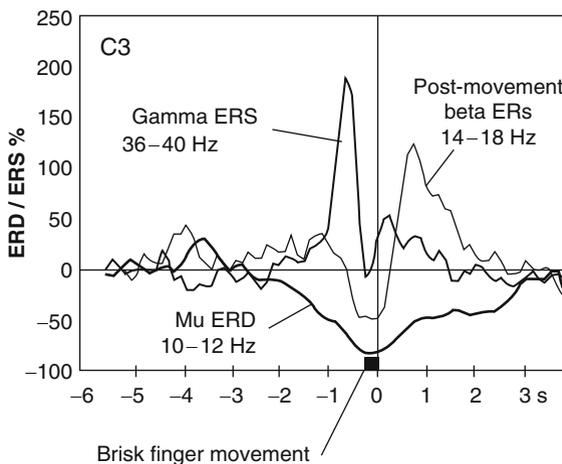
Fig. 3 Event-related desynchronisation (ERD) and event-related synchronisation (ERS) from one normal subject during self-paced voluntary movement. EEG recorded from C3. The results for three frequency bands are shown: alpha band (mu) 10–12 Hz ERD; beta 14–18 Hz ERD–ERS, and gamma 36–40 Hz ERS. The data analysis is triggered with respect to movement offset (vertical line at 0 s). Note that the ERDs or ERSs of different frequency bands have different dynamics: about 2 s before the movement the mu ERD appears, followed by a pre-movement beta ERD that changes to a postmovement ERS; a burst of gamma ERS appears just before the movement. (Adapted from Pfurtscheller et al. 1993)

3.4
DC

Is it possible to record DC on MEG or EEG? And if so, what is its physiological meaning? There are a lot of physical limitations (electrode impedances, electrode polarisation, skin/electrolyte junction) that do not allow EEG signals to be recorded down to 0 Hz, which would correspond to real DC, or "direct current". Also, environmental low-frequency noise imposes limitations on MEG with similar consequences. The point, however, is not to record down to the real DC level but to extend the effective frequency band to very low frequencies on the order of 0.1 Hz. (For a discussion of misconceptions of the meaning of "DC" in electroencephalography, see Niedermeyer's footnote in Speckmann and Elger 2005). The recording of ultraslow MEG/EEG signals can be achieved using appropriate techniques, as discussed, for example, for the EEG by Vanhatalo et al. 2005 and for the MEG by Burghoff et al. 2004. Phenomena such as the contingent negative variation (CNV) and the Bereitschaftspotential (readiness potential) are typical cases of very slow shifts of electric potential or magnetic fields that can be typically recorded using appropriate recording and analysis techniques. During slow-wave sleep, ultraslow-frequency components (around 0.5 Hz) have been recorded in the human EEG (Achermann and Borbély 1997; Amzica and Steriade 1997) and in MEG (Simon et al. 2000), which correspond to the ultraslow oscillations that can be recorded intracellularly from cortical neurons through layers II to VI, and consist of prolonged depolarising and hyperpolarising components, as have been analysed in detail by Steriade (2006).

4
Origin of the EEG/MEG II: Generators, Volume Conduction and Source Estimation

In order to take the next step towards an understanding of how EEG/MEG signals recorded outside the skull are generated, we have to take the folding of the cortex into consideration. The fact that the cortex is folded, forming gyri and sulci, implies that some populations of neurons have apical dendrites that are perpendicular to the overlying skull (i.e. those that are at the top of a gyrus), whereas others are parallel to the skull (i.e. those that are on the wall of a sulcus). The orientation of the neurons with respect to the skull is an important factor in the appearance of the EEG and MEG signals recorded outside the skull. This is particularly the case for the MEG, since the latter "sees" only those magnetic fields that are perpendicular to the skull due to the physical properties of magnetic fields, the way MEG is measured and the approximately spherical shape of the head. In effect, the observed magnetic fields are generated by neuronal currents that are oriented tangentially to the skull. In contrast, those that are oriented radially to the skull do not generate a magnetic field outside the head but contribute to the EEG.

The area of cortex within which the neuronal population must be synchronously active to produce a measurable EEG/MEG signal at the scalp is an important consideration when interpreting these signals. To address this problem, we must first point out some classical

concepts about cortical organisation. The cortex is organised according to the columnar principle, as proposed in the 1970s by Mountcastle (see review from 1997), which means that the basic unit of the mature neocortex is the *minicolumn*: "a narrow chain of neurons extending vertically across the cellular layers II/VI, perpendicular to the pial surface", with a cross-section of diameter ~40–50 μm. A primate minicolumn contains about 80–100 neurons, although this number may vary between areas; in the striate cortex the cell density appears to be 2.5 times larger. Many minicolumns are bound together by short-range horizontal connections, and thus form what has been denominated *cortical columns* or cortical modules (Mountcastle 1997). One *column* in the somatic sensory cortex contains about 80 minicolumns and is roughly hexagonal with a width of about 300–400 μm (Favorov and Diamond 1990). These estimates can be used to give a rough answer to the question formulated above. Assuming that a minicolumn with a diameter of 40 μm contains 100 cells, the cortical surface corresponding to 50,000 cells should form a patch with a cross-sectional area of about 0.63 mm^2. If this cortical patch took a circular form, then its diameter would be about 0.88 mm. Buxhoeveden and Casanova (2002) noted that, from a functional perspective, cortical columns may exist in different dynamic states, and they coined the term "physiological macrocolumn" to indicate a set of cortical columns that cooperate in a given functional state or neural process. These physiological macrocolumns must be considered dynamic ensembles such that the number of columns contributing to a macrocolumn may vary as a function of time. It is important to note that neurons in separate columns can present synchronous oscillatory activities, as mediated by tangential and recurrent connections between different columns (Gray et al. 1989; Freiwald et al. 1995).

A basic problem in electroencephalography/magnetoencephalography is how to estimate the neuronal sources that are responsible for a certain distribution of electrical potentials or of magnetic fields recorded at the scalp. This is called the inverse problem of EEG/MEG, and is an ill-posed problem in the sense that it has no unique solution: there are an infinite number of possible source configurations that give rise to a given set of measured scalp potentials or magnetic fields (von Helmholtz 2004). Therefore, the estimation of EEG and MEG sources requires assumptions about the nature of the sources. The simplest source model is a current dipole, as indicated in the previous section. However, such a model does not imply that somewhere in the brain there is a point current dipole. Rather, it suggests that the EEG/MEG scalp distribution is best represented by an *equivalent* dipolar current source. This choice has been shown to be useful and accurate for certain types of activity, such as the event-related potentials (ERPs) and focal epileptic spikes. In such circumstances, the solution of the EEG/MEG inverse problem that is obtained based on the equivalent current dipole can be thought of as the centroid of the dipole layers that are active at a certain moment, in the statistical sense. An increase in the number of dipoles can easily lead to rather complex and ambiguous interpretations. Nevertheless, methods have been developed in order to obtain estimates of multiple dipoles using only the a priori criterion that they must be located at the surface of the cortex. An example of an algorithm that performs such an analysis is MUSIC (multiple signal classification). An alternative approach is to use linear estimation methods that apply the minimum norm constraint to estimate the sources within a given surface or volume of the brain.

The other main component of any estimation of EEG/MEG sources is the conduction problem, which allows the calculation of the field values given a postulated generator configuration (the so-called forward part of the inverse problem). A commonly used forward model is that of the three concentric conducting spheres, for which a convenient analytical expression for the surface potentials (or magnetic field) due to a dipole is available. However, realistically shaped volume conductor models derived from the individual subject's MRI images have been shown to improve the accuracy of the localisation of the sources, particularly in nonspherical parts of the head (Fuchs et al. 2007). Individual boundary element method (BEM) models derived from the subject's MRI represent the "gold standard" and have clear advantages over simplified spherical shell models. Anisotropic volume conduction properties of the bone layer or the white matter fibres can be modelled by the finite element method (FEM), but the latter require considerable computational power and are thus not used in daily applications. To reduce the computational effort, head models derived from an averaged MRI dataset have been proposed (Fuchs et al. 2007). New approaches are currently being explored that combine fMRI and EEG/MEG data in order to create more specific spatial constraints in order to reduce the solution space for the estimation of the underlying neuronal sources. This aspect of EEG and fMRI data fusion is addressed in more detail in the chapter "EEG–fMRI in Animal Models".

In general, the problems posed by the complexity of the volume conductor, including scalp, skull, cerebrospinal fluid layer and brain, are easier to solve in the case of the MEG than of the EEG, since these different media have different conductivities, which affects the EEG much more than the MEG. Therefore, a major advantage of MEG over EEG is the relative simplicity of the forward modelling and its consequences for source localisation. This means that when a dipole source algorithm is used on the basis of MEG recordings, a single homogeneous sphere model of the volume conductor can lead to a satisfactory solution.

The conductivity values that should be used for the different shells surrounding the sources, brain, cerebrospinal fluid, skull and scalp have been estimated in a number of studies, using both in vitro and in vivo measurements. In general, we can assume that the brain and scalp have the same conductivity. The conductivity of the skull is much lower than that of the brain, but the estimation of the ratio of the brain and skull conductivities has been a matter of debate. Our group (Gonçalves et al. 2003) estimated the conductivities in vivo using two different methods: electrical impedance tomography and a combined analysis of the evoked somatosensory cortical response, recorded simultaneously using MEG and EEG, since the former is not affected by the different conductivity of the skull, in contrast to the latter. The electrical impedance tomography results show a wide variation in the ratio of resistivities r_{skull}/r_{brain} among subjects, but in all cases studied the ratio was lower than the classically accepted value of 80 (Rush and Driscoll 1968), and it was in the range of 20–50. However, the r_{skull}/r_{brain} ratios of the individual subjects are readily reproducible. These results indicate that the r_{skull}/r_{brain} variations over subjects cannot be disregarded when solving the EEG inverse problem when a spherical model is used. In order to obtain an estimate of the sources of a given potential distribution over the scalp as precisely as possible, conductivities measured in the same subject should preferentially be used.

5
Localisation Methods Applied to Spontaneous Oscillatory Activities

A basic question in EEG/MEG studies is whether the main rhythmic activities—alpha and mu rhythms on the one hand and sleep spindles on the other—are generated in distinct or overlapping cortical areas. In order to solve this question, advanced spatiotemporal analysis methods are necessary. We should note that the estimation of equivalent dipole models is only meaningful if the scalp field has focal character and the number of possible active areas can be anticipated with reasonable accuracy. The recent development of a new algorithm (Manshanden et al. 2002) aimed at estimating sources of large data sets, as is the case for this kind of spontaneous EEG oscillation, allowed the issue of whether generators of spontaneous MEG/EEG alpha and mu rhythms and sleep spindles are distributed over distinct or over overlapping cortical areas to be addressed. The basic approach consisted of finding well-fitting dipoles using a dipole model applied to successive time samples of a burst of an oscillation. The equivalent dipoles encountered were plotted on the corresponding MRI slice of the brain, as shown in Fig. 2 (A for the EEG, and B for the MEG). It is important to consider what equivalent dipolar sources of spontaneous brain activity may represent. Sleep spindles, alpha rhythms and mu rhythms are spontaneously occurring brain rhythms that can be recorded from the scalp. This suggests that extended cortical areas are involved in the generation of these signals. The use of equivalent dipoles as source models for these distributed brain activities yields an oversimplified solution to the problem of determining the underlying sources of these signals. The equivalent dipoles should be viewed simply as descriptors of the "centre of gravity" that best describe, in a statistical sense, the spatial distribution of the corresponding active cortical area at a given time. The positions of the dipoles with respect to the cortical surface depend on the extent and geometry of the activated cortical area: superficially positioned dipoles (i.e. near to the cortical surface), such as those of the mu rhythm, correspond to more localised cortical activity, while deep-lying dipoles, like those of sleep spindles, instead represent the activity of extended cortical surfaces. Thus, dipole locations provide only an approximation of the localisation of the active brain area and the extension of the area. When comparing the results of the alpha rhythms and of the sleep spindles (Fig. 2), we should emphasise that there is no overlap of the centres of gravity of these two kinds of rhythms. This indicates that different regions of the cortex are involved in the generation of these brain rhythms. The same applies to the mu rhythm, which appeared to be generated in a different brain region compared to the alpha rhythm and sleep spindles. The superficial location of the mu rhythm dipoles (especially in MEG, Fig. 2b) suggests that the mu rhythm is generated in a relatively well-localised cortical area. Thus, the equivalent dipole model appears to be an adequate model for the estimation of mu sources. A comparison of the results obtained in the same subject based on EEG (Fig. 2a) or on MEG (Fig. 2b) data shows that the dipoles estimated using the latter occupy a more circumscribed number of voxels than those based on the former; this is particularly clear for the spatial distribution of the dipolar sources of sleep spindles and the mu rhythm. The close spatial relationship of the MEG mu rhythm dipoles to the dipoles of the N20 component of the medial nerve somatosensory evoked fields demonstrates that the mu rhythm arises from the cortex around the central sulcus.

5.1
EEG-Correlated fMRI

The advent of simultaneous EEG–fMRI acquisitions (Lemieux et al. 1997; Goldman et al. 2002; Krakow et al. 2000) has allowed the study of the haemodynamic correlates of spontaneous variations of alpha rhythm (Laufs et al. 2003; Moosmann et al. 2003), focussing on group results. In this kind of work it is important to study individual subjects, since there is a considerable variability with respect to these phenomena among normal subjects. Gonçalves et al. 2006; de Munck et al. 2007 found a negative correlation between the BOLD signal and the average power time series within the alpha band (8–12 Hz) in extensive areas of the occipital, parietal and frontal lobes, and a negative correlation was found between heartbeat intervals (reverse of heart rate) and alpha power (de Munck et al. 2007) although one subject only showed positive correlations, thus contradicting the negative BOLD alpha power cortical correlations that were found in most subjects. In small thalamic areas, the BOLD signal was positively correlated with the alpha power. Results suggest that the resting state varies among subjects, and sometimes even within one subject. As the resting state plays an important role in many fMRI experiments, the simultaneous recording of fMRI and EEG is advisable; see the chapters "Brain Rhythms", "Sleep", "EEG–fMRI in Adults with Focal Epilepsy", "EEG–fMRI in Idiopathic Generalised Epilepsy (Adults)" and "EEG–fMRI in Children with Epilepsy" for further discussions of this issue in the healthy and pathological brain.

6
Conclusions

Knowledge of the electrical and magnetic fields generated by local neuronal networks is of interest to the neuroscientist because these signals can yield relevant information about the activity modes of neuronal populations. This is particularly relevant when attempting to understand higher-order brain functions such as perception, action programming, and memory trace formation. It is becoming increasingly clear that these functions are mediated by dynamical assemblies of neurons. In this respect, knowledge of the properties of the individual neurons is not sufficient. It is necessary to understand how populations of neurons interact and undergo self-organisation processes to form dynamical assemblies. The latter constitute the functional substrate of complex brain functions. These neuronal assemblies generate patterns of dendritic currents and action potentials of course, but these patterns are usually difficult to evaluate experimentally due to the multitude of parameters and the complexity of the structures. Nevertheless, the concerted action of these assemblies can also be revealed in the LFPs that can be recorded at distance from the generators as EEG or MEG signals. However, extracting information from EEG or MEG signals about the functional state of a local neuronal network poses a number of nontrivial problems that must be solved by combining anatomical/physiological with biophysical/mathematical concepts and tools. Indeed, given a certain EEG or MEG signal, it is not possible to precisely reconstruct the behaviour of the underlying neuronal elements, since this

inverse problem does not have a unique solution. Therefore, it is necessary to assume specific models of the neuronal elements and their interactions in dynamical assemblies in order to make sense of the LFPs. This implies that it is necessary to construct models that incorporate knowledge about cellular/membrane properties with those for the local circuits, their spatial organisation and organisation patterns. Furthermore, intracranial EEG studies (such as those in patients with epilepsy being evaluated for surgery) demonstrate that a significant amount of brain activity does not appear in the EEG or MEG. This consideration, along with the fundamental limitations of the EEG/MEG inverse problem and the difficulty involved in estimating large or complex networks of generators suggest that functional imaging combined with EEG can play a significant role in improving our understanding of brain activity.

References

Achermann P, Borbély AA (1997) Low-frequency (<1 Hz) oscillations in the human sleep electroencephalogram. Neuroscience 81(1):213–22

Adrian ED, Mathews BHC (1934) The interpretation of potential waves in the cortex. J Physiol 81:440–71

Amzica F, Steriade M (1997) The K-complex: its slow (<1-Hz) rhythmicity and relation to delta waves. Neurology 49(4):952–9

Baker SN, Kilner JM, Pinches EM, Lemon RN (1999) The role of synchrony and oscillations in the motor output. Exp Brain Res 128:109–17

Berger H (1929) Über des Elekrenkephalogramm des Menschen. Arch Psychiat Nervenkr 87: 527–70

Bouyer JJ, Montaron MF, Vahnée JM, Albert MP, Rougeul A (1987) Anatomical localization of cortical beta rhythms in cat. Neuroscience 22(3):863–9

Burghoff M, Sander TH, Schnabel A, Drung D, Trahms L, Curio G, Mackert BM (2004) DC-magnetoencephalography: direct measurements in a magnetically extremely-well-shielded room. Appl Phys Lett 85:6278–80

Buser P, Rougeul-Buser A (2005) Visual attention in behaving cats: attention shifts and sustained attention episodes are accompanied by distinct electrocortical activities. Behav Brain Res 164(1):42–51

Buxhoeveden DP, Casanova MF (2002) The minicolumn hypothesis in neuroscience. Brain 125: 935–51

Buzsáki G (2006) Rhythms of the brain. Oxford University Press, Oxford

Caton R (1875) The electric currents of the brain. Br Med J 2:278

Cram JR, Kohlenberg RJ, Singer M (1977) Operant control of alpha EEG and the effects of illumination and eye closure. Psychosom Med 39(1):11–8

de Munck JC, Gonçalves SI, Huijboom L, Kuijer JP, Pouwels PJ, Heethaar RM, Lopes da Silva FH (2007) The hemodynamic response of the alpha rhythm: an EEG/fMRI study. Neuroimage 35(3):1142–51

Eckhorn R, Bauer R, Jordan W, Brosch M, Kruse W, Munk M, Reitboeck HJ (1988) Coherent oscillations: a mechanism of feature linking in the visual cortex? Multiple electrode and correlation analyses in the cat. Biol Cybern 60(2):121–30

Favorov OV, Diamond ME (1990) Demonstration of discrete place-defined columns—segregates—in the cat SI. J Comp Neurol 298(1):97–112

Freeman WJ (2005) Origin, structure, and role of background EEG activity. Part 3. Neural frame classification. Clin Neurophysiol 116(5):1118–29

Freeman WJ, van Dijk BW (1987) Spatial patterns of visual cortical fast EEG during conditioned reflex in a rhesus monkey. Brain Res 422(2):267–76

Freiwald WA, Kreiter AK, Singer W (1995) Stimulus dependent inter-columnar synchronization of single unit responses in cat area 17. Neuroreport 6:2348–52

Fuchs M, Wagner M, Kastner J (2007) Development of volume conductor and source models to localize epileptic foci. J Clin Neurophysiol 24(2):101–19

Goldman RI, Stern JM, Engel J Jr, Cohen M (2002) Simultaneous EEG and fMRI of the alpha rhythm. NeuroReport 13(18):2487–92

Gonçalves SI, de Munck JC, Pouwels PJ, Schoonhoven R, Kuijer JP, Maurits NM, Hoogduin JM, Van Someren EJ, Heethaar RM, Lopes da Silva FH (2006) Correlating the alpha rhythm to BOLD using simultaneous EEG/fMRI: inter-subject variability. Neuroimage 30(1):203–13

Gonçalves SI, de Munck JC, Verbunt JP, Bijma F, Heethaar RM, Lopes da Silva F (2003) In vivo measurement of the brain and skull resistivities using an EIT-based method and realistic models for the head. IEEE Trans Biomed Eng 50(6):754–67

Gray CM, König P, Engel AK, Singer W (1989) Oscillatory responses in cat visual cortex exhibit inter-columnar synchronization which reflects global stimulus properties. Nature 338(6213): 334–7

Hämäläinen MS, Hari R, Ilmoniemi R, Knuutila J, Lounasmaa O (1993) Magnetoencephalography. Theory, instrumentation and applications to the noninvasive study of human brain function. Rev Mod Phys 65:413–97

Hari R, Salmelin R, Mäkelä JP, Salenius S, Helle M (1997) Magnetoencephalographic cortical rhythms. Int J Psychophysiol 26(1–3):51–62

Huang TY, Cherkas PS, Rosenthal DW, Hanani M (2005) Dye coupling among satellite glial cells in mammalian dorsal root ganglia. Brain Res 1036(1–2):42–9

Hughes SW, Crunelli V (2005) Thalamic mechanisms of EEG alpha rhythms and their pathological implications. Neuroscientist 11(4):357–72

Hughes SW, Lörincz M, Cope DW, Blethyn KL, Kékesi KA, Parri HR, Juhasz G, Crunelli V (2004) Synchronized oscillations at alpha and theta frequencies in the lateral geniculate nucleus. Neuron 42:253–68

Krakow K, Allen PJ, Symms MR, Lemieux L, Josephs O, Fish DR (2000) EEG recording during fMRI experiments: image quality. Hum Brain Mapp 10:10–15

Laufs H, Kleinschmidt A, Beyerle A, Eger E, Salek-Haddadi A, Preibisch C, Krakow K (2003) EEG-correlated fMRI of human alpha activity. Neuroimage 19:1463–76

Lehtelä L, Salmelin R, Hari R (1997) Evidence for reactive magnetic 10-Hz rhythm in the human auditory cortex. Neurosci Lett 222(2):111–4

Lemieux L, Allen PJ, Franconi F, Symms MR, Fish DR (1997) Recording of EEG during fMRI experiments: patient safety. Magn Reson Med 38:943–52

Lopes da Silva F (1991) Neural mechanisms underlying brain waves: from neural membranes to networks. Electroencephalogr Clin Neurophysiol 79(2):81–93

Lopes da Silva FH (2002) Electrical potentials. In: Ramachandran VS (ed) Encyclopedia of the human brain. Elsevier, New York, pp 147–67

Lopes da Silva FH, Storm van Leeuwen W (1977) The cortical source of the alpha rhythm. Neurosci Lett 6:237–41

Lopes da Silva FH, van Rotterdam A (2005) Biophysical aspects of EEG and magnetoencephalographic generation. In: Niedermeyer E, Lopes da Silva F (eds) Electroencephalography: basic principles, clinical applications and related fields, 5th edn. Lippincott, Williams & Wilkins, New York

Lopes da Silva FH, van Rotterdam A, Storm van Leeuwen W, Tielen AM (1970) Dynamic characteristics of visual evoked potentials in the dog. II. Beta frequency selectivity in evoked potentials and background activity. Electroencephalogr Clin Neurophysiol 29(3):260–8

Lorente de Nó R (1947) Action potential of the motoneurons of the hypoglossus nucleus. J Cell Comp Physiol 29:207–87

Mainen ZF, Sejnowski TJ (1996) Influence of dendritic structure on firing patterns in model neocortical neurons. Nature 382:363–6

Manshanden I, De Munck JC, Simon NR, Lopes da Silva FH (2002) Source localization of MEG sleep spindles and the relation to sources of alpha band rhythms. Clin Neurophysiol 113(12): 1937–47

Moosmann M, Ritter P, Krastel I, Brink A, Thees S, Blankenburg F, Taskin B, Obrig H, Villringer A (2003) Correlates of alpha rhythm in functional magnetic resonance imaging and near infrared spectroscopy. Neuroimage 20:145–58

Moruzzi G, Magoun HW (1949) Brain stem reticular formation and activation of the EEG. Electroencephalogr Clin Neurophysiol 1(4):455–73

Mountcastle VB (1997) The columnar organization of the neocortex. Brain 120:701–22

Munk MH, Roelfsema PR, König P, Engel AK, Singer W (1996) Role of reticular activation in the modulation of intracortical synchronization. Science 272(5259):271–4

Murakami S, Okada Y (2006) Contributions of principal neocortical neurons to magnetoencephalography and electroencephalography signals. J Physiol 575(3):925–36

Niedermeyer E (2005) The normal EEG in the waking adult. In: Niedermeyer E, Lopes da Silva FH (eds) Electroencephalography: basic principles, clinical applications and related fields, 5th edn. Lippincott, Williams & Wilkins, New York

Nunez PL (1995) Neocortical dynamics and human EEG rhythms. Oxford University Press, New York

Okada YC, Wu J, Kyuhou S (1997) Genesis of MEG signals in a mammalian CNS structure. Electroenceph Clin Neurophysiol 103:474–85

Paskewitz DA, Orne MT (1973) Visual effects on alpha feedback training. Science 181(97): 360–3

Pfurtscheller G, Lopes da Silva FH (1999) Event-related EEG/MEG synchronization and desynchronization: basic principles. Clin Neurophysiol 110(11):1842–57

Pfurtscheller G, Stancak A Jr, Neuper C (1996) Post-movement beta synchronization. A correlate of an idling motor area? Electroencephalogr Clin Neurophysiol 98:281–93

Roopun AK, Middleton SJ, Cunningham MO, LeBeau FE, Bibbig A, Whittington MA, Traub RD (2006) A beta2-frequency (20–30 Hz) oscillation in nonsynaptic networks of somatosensory cortex. Proc Natl Acad Sci USA 103(42):15646–50

Rougeul A, Bouyer JJ, Dedet L, Debray O (1979) Fast somato-parietal rhythms during combined focal attention and immobility in baboon and squirrel monkey. Electroencephalogr Clin Neurophysiol 46(3):310–9

Rougeul-Buser A, Buser P (1997) Rhythms in the alpha band in cats and their behavioural correlates. Int J Psychophysiol 26(1–3):191–203

Rush S, Driscoll DA (1968) Current distribution in the brain from surface electrodes. Anesth Analg 47:717–23

Simon NR, Manshanden I, Lopes da Silva FH (2000) A MEG study of sleep. Brain Res 860(1–2):64–76

Speckmann E-J, Elger CE (2005) Introduction to the neurophysiological basis of EEG and DC potentials. In: Niedermeyer E, Lopes da Silva FH (eds) Electroencephalography: basic principles, clinical applications and related fields, 5th edn. Lippincott, Williams & Wilkins, New York, pp 17–29

Stennett RG (1966) Alpha amplitude and arousal: a reply to Stennett. Psychophysiology 2(4): 372–76

Steriade M (2006) Grouping of brain rhythms in corticothalamic systems. Neuroscience 137(4): 1087–106

Steriade M, Contreras D, Amzica F, Timofeev I (1996) Synchronization of fast (30–40 Hz) spontaneous oscillations in intrathalamic and thalamocortical networks. J Neurosci 16(8): 2788–808

Stuart GJ, Sakman B (1994) Active propagation of somatic action potentials into neocortical pyramidal cell dendrites. Nature 367:68–72

Surwillo WW (1967) The inverted-U relationship: a reply to Stennett. Psychophysiology 3(3):321–2

Surwillo WW (1965) The relation of amplitude of alpha rhythm to heart rate. Psychophysiology 1(3):247–52

Traub RD, Jefferys JGR, Miles R, Whittington MA, Tóth K (1994) A branching dendritic model of a rodent CA3 pyramidal neurone. J Physiol 481:79–95

Traub RD, Miles R (1991) Neuronal networks of the hippocampus. Cambridge University Press, New York

Vanhatalo S, Voipio J, Kaila K (2005) Full-band EEG (FbEEG): an emerging standard in electroencephalography. Clin Neurophysiol 116(1):1–8

Von Helmholtz HLF (2004) Some laws concerning the distribution of electric currents in volume conductors with applications to experiments on animal electricity. Proc IEEE 92(5):868–70

EEG - fMRI
Physiological Basis, Technique, and Applications
Mulert, C.; Lemieux, L. (Eds.)
2010, XXIII, 539 p. 142 illus., 102 illus. in color.,
Hardcover
ISBN: 978-3-540-87918-3