1. 'Alphas' and information processing — phenomenon vs. epiphenomenon

[E. Başar] One of the aims of our conference was to point out that alpha (10-Hz) rhythms of the brain are not simply 'noise' and that such rhythms do not only correlate with an idling state of the brain. Another aim was to discuss 10-Hz oscillatory phenomena at the neuronal level.

One of the very hot topics during the last 10 years was the question whether the alpha activity is a chaotic signal; many speakers of this conference presented relevant data with regard to this point.

As stated in the prologue, one of my main aims was to search functional correlates of different 10-Hz oscillatory potentials recorded in various parts of the brain. As we have seen, various 10-Hz phenomena can be observed — not only in the occipital areas but also distributed throughout the brain. It is not possible to describe these different alphas with unique functions — we will see and discuss whether or not we call them alpha.

My view that there are several alphas is based on a functional interpretation of various results: 10-Hz oscillatory phenomena at the neuronal level and the possibility to measure 10-Hz rhythms in several types of brains including invertebrate ganglia gives rise to the strong impression that this signal existed from the beginning of evolution. When I try to combine all these statements and findings I tentatively assume that alpha is one of the most important fundamental building blocks of function in the central nervous system.

My intention to bring some speakers here was the following: I asked them to combine ideas and conclusions with regard to this alpha conference within five or maximal 10 min. Their conclusions or ideas may turn out to be different from my conclusions or from other colleagues' conclusions but they can have correlated meanings. Okay, if there are discrepancies, then we'll have discrepancies, and we'll have to survive. I have also asked them if they want to give some speculative statement or proposition — we intend to have a brainstorming session related to the alpha discus-
sion. Professor Buser will provide important remarks. Firstly, I will call some speakers to give an exposé and I hope we will have several questions from the audience because this panel belongs to all of us.

[Hari] Okay, I have prepared three transcripts about ideas that came to my mind.

Firstly, I thought about the significance of these rhythms. What has been shown here is that there are clear correlations between alpha states and stimulus processing in the brain. However, these do not mean causal relationships. We should remember that if we observe a modified evoked response that does not mean that the brain's response to the stimulus has changed because there are several possibilities for intermingled sources for evoked responses and alpha signals and their contributions just superimpose in our recordings. And this has not been taken into account.

I also think that epiphenomena have not been ruled out — to be provocative. I would very much like to see some kind of hypothesis about how these alpha modifications take place. There we really need animal data. Robert Koch claims that 40-Hz oscillations, while somehow driving cells, change the space constants of these cells so that functionally the cell gets shorter and longer depending on the phase of these oscillations. Something like that might take place during alpha as well and could be tested in animals.

One thing that I already mentioned in my talk is that there is some confusion about terminology: some people call everything that is at the back of the head 'alpha', independently of its shape, that means: independently of its frequency content. Some call all sinusoidal 10-Hz oscillations in different parts of the brain with the same name. We should do something about this. It seems clear that many local rhythms exist, and I would urge everybody working in this field to always specify clearly both the site of origin and the frequency of the rhythm that he/she is speaking about.

One thing closely related to this terminology is that in some cases there are frequency components which are close to being harmonics. We should then find out whether they really are harmonic components; we have now seen in several presentations clear functional, and even location differences between close-by 10- and 20-Hz rhythms.

A final statement about the future: We have already heard some speculations about the single cell models to explain the alpha rhythms. We should now proceed from single cell to population dynamics to make any sense of the rhythms which are recorded with scalp EEG or with MEG. We know that at least tens of thousands, maybe hundreds of thousands of neurons have to be simultaneously active to produce these signals. Then finally, in order to proceed from phenomenology to physiology, we really need to go to sources. We have to make any effort to specify what are the sites in the brain which generate these signals we are measuring. Moreover, I think that all these reactivity measures, coherence etc. should be stated in terms of sources rather than signals.

[Buser] One of the important questions in this general discussion is that of the functional meaning (or significance) of the electrophysiological oscillations in general. The issue was just raised by Dr Basar and Hari: are rhythms simply epiphenomena or do they reveal neuronal events that directly influence information processing in the brain?

Let me start taking two extreme examples. At first, the case with rhythms in the 40 Hz band. Regarding these rhythms, the very interesting hypothesis has been recently suggested (Gray et al.), that these oscillations achieve interneuronal synchronization, thus permitting spatio–temporal coding and interareal binding, thereby playing what (in my opinion) is really a causal role in the perceptual process and not only being an epiphenomenon. A second class of rhythms are those characteristic of slow wave sleep, a state which everyone would probably recognize as being accompanied by a loss of consciousness and hence by a suppression of most of the information processing in higher brain structures. Here again, slow sleep spindles may of course be regarded as epiphenomena. But I would rather tend to consider that they directly interfere with processing, through imposing a rhythmic synchronous firing on a large neuronal ensemble. The latter condition may be hardly compatible
with the subtle and complex transfer and interchange of messages in the thalamocortical pathways or within the cortex itself, that presumably underlie conscious processes. One may thus be inclined to consider that: (1) in both conditions rhythms express neuronal processes which are of major importance to the functioning of the brain (meaning that they have a functional role); (2) synchronization may however produce distinct effects, perhaps depending on the frequency of the rhythms and/or their distribution within the brain, either facilitating a cognitive function such as perception, attention, or memory, or else impeding it, — down to a quasi-suppression of any cognitive activity. Clearly then, I am inclined to favour the ‘functional role’ hypothesis, and to reject the alternative, ‘epiphenomenological’ one, although I must confess that all proofs are not really available so far.

Let us now consider the possible meaning of other classes of waking rhythms, mainly those in the 10–20 Hz band (our concern in this meeting). We ourselves gathered some data in cats, showing that during the development of mu rhythms (at 14 Hz), responses to a given tactile stimulus were reduced with respect to controls in the thalamic nucleus ventralis posterior (where these rhythms occur together with those in the cortical somatic cortex). These data indeed suggested to us that mu rhythms may be concomitant to ‘reduced processing’. A hypothesis that meets Pfurtscheller’s idea that ‘desynchronization reflects that a population of neurons is getting ready to process’.

[Petsche] I have one brief question to Dr. Hari’s statement about phenomenon and epiphenomenon: what is the difference between phenomenon and epiphenomenon, and why do you think phenomena are good and epiphenomena are bad? In my mind there are only epiphenomena we can observe.

[Hari] I was thinking that the epiphenomenon is like smoke from the fire, and then we are not looking at the fire itself.

[Buser] Following the remarks by Dr Hari and Dr Petsche, one may even elaborate further on what can be meant by epiphenomenon vs. causal action. A good example is I think the relationship between the membrane potential of thalamic neurons and their firing pattern, as suggested by Linas, based on his in vitro studies. Guinea-pig thalamic neurons may undergo two distinct states depending on their level of polarization, one of rhythmical firing, with their transfer capabilities to the cortex being reduced or suppressed and another one, when these cells do not fire rhythmically, and can act as real thalamocortical relay cells, presumably achieving the transfer of sensory messages from the thalamus to the cortex. With this class of hypotheses, one may well believe in causality rather than in epiphenomenology.

Needless to say though that if it were on the contrary demonstrated that rhythms are but a side-product, being for instance simply due to immobility of the subject and not directly influencing central processing, the ‘epiphenomenal hypothesis’ would have to be accepted.

[E. Başar] May I also make a comment — what is an epiphenomenon? This is really a philosophical question, because according to Plato we are in a cave: we’ll never learn exactly about these waves, we’ll never learn about gravitation and so on. As Hellmuth Petsche said: an epiphenomenon may also be good.

[Rougeul-Buser] About the question of whether the 20-Hz rhythm is a harmonic of mu or not, I can add the following observation: we recently discovered that when a cat is waiting for a target to appear, it develops not only mu rhythms, but also 20 Hz. As soon as the animal is about to perform its response, the mu rhythms markedly decrease while the 20 Hz increase. So the 20 Hz can by no means simply be a harmonic of the alpha band.

[Niedermeyer] It is a difficult task to follow Professor Riitta Hari because she started the discussion so beautifully. First, I would like to talk about the question of 10 Hz vs. 20 Hz rhythms: are these true or just apparent harmonics? As to this question, I gained a lot from Pfurtscheller’s presentation because mu rhythms may present itself at the classical 10-Hz frequency or at about 20 Hz (presumed to be a true harmonic phenomenon). According to Pfurtscheller’s recent work there is evidently a somatosensory and a motor rhythm. The motor rhythm appears to be the fast one (and thus the non-classical
This is also the motor rhythm described by Jasper and Penfield (1949) in locally anesthetized patients by means of intraoperative electrocorticography. The Rolandic 10-Hz frequency — the classical mu rhythm — accordingly would be reserved for the somatosensory rhythm. In animals, both posterior alpha rhythm and mu rhythm tend to occur in somewhat faster frequencies (referring to the work of Rougeul-Buser). Keeping in mind slight variations in alphalt frequencies in animals, conclusions based on frequencies of rhythms have to be made with caution.

Secondly, I think that one of the most important items here on our agenda is the combined view of EEG and evoked potentials: where in the world do you find a group of people who jointly discuss both EEG and evoked potentials in such an integrative view. This is truly one of the most important aspects of our conference. The uniting link of EEG and EP appears to be the ‘alpha response’ as a segment of the evoked response. The alpha response, I think, requires a few comments because what you see is a double: a double potential of stunted character. It is immediately inhibited and does not proceed into an alpha rhythm — it hence remains an alpha fragment. It stays in that frequency whereas human alpha rhythm undergoes greater frequency variability.

Third, I wish to add a few comments to those three alphalt rhythms which I had the great pleasure to mention first in this series of presentations. I think that they do present distinct electroencephalographic entities. All three of them have their own psychophysiological and even clinical meaning. The Rolandic mu rhythm is the most clearly defined one. It is, as you know, a sensory-motor rhythm and nothing else. And therefore it does not impinge on other areas such as the nearby association cortex. Cognitive processes play a role both in the classical posterior alpha rhythm and also in that still quite controversial temporal rhythm. At this point, Professor Hari and I have agreed to disagree a bit in the most amicable way as to whether the ‘third rhythm’ and the ‘tau rhythm’ represent different entities.

Fourth, special problems can arise from spatial frequency analysis. Take the example of a frontal alpha and the posterior alpha rhythm. If the latter is very pronounced, it can reach the vertex area and may encroach on the electrodes, F3 and F4 — but that’s it! An apparent alpha rhythm in frontopolar leads is due to an active ear reference electrode and disappears with bipolar EEG recording technique. We must never just mix up alpha frequencies — which may be anywhere — and real organized alpha rhythm. Finally, let me ponder about the question of our future! I think, what transpires from this conference is the renewed emphasis on EEG in psychophysiology and cognitive function. This is enormously important. The neuroimaging methods are fierce competitors but I think that, in this competition, the electrophysiological methods will remain indispensable.

[**E. Başar**] Thank you very much, Prof. Niedermeyer, for these remarks. You are completely right: alpha is not the only EEG frequency related to function. As stated in the prologue, we have tried to put together with Prof. Bullock all sorts of rhythms, not only the 10-Hz rhythm. I also belong to the 40-Hz people — it is because we are in an alpha conference that we stress the functional relevance of alphas.

[Niedermeyer] I agree.

2. Synchronization and desynchronization: cortex and thalamus

[**E. Başar**] Now, Gert, we are moving to your territory, Prof. Niedermeyer already started to talk on Rolandic mu rhythm and I'm sure you are going to talk about motor functions.

[**Pfurtscheller**] As to the first statement of Prof. Riitta Hari, one question concerns the types of rhythms within the alpha band; already Riitta Hari has pointed out that we can differentiate between different types:

One group of these belongs to the occipital alpha rhythms which are blocked with visual processing. Since the time of Grey Walter it is known that not only one type but a great variety of occipital alpha rhythms exists, some of these are affected by eye opening, some are not etc.

Another group of rhythms within the alpha band are the mu rhythms. We have seen that different types of mu rhythms exist. Some are
responsive to foot movements, some only responsive to hand movement and some others only responsive to face movement. This specificity of mu rhythms was already reported by Jasper and Penfield in 1949 and recently by Arraco et al. in the EEG Journal in Baltimore.

The third group perhaps belongs to the auditory cortex; the tau and the third rhythm have been reported on and we can hypothesize that these types of rhythms may have something to do with functioning of the auditory cortex.

How can we differentiate between these different types of activities within the alpha band? We can differentiate by their reactivity, that's one important parameter, especially when considered for a special type of reactivity, namely the blocking or the suppression. The localization of this blocking is highly specific and localized to the corresponding active cortical area.

So that's one point and now another one: I'm very happy that Professor Buser — just before — mentioned the question concerning the meaning of oscillations. This question is quite an important one. We can differentiate between at least two types of oscillations of neural networks, each having different functional properties. One type of oscillation may be linking several areas, if they have similar resonance frequencies; this is a group of oscillations around 40 Hz and perhaps related to binding or sensory-motor integration — so this type of oscillation belongs to an active processing mode of the network. The other group of oscillations is related to the interruption of thalamocortical processing; one example are the spindles in sleep; another is the enhanced mu rhythm over the hand area during visual processing or foot movement. I believe these oscillations, in or close to the alpha band, have to do with a so-called resting or idling mode of networks. So synchronization can have a different meaning as stated by Prof. Buser and depends on the frequency: Oscillations around 40 Hz represent a processing mode, i.e. active neural networks, while oscillations in the alpha band can be interpreted as a resting or idling mode of neural networks.

Desynchronization of alpha band activity can be interpreted as a sign of cortical excitability, where a population of neurons — networks — is getting ready to process. Let us consider a simple, discrete, finger movement: for such a finger movement, one needs different cortical areas such as, the motor cortex, the somatosensory cortex, the premotor cortex, the supplementary motor area etc., and all these distributed cortical areas must become ready for execution of movement. A sign of getting ready may be the desynchronization of mu and central beta rhythms. One comment I would like to make, concerning the question about relationships of mu and central beta. On the one side there is high bicoherence between both rhythms. This means that the central beta can be seen as harmonics of the mu rhythm. On the other side, mu and central beta show different time courses of desynchronization, with the beta displaying a faster rebound. The same observations were made by Riitta Hari with MEG measurements. So mu and central beta are phase-linked, but they have different origins, as shown. An interpretation is that there are different neural generators, for central beta and mu rhythms, but they are harmonically related.

I would like to repeat shortly an observation made in patients: we made measurements in patients with cerebral ischemia and had one patient with a very circumscribed lesion in the basal ganglia. We found in this patient with a hemorrhage in the basal ganglia an very large enhanced amplitude mu rhythm over the ipsilateral side not reacting to any stimulation and movement. So in this case it can be assumed that the input to the motor cortex from the basal ganglia was disturbed; and as a result of this ipsilateral, a large amplitude non-reactive mu rhythm was found.

[Samson-Dollfus] I want to point out the pathologic alpha, not in the sense of the seizures that we know quite well, but in the sense of alpha synchronization. What I did not say this morning is that in microcephalic, or lissencephalic newborn infants you have alpha, and quite in the sense of alpha and no mu rhythm. This is quite abnormal and is one of the things that I want to tell.

Another thing is the alpha coma when people are unconscious: the awakening reaction is with slow waves but during coma you have alpha. You
have to differentiate this from the locked-in syndrome where people are conscious and they have an alpha with occipital-rolandic localization. If you try to stimulate them then they react with decreasing alpha and not with an increase in alpha.

I think we should like to know more about the meaning of this abnormal feature, the pathologic alpha.

[E. Başar] Thank you for your comments: especially with respect to alpha oscillations, pathology is very important.

[Niedermeyer] I am confused in relation to spindles and the making of spindles.

If you read Steriade who has done really leading work in the genesis of spindles (which was also expressed in a big joint paper in the EEG journal in 1990 with Lopes da Silva and so many other authors) spindles are generated from the reticular portion of the thalamus, from the onion shell reticular nucleus of the thalamus, so they would be a thalamogenic pattern.

I hear from Pfurtscheller that spindles are due to the interruption of thalamocortical connections. I have a personal observation in human beings with thalamic electrodes in the ventrobasal complex with spindles from the thalamus appearing independently of cortical spindles so that all this creates tremendous confusion and clarification would be helpful.

[Pfurtscheller] I would like to refer to the same literature. One is the paper from Llinas and Steriade in ‘Physiological Reviews’ and another is the article from Prof. Lopes da Silva in the EEG-Journal. The interpretation in this paper was that spindles are caused by the interruption of thalamocortical signal processing. I hope Prof. Lopes da Silva agrees with this.

[E. Başar] May I comment on this shortly? Dr Steriade was invited but could not come; we would certainly have learnt a lot from him. I have the following objection: spindles of the type just mentioned are not observed in physiological conditions similar to the alpha correlates discussed here.

[Rougeul-Buser] Let me discuss somewhat more on the role of nucleus reticularis thalami (n.R.T.). According to some authors, this nucleus plays a pivotal role in all types of cortical (and corresponding thalamic) rhythms. We recently challenged this conclusion (Rougeul and Canu), showing that n.R.T. is probably not involved in what we designate as focal rhythms, involving restricted thalamocortical channels (alpha, mu and beta rhythms). On the other hand, we were able to confirm that sleep spindle generation indeed involves n.R.T., as postulated by others before.

[E. Başar] I think with sleep spindles one has to make an exact differentiation, because sleep spindles are around 14 Hz and so on. Nembutal spindles are different, i.e. a family of spindles is recorded.

[Rougeul-Buser] As already stressed, barbiturate spindles have very little to do with any rhythm in the normal behaving animal. Moreover, we administered a variety of drugs to cats and some of them significantly altered the attentional behaviour of the animal. Those were drugs acting on the noradrenergic or the dopaminergic system. We never observed the development of spindles of the barbiturate type in any of these conditions. As far as localization is concerned, we were able to identify rhythms in human subjects, that displayed the same kind of reactivity as in animals, but did not belong to the same frequency band.

My second point is this: using arrays of intracortical electrodes in animals we were able to record a variety of rhythms, with rather accurate cytoarchitectonic locations of their site of occurrence. Let me add that the concept of a correlation between rhythms and cortical sites is not new. It was indeed developed in this country in the 35–40s by Kornmüller, working close to Oscar and Cecile Vogt, but these results (on the rabbit) were almost completely overlooked later on.

[Buser] About barbiturate spindles, I fully agree that they definitely are ‘narcotic artifacts’. They are of course much easier to study than waking rhythms in behaving animals, and they were indeed very well explored by several groups. Unfortunately they have very little to do with normal activity. One of the most popular barbiturate spindle models to explain rhythmic activity fell down when it was shown that recurrent collat-
erals were very few if any, in the relay cells of the thalamic nucleus ventralis posterior of the cat (where they were analyzed): these collaterals had been considered essential in this model.

3. Localization of ‘alphas’ in the brain — EEG and MEG

[Başar] We come to the next speaker, Sam please.

[Williamson] Since this is a very friendly group of colleagues, I would like to first propose that we give a new name to the ‘40-Hz oscillation’ that was mentioned earlier. For the sake of accuracy, we do indeed need one. I refer to the figures in the first paper by Gray and Singer showing an oscillatory phenomenon in the visual cortex that appears when two stimuli have certain spatial relationships. If you look closely, you see that the frequency was actually closer to 50 Hz. I recognize that reporting a ‘50-Hz’ signal in Europe could be embarrassing, so we may be sympathetic to their using the term ‘40 Hz’ (we in the Americas are safe from this difficulty, since our power line frequency is 60 Hz). Nevertheless, we should face the facts and realize that these signals are often not 40 Hz, but can be closer to 50 Hz. A conservative might choose to call this the ‘zeta rhythm’, since it may well be the last rhythm to be found. On the other hand, should additional rhythms be detected, we could avoid embarrassment and call them ‘zeta prime’, ‘zeta double-prime’, and so forth. But such an absurdity of using ad hoc terms should encourage us to adopt an alternative philosophy! A reasonable terminology could be based on the name for the bandwidth in which the oscillation is observed: namely the ‘gamma rhythm’. This is consistent with the basis for the terminology we use for the name of this conference: the alpha rhythm. Indeed, the term ‘gamma rhythm’ is gaining acceptance as a term to replace the so-called ‘40 Hz’ phenomenon, at least among physiologists.

One reason I am really delighted with this conference is the fact that |E. Başar| have never heard the term ‘paradoxical extremum’ used. This term refers to the situation where an extreme value for the electric potential is observed on one side of the head when you know the source — perhaps a lesion — is on the other side. The ‘paradoxical extremum’ has always bothered me. Before our group began to develop the methods of magnetic source imaging, my research focused on the physics of magnetism and superconductivity. Measurements of the magnetic field patterns of a solid material, such as a magnet or superconductor, provided insight on their dynamical processes. The physical laws that relate the measured field pattern to the source make this possible. So it was no surprise that a strong current source within the brain, if directed toward the opposite hemisphere, could produce a strong maximum in the electric potential. There is simply no reason to expect that a signal detected on one side of the head necessarily comes from a source on that side. Anyone whose interest is limited to describing the potential pattern across the scalp is characterizing the epiphenomena. This pattern may have no obvious spatial relationship to the configuration of the actual neural sources! Much important information is lost by such a ‘superficial’ view! Even for the most complicated potential pattern, mathematical solutions exist to provide a best estimate for the actual distribution of neural activity across the cerebral cortex. We need not limit our source models to just a few current dipoles.

In fact, I would like to echo Riitta Hari’s plea — we need to pay attention to the properties of cortical as well as subcortical sources. It makes sense to determine their locations, and with the best accuracy possible. Four millimeters across the central sulcus makes a big difference between identifying a rhythm as characteristic of somatosensory cortex vs. motor cortex. It would pay us well to try to do better in localizing sources, for those who have enough patience to attach more electrodes to the scalp, or would like to come to some of the centers that are being developed now for magnetic source imaging with large sensor arrays. All of us will soon have such opportunities, because these systems are too expensive for one group to run. Also, most research groups are not sufficiently large to run such systems on a full-time basis. Shared facilities make economic and scientific sense. It is reasonable — when the
experimental paradigm is likely to involve many areas of the brain — to take advantage of large arrays and the accompanying analysis routines for identifying the pattern of neural activity.

Granted, some of us who have never used a central facility may find it difficult to think of going to another lab to run a study. I will argue, however, that the inconvenience can be well justified for an important experiment. Indeed, if you can collect data simultaneously that characterizes the neural activity across the whole cerebral cortex, is it not a better-controlled condition than running a sequence of studies with fewer sensors, or electrodes, on consecutive days?

4. ‘Alphas’ and information processing — relations to psychological variables

[Williamson, continued] With that said, I come to my main point: the singular absence in our discussions of the topic of memory. This subject was mentioned in a couple of the presentations, but without emphasis. However, memory functions are incredibly important to humans! So, I wonder why we do not pay more attention to the subject. My research group recently encountered a memory function quite by accident (Lü et al., 1992), when we observed habituation of responses in the primary auditory cortex. Like those who studied this before, we were impressed by how weak the N100 responses become when tone bursts are presented at a rapid rate (e.g. Celesia, 1976; Picton et al., 1976; Hari et al., 1982).

On going to the literature, we encountered articles that used the words ‘adaptation’, ‘refractory’, or ‘habituation’. There are important distinctions between them. Electrophysiological studies of the auditory system in cats carried out by Wickelgren (1969) and Weinberger et al. (1990) at the University of California at Irvine revealed four criteria for the effect of the interstimulus interval to be attributable to a central process. The critical one is ‘dishabituation’. Dishabituation provides evidence for a central mechanism, in distinction to a peripheral effect at the sense organ. Being reminded by Näätänen and Picton (1987) that dishabituation had yet to be demonstrated for the human auditory cortex, we carried out what may well be the first demonstration of the effect in the human auditory system (Lü et al., 1992; Williamson and Lü, 1995).

I am not sure how many colleagues know what dishabituation is, but on reading the literature you come to realize that it is a simple concept. By repeating a stimulus at a fixed short interval, the N100 response becomes progressively weaker for the first half-dozen presentations and stabilizes at a lower value. The amplitude of the response for this condition decreases toward zero the shorter the interstimulus rate. If a different stimulus is presented at the moment when a standard was expected, perhaps with a different tone frequency or lower amplitude, a strong response is produced. No surprise there! The really interesting feature is the response to the following standard tone: Its amplitude is also greatly enhanced! That is called ‘dishabituation’. Wickelgren (1969) and Weinberger et al. (1990) demonstrated in cats that dishabituation is indicative of a central process — namely, a memory function.

Taking this conclusion seriously, the increase in response strength with increase in ISI can be given a simple interpretation. The observed response represents the net movement of electric charge that is required to drive the neural activation trace to full activation. If the interval between stimuli is short, the response is weak because the neural activation trace has decayed very little. But if the interval is long, the response is strong because the activation trace has decayed a great deal. The data for response strength vs. ISI show quite clearly that the hypothetical activation trace decays exponentially toward zero following the N100 response. Our studies across four normal subjects provided lifetimes that range from 0.8 to 3.4 s.

This finding would remain just a curiosity unless you take the next step. I say this to provide reinforcement of my plea that we all become more interested in behavioral studies that can complement our EEG or MEG studies. In this case, we set up a paradigm in which one of these same subjects was presented in one ear with a ‘test tone’, followed a short time later in the other ear by a ‘probe tone’. The delay between the two was randomly chosen with equal probabilities
from 1, 2, 4, 6, and 8 s in one block and 0.8, 1.5, 2.5, 3.5, and 6.3 s in another block. The frequency of the test tone was randomly selected with equal probabilities from 800, 900, and 1100 Hz. The probe was identical to the test except that it was louder or softer. The interesting feature of this paradigm is that the probe tone's intensity was randomly drawn from a list whose intensities spanned a range above and below the intensity of the test tone. However, the mean loudness of the probe tones, together with that of the test tone, was 2.9 dB (sound pressure level) lower than the loudness of the test tone. In separate studies, a different list was used whereby the mean loudness was 2.5 dB greater than that of the test tone. The difference in loudness was sufficiently small that the subject could not judge which list was used. Each subject carried out a total of about 6000 trials over the course of several weeks.

The result of this behavioral study was the clear demonstration that each subject's judgement of the test tone intensity decayed exponentially to the mean loudness of all the tones presented in the session (Fig. 1). The decay was toward greater loudness for the set of probe tones whose loudness was biased upward, and the decay was toward diminished loudness for the set that was biased downward. For neither case could the subject judge which tone set was being used. This decay toward the environmental mean is an example of the 'central tendency' — well known in psychology — whereby a person's judgement of the attributes of a particular stimulus decays over the course of a few seconds to the mean of all the

Fig. 1. Peak magnetic field strength near the scalp produced by the N100m component of the auditory evoked response from the primary auditory cortex. The curves are fits to the expression $Ae^{(t - t_o)/\tau}$, where $t_o$ represents the time when the hypothetical neural activation trace begins to decay and $\tau$ is the lifetime describing the decay.
person's recent experiences. In a sense, the decay of this short-term memory ('iconic' memory) leaves the person to rely on another memory function that registers the longer-term experience. What is remarkable about the experimental result is the fact that the decay of the subject's judgement of loudness is described by a simple exponential. Moreover, for each subject the lifetime matched the value obtained from neuro-magnetic studies of the decay of the neural activation trace of the primary auditory cortex (Li et al., 1992). The agreement was better than 0.2 s (Fig. 2). These experiments provide the first example where a 'physiological' measure predicts the duration of a memory.

I have always enjoyed looking at maps of potential and field patterns recorded across the scalp. But the more important concern should be: 'what do they mean?'. Most of us feel rather comfortable putting electrodes on the scalp and recording scalp voltages, or of positioning a cryogenic dewar and recording magnetic fields. But we do not feel comfortable running 6000 trials of a behavioral paradigm to establish a measure of performance for the individual. I suggest that more of the latter is really what we need. It will make our research much more significant for elucidating human brain functions, when we can show the connections to performance. I refer here to cognitive, sensory, and motor performance, among others. Characterizing such dynamical features of brain activity are how the EEG and MEG can be best exploited. In this way they provide an invaluable complement to other measures of brain activity, as provided by positron emission tomography and functional magnetic resonance imaging.

[Buser] All in all I perfectly sympathize with Dr Williamson's suggestion to study more thoroughly the correlation between evoked potentials and cognitive functions with respect to fine localization. In fact those using MEG are in a way much happier than we are, since they have a method with both high spatial and temporal resolution. We need to know more about localizations of the various components of the EPs. We also would need to know more about the localization of the 'spontaneous' ongoing activities. The question remains open as to what extent we can expect to do so using EEG procedures. Gert Pfurtscheller was I think very fortunate to be able to achieve such refined localization and I would enthusiastically follow him in this line, using a high number of electrodes and maybe an algorithm such as current source density computation. The general question evidently remains open, whether localization over the scalp is reliable or not. What puzzles me (and certainly many others as well), is that the largest EP components, say for instance P300, are obtained with scalp electrodes placed on the vertex (Cz, Fz or Pz), spots where we suspect that they are not localized just underneath. Having been for more than 20 years close to people who practice EEG explorations in human epileptics, I have personally remained very sceptical whenever I saw them trust the localatory possibilities of their scalp derivations.

[Niedermeyer] I think I have to say something about Professor Buser's comment because underneath the vertex lies the most posterior portion of the supplementary motor area. Secondary evoked
potentials in response to incoming signals may arise over the motor area. In sleep, this processing is exemplified by the K-complex with its maximum over the vertex. I have heard a very nice comment from Heinz-Gregor Wieser (pers. commun.) who is also quite a thinker in this field. Imagine archaic men sleeping and exposed to all the dangers of hostile tribes and wild beasts. In such a sleeper, all the incoming messages should be processed to the supplementary motor area in order to make the sleeper ready to fight or flight. This is a very teleological explanation, but it is certainly quite interesting and plausible.

Secondly I wish to comment briefly on Professor Williamson's suggestion to place greater emphasis on behaviour. Aren't we working anyway on the basis of behaviour, and its underlying mechanism?

[Williamson] We can enjoy a little argument on this subject! If you know what brain signals come between a certain time and a later time, and if you don't know what the person is doing in that interval and subsequently, what has one really learned? In other words: if we can see the ensemble of activity in the brain and not correlate it with the behavioral action afterwards, what has that told us except that the brain is amusing itself? We are doing only half of the job. The fact that most of us are not experts in behavioral measures does not give us an excuse to avoid what I consider a responsibility. It is much easier these days to form a collaboration with an expert in another discipline. Perhaps we at New York University are a little more fortunate than some, because our lab is in a 12-story building that is connected on every floor with two other buildings. Some floors house physics faculty; others, psychology faculty; and still others, physiology faculty. This makes it easier to meet colleagues in these other disciplines and strike up professional relationships. The tradition of housing each department's laboratories in separate buildings separated by large areas of green grass hinders such multi-disciplinary research!

[Niedermeyer] In the history of EEG there are nice examples of attempts to use the EEG for the evaluation of behaviour: think about the Green-Arduini response (Green and Arduini, 1954), with the arousal of an animal comes a strong hippocampal 6-Hz rhythm which, is not demonstrable in man. This rhythm is presumed to be related to learning mechanisms. This is a fine EEG correlate of behaviour. Another example is the work on EEG and conditioning — starting with Durup and Fessard (1935) and reaching a pinnacle in the mid-1950s (Gastaut, 1958). Here again we have EEG correlates of behaviour. Such observations will emerge from time to time.

5. Is precise localization of 'alphas' necessary, and how can it be achieved?

[E. Başar] Before we start with Prof. Galambos' ideas I suggest to go to another dimension and ask Prof. Petsche to tell us more about coherence. Coherence is a measure not only of 10-Hz activity but of the correlations between several parts of the brain. Furthermore, we could not discuss this topic without the ideas of Dr. Lehmann. I would appreciate questions from our younger colleagues, because they are going to carry out better research in future. So please, Hellmuth, can you make some comment on alpha.

[Petsche] I want to underline Professor Buser's words: involuntarily, when dealing with EEG we put most emphasis on localization. I am in doubt whether this is really the only information we may obtain from EEG. To substantiate this idea, I prefer to say a few words about the usefulness of coherence in general instead of repeating thoughts about the function of the alpha bands which were the topic of my previous paper.

As you probably know, I have been engaged in the field of EEG since 1949 and the first problem I came across in those days was the ambiguity of the concept of 'synchronization', since the difference of two unipolar recordings proved to be non-identical with the bipolar recording of the same electrodes. After having realized that the reason for this are phase shifts, I looked for methods to record them and devised toposcopic displays. Since then the phenomenon of synchronization has been my main field.

Looking for a useful model to study synchronization, I chose the rabbit hippocampus because of its very rhythmic theta; later on I studied
epileptic events in its cortex in order to deal with simple phenomena. However, these phenomena turned out to be very complex as soon as we had developed a method using many microelectrodes in a narrow space. Our observations on the three-dimensional spatio–temporal behaviour of these events by means of spectral analysis, current source density and coherence analysis convinced us that volume conduction, which was thought to play a major role in the clinical EEG, is almost negligible: potential gradients of up to 10 mV/mm were found on the cortex; and within the cortex, even steeper gradients were found. These results started us trying such analysis on the scalp EEG in man.

A spur to do this came from outside when in 1970, I was asked to take over a Foundation with the aim of looking for possible effects on the brain of listening to music. This Foundation was thought of as a present to honour Herbert von Karajan on his 70th birthday. Since the first experiments on myself were successful, we designed a practicable method for the analysis of EEG epochs of optimal duration. Thus we were able to study, in addition to music, a great variety of mental tasks. The results showed us that the determination of the electrical relationships between brain regions yields more insight into the reflections of thinking in the EEG than the recording of the spatial distribution of power. More and more it became evident that attentional processes underlie these findings.

In this context I should refer to the name of Livanov that Professor Shevelev mentioned. Livanov was the first to claim that an increased degree of synchronization of different brain regions means increased functional binding. He concluded this from his experiments on rabbits and went over to studying mental events in the human EEG by means of covariance analysis.

In my opinion, one advantage of coherence over power analysis is that it takes into account the structure of the underlying cortical network whose main aim seems to be a maximum possible convergence and divergence of signals parallel to the surface. This may also be the reason why the results of coherence analysis proved to be more meaningful than those from locations of power in our studies on cognition. I think, for this reason coherence analysis should be applied also to patients. I wouldn’t like to be misunderstood: I am not against using the EEG for localization; there are many effective methods for this purpose, but one ought not to forget to consider the surface of the brain as a whole and try to exhaust the possibilities of this for learning more about electrical features of behaviour.

In this respect, I would also like to add that, when discussing the meaning of the ‘alphas’ we should not forget that other frequency ranges may be significant for mentation also. We found significant task-dependent coherence changes with respect to the EEG at rest in all frequency bands. However, the number of experiments is still too small to say more about the possible functional meaning for any special cognitive act.

[Hari] One comment: I understood that you are making from these data, conclusions about activation and connection between different brain areas. If we do not go to sources, in some cases there might be difficulties. For example, if you have deep in the Sylvian fissure, a current source which is oriented like this, it would cause you very clear coherence between the frontal and temporal regions. If you do not model the sources you cannot say exactly that a certain coherence is related to activity in certain brain areas. Such conclusions would be true for radial, but not for tangential sources.

[Petsche] Well, I wouldn’t like to make this conclusion, it’s too far from me. But I would comment on your remark: I would rather put interest on the fact that at the same time we have on the other side, on T4, we have decreases of coherence: this is just what we find in simultaneous translation for instance: there we have an activation of coherences, and decrease on the other side. And if the language was more difficult to translate, we also have activations on the other side.

[Hari] I don’t doubt these data at all but I think that in many cases source modeling might be useful and make the results more accurate.

[Buser] Now, I think of a short word after Hellmuth — but after all it would be very nice to
have younger people speak and not only the old crocodiles, if I may say so.

[Lehmann] I am only a pet crocodile. I reject this sort of division between MEG and EEG strategies. You can do the same thing with the electric component of the brain field as with MEG. Unfortunately there is widespread opinion that there is a basic difference. This is completely unsubstantiated. So if you'd put your mind — somewhat less money, but more effort is needed — into EEG analysis you would very probably get the same results in terms of localization. That game is wide open. Unfortunately, the EEG world has few trained physicists — but many EEG theoreticians. The MEG world has many trained physicists who know how to put their words right. I would like to add that as to your catalogue of options — my proposal is to enrich this catalogue in terms of modes of cognition which we think is very important. And I would like to add that in our laboratory we try to be data-driven as opposed to concept-driven, and concept-driven things are for instance dipole localizations; they are based on a lot of constraints — we've seen this with Dr Williamson; you change your model a little bit and the dipole goes completely somewhere else. So then I think the name of the game is — in science — to come to conclusions about the nature of the data, to describe relationships which hold with a correlation of 1.0. The less theory, the better it will be. At the end there needs to be a lot of facts and few theories.

In fact, this is absolutely necessary because if there are very many non-agreeing theories, then — obviously, many of them must be wrong. So the implication is to be data-driven, to think about the brain as a whole, because there is only one brain and one mind at one moment in time and to consider what you can do in this respect. I side up with Dr Petsche when he says that localization isn't all, particularly if localization is one of the weakest points in our field — MEG and EEG alike, because if you know it is a bit more to the left or a bit more to the right, what have you learnt?

It might be more important to be absolutely sure about the antecedents and the consequences of one certain operation in the brain, and it might also be more important to know about the states which constrain certain operations in the brain than to know exactly where it happens. So we are thinking in terms of functional states, of induced states, of constraining states, of recursively active states; we parse them down into microstates. We think that different brain electric or magnetic field configurations must in all likeliness subserve different brain functions, and that would be a starting-point for a data-driven analysis.

[Basar] I was just going to ask you to say more to us about microstates. Before doing so maybe a short question by Prof. Samson-Dollfus.

[Samson-Dollfus] It is just a comment about what Prof. Basar and Dietrich Lehmann said. It is about the EEG and the MEG, and I know that most electroencephalographers do what they have learnt for about 50 years or more — they don't change at all. I am sure that it will be quite possible for them — instead of always using the 10–20-system — to use their 16 or 21 channels to localize and to have only 2 cm between two electrodes.

[Basar] You are right, EEG and MEG should be complementary. However, I'm inclined to say also that MEG is very important. If we have two SQUID detectors with a distance of 2 cm you may see a 10-Hz or alpha enhancement in one position and an EEG blocking in the other. We cannot observe such small-scale differences with the EEG, i.e. we have to combine EEG and MEG.

[Rougeul-Buser] I would say that — as far as localization is concerned — that in animals we recognize the same rhythms as in man, not always with the same frequency, of course, but with the same reactivity, and we are able to record with intracortical electrodes, and with very careful cytoarchitectonical studies we may localize foci of rhythms like mu, like beta, like alpha, and to recognize that electrodes which recorded the rhythm are situated in strict areas, anatomically recognized structures, and outside the limit of these areas, we cannot record these rhythms sometimes, with very, very small amplitude.

[Buser] Of course, one may, as Dr Petsche rightly pointed out, consider more holistic view-
point and somehow forget about refined localisations. This approach may be well justified, but it really implies a different viewpoint on how the cortex works and how we should explore it. A good example (in reply to Dr Lehmann) is I think the case with the 'readiness potential', or 'Bereitschaftspotential' as described by Kornhuber and Deecke (a slow potential developing just before execution of a self-paced movement). As is well known the scalp distribution of this potential is rather broad and non-localisatory of a 'motor' activity as such, suggesting a large contribution of many brain areas to the 'motor decision'. Contrastingly, when studies were performed with implanted electrodes on chronic patients explored for their epileptic focus (as we did with Lamarche at St. Anne Hospital in Paris), it turned out that the active sites were very few: they were limited to the contralateral primary area and to both supplementary motor areas. So, depending on the technical approach, the general philosophy that we may deduce from our observations may vary quite a lot.

6. 'Alphas' and information processing: microstates in the EEG and why to use psychological approaches

[E. Başar] Now, we will hear about microstates from Prof. Lehmann.

[Lehmann] I don't really know what to say now because I believe I have said it all — at least as much as I know. The concept is that different brain electromagnetic field configurations reflect different brain functions. So then — if that is acceptable then all what remains is to find out whether there are extended time epochs during which the configuration is stable. And this is the entire idea of microstates.

Of course, as described in my talk, it is quite obvious that of such short microstates of sub-second duration are embedded — not in the sense of the correlation dimension — they are embedded into, or they are part of a macrostate as defined by global brain field polarity reversal frequencies. It is well known that these frequencies are different in different macrostates such as sleep and wakefulness. One might hypothesize that the functional significance of a given microstate depends on the embedding macrostate. Then, the vocabulary, the 'dictionary' of the microstates is to be worked out for the different global functional macrostates.

There are side issues like limited capacity, conscious processing, and large capacity parallel processing — processing steps theoretically must be reflected in brain electric fields. That is experimental design. It is the task of an experimental design to sort this out and one is of course able to trace unconscious processes up to a certain point if the experiment is designed all right or one could focus on conscious processes.

At the end, as I also said, I think there is some sort of over-optimistic belief in science that it is possible to identify causes and consequences. In science, there is nothing but correlation, there is no room for cause and effect. You only can say: if I do this and if I constrain all these other factors, then that will follow. If this is repeatedly so, one says 'I've understood', as you know.

But why talk about 'understanding'? The term is simply used to refer to the set of reliable correlations between factors. A good example of this is what Mario Bunge said about speed and consciousness. Why do you say you know what speed is? You might say you've understood speed because you know that if this and this and that fact is met, then speed will be there. And why do people say consciousness cannot be understood? Because it's complicated and we do not believe that we will be able to know all the correlations which must be there to produce consciousness. In other words: science is nothing but a collection of facts between which we established convincing correlations of near 1.0.

[E. Başar] Thank you very much. Prof. Karakaş, a question?

[Karakaş] It's not a question but a comment, and I would like to follow Prof. Williamson. For the 3 days we have been listening to different technologies and approaches to the analysis of brain potentials which was really very enlightening.

If the aim in these studies is to study autonomic functions, I agree. But it was very often
cited that what is studied is cognition, attention, expectancy, or information processing, i.e., behavioral concepts with different terminology.

By side with Professor Hari, we shouldn’t do phenomenology, which means: subjective experiences. In this case, the subjective experiences of the scientist. When we use this or that paradigm and we say that this or that paradigm derives or triggers this or that cognitive state, this is phenomenology.

Neither should we do anthropomorphizing; this is also reading cognitions or feelings into something we record from the animal.

But there is a science, as Prof. Williamson has said — behavioral psychology. And in behavioral psychology we have been studying phenomena for years. Now we have a set of reliable phenomena. How have these been attained? Not through conceptual or theoretical approaches. There is technology to do it, and the limiting factor is to start with operational definitions. And to objectify the data, which means: you have to repeat it in different laboratories, it has to be repeated by different scientists, statistical manipulations have to be done, cross validations have to be done, and after many experiments some phenomena stand out. Now if we are studying cognition we shouldn’t rely on our phenomenology but we should start with these reliable phenomena which have been tested. If you want to understand a rose, you start with a rose.

[E. Başar] Thank you very much for these comments.

[Stam] A short question to Professor Lehmann; so I go back to the microstates. Have you any data on the correlation dimension as it changes or doesn’t change with the microstates?

I think it is an interesting subject if you assume that the dimension might be a way to characterize globally a brain state particularly if you use spatial embedding (about which you have published a paper)?

You said the microstates are characterized by certain constant factors if you use the amplitude of the fields to characterize them but it might be an interesting question to know whether each microstate corresponds either to different attractors which succeed in time or that we have one attractor which covers all of them.

[Lehmann] First of all, well we haven’t done it, you’re right it might be interesting. If you look at the entire field and compute, following Ruelle, Eckmann and Dvorak, the global correlation dimension using the number of electrodes as embedding dimension, then there is only one number which can be smaller or bigger. Of course this knowledge will contribute to our characterizations, but I don’t think it’s overly promising. We have looked at this global correlation dimension in terms of brain electric macrostates, analyzing longer epochs in the many-seconds-range, and that seems to describe a new aspect which adds to the spectral values — as you know there is no very close correspondence — but what you propose directly we haven’t done yet.

[Narici] I would like to make a comment—question about what Sam Williamson said before. I understood and I agree with his sponsoring of event-related EEG or MEG in cognitive studies: as you want to study the rhythms you want to study event-related EEG — am I correct, very schematically? I think this is of course perfectly correct.

However, there should also be an effort in a parallel study involving a very simple experiment. I’m trying to explain now: with an event-related MEG you can classify, for example, different kinds of rhythms. That’s what I think has been the main topic of this conference — functionally classifying rhythms. Some support for understanding and modeling this different kind of EEG can come from a simpler experiment that has to be linked with the event-related EEG: I’m talking about evoked EEG, like one single stimulus or very simple stimuli performed in a specific way. The reason why I am saying this is as follows: if you manage to link the same clusters that you find with event-related EEG with some clusters that you find with simpler experiments you may have a much stronger tool for modeling. At least you can understand better the interconnection with the stimulation and you may have more robust indicators and probably more robust classifiers even for pathology.
7. Neural substrates of alpha phenomena

[E. Başar] Thank you. After this comment we will start with Professor Galambos.

[Galambos] Let me briefly summarize and extend some points made in my formal paper. My colleagues and I are interested in describing the physiological events responsible for the EEG, the evoked potentials, and the standing DC potentials recordable on and in the cortex (this morning I did not mention the cortical axon spikes which were presumed to produce the EEG once, but are no longer). Our experiments address the question whether current concepts of the mechanisms involved need to be revised. We believe the new rat preparation demonstrates that the electroretinogram (ERG) and the cortical EP display several functional similarities which, in the case of the retina can be traced to the properties of the glial compartment. Others have already demonstrated that in the retina it is the Müller cell transport of potassium ion that initiates the current flow recorded as the ERG outside of the eye; our experimental question is whether in the cortex, where the corresponding glial compartment is composed of astrocytes interconnected by gap junctions, the current flows detected at the scalp surface are similarly initiated by glial transport of the potassium ions down-loaded into the extracellular space during synaptic activities. This is not a new idea, of course; what is new is that the rat preparation allows the direct comparison of the retinal event, where there is little question the glial compartment is directly involved, with the cortex, where one can cite only meager and indirect evidence that the glial compartment may be involved. A figure in my paper presents the idea that the retina and cortex are twins anatomically, a concept readily accepted because it is firmly based on embryological facts; whether cortex and retina are similarly twins in the way they generate their slow waves is not a new idea, but it may now be open to experimental examination. The data should tell us whether there is merit to this different way of conceptualizing the mechanism of cortical electogenesis.

[Buser] Here we come again to grips with the relationship between spikes or synaptic potentials (those whose neuronal substrate is definitely well known) and the slow potentials whose substrate is — as far as I know — still ill identified (I wish I’d be wrong!). There are very interesting points in these remarks, including the mention that you briefly made on the possibility that glial cells and ‘extracellular’ ion concentrations may play a major role in finally establishing the so far missing link between spikes and slow phenomena. Thank you for raising this important issue. The analogy with the retina and — I presume — with the electroretinogram is of course a bit risky but may be very interesting and fruitful to force people to think in a different system, introducing as you said extraneuronal ion fluxes as possible causes of some of the observed phenomena.

[E. Başar] We are running out of time... just a short question from Prof. Petsche, then I need one or 2 min, then we will hear Prof. Buser’s statements.

[Petsche] It is not a short question, Erol, it is a short comment on what Prof. Galambos said and what I can only underline. When I was at UCLA in the 1960s to work on the hippocampus, I learned to introduce a microelectrode whilst simultaneously looking at the CRD-screen and listening to a loudspeaker. When doing so, listening turned out to be a better monitor than watching the screen; the steady sh-sh-sh of the theta rhythm was heard throughout the hippocampus even if spikes were relatively seldom encountered. This means, something must go on without becoming evident in spike discharges. This made me believe that the main information would not be transmitted by spikes but could perhaps take place in the fibre feltwork. The cellular discharges may have the function of long-distance calls as compared with the events in the local telephone network that serves the regional communication.

[E. Başar] Thank you very much. With respect to the comments of what we do see and we do not see in evoked potentials or in EEG, I’m going to show you a painting from my mother’s collection: we always asked the question: who is this lady? where is she coming from? where was she born? what she was doing? I guess there are several
situations in the life — you don’t know the name of the lady and the origin but you’re living with her. This is somewhat similar to the question of sources of EEG, evoked potentials and MEG. And also this lady is somewhat an epiphemomenon.

8. A critical review of the comments

**[E. Başar, continued]** I think we have learnt a lot about 10-Hz oscillations and 40-Hz oscillations and their correlations with cognitive behaviour. May I ask Prof. Buser to give a number of conclusions or statements? Afterwards, I would appreciate questions from young colleagues.

**[Buser]** It occurred to me that several points were raised during our meeting and during the final discussion. Let me put them as questions, rather than as final statements.

1. Where is the human alpha rhythm localized? There is indeed a strong need to elaborate more on the scalp distribution of the alpha, as identified through its fundamental properties of being suppressed by eye opening and favoured by either eye closure or fixation on a Ganzfeld, with the possible use of more sophisticated techniques such as current source density measurements. How many types of alpha rhythms are there? This point was particularly raised by Lopes da Silva and Pfurtscheller, among others.

2. Functional diversity of rhythms in the alpha band. If we broaden our scope, we come to the question of rhythms in the same overall frequency band, but distinct by their localization and also, possibly, their reactivity pattern. The traditional mu rhythms as already described by older authors (Dr Niedermeyer rightly quoted Gastaut) is part of the story. Other rhythms in the same band with distinct reactive patterns and localization may also be taken into consideration.

3. Functional meaning of the alpha band rhythms. Several questions were raised during the meeting, related to the ‘functional meaning’ of rhythms in the alpha band and in many other frequency bands as well. I shall not come back to this point which has been discussed at large now and there in the presentations and in the above discussion.

4. How about the thalamus? Not much has been said on that part of the brain. I would just like to stress the fact that we should, I think, emphasize the role of the thalamocortical channels in information processing, rather than just consider the cortex as an isolated entity. To speculate, I am inclined to consider that a better knowledge of the neuronal operations going on in the thalamic networks will probably provide a key to a better understanding of a variety of cognitive and higher level brain functions.

5. About non-linearity and deterministic chaos: I regret that we were so discreet on this very important question in our final discussion.

**[E. Başar]** I want to thank Prof. Buser who has summarized the most important landmarks of this conference. Please, Dr Brandt, you were raising a question.

**[Brandt]** I want to add to the last point that was raised about the non-linear approach and which has come up from time to time at a meeting: the point I want to make as an engineer is that one does not take a screwdriver to a nail; one does not take a hammer to a screw. When one looks at these devices you figure out a model and then you figure out a tool to use. When we think about these questions we have to really think about whether we’re dealing with a non-linear phenomenon, a linear phenomenon or some combination.

There have been a number of talks and a couple of posters that have dealt with these issues. Lopes da Silva talked about how on the cellular level a cell can behave with a given model in which the inputs and in which the parameters can change but the model remains the same. But that leads to very different kinds of behaviours. Taking that into account one has to remember that one has to use the right kinds of tools.

I will give you an example of this: there has been a lot of talk about coherence, measures of coherence between different regions of the brain.
Now, I'm not going to say that this is wrong but what I will say is that since coherence is a measure of the linear relationship between two locations on the scalp — relationship in the frequency domain — one can get a very low coherence and yet still have a significant relationship between these two regions.

Another point is: we've heard a lot of talk about Fourier analysis. We all know that the EEGer does not do Fourier analysis — you don't decompose a clinical record into sines and cosines, I hope. But you look for peaks, do some counting and like that — that's more akin to the tool period-amplitude analysis. So I just wanted to mention that as engineers we need suitable tools — we have to think about the right kinds of tools which fit a model, a hypothesis.

9. There may be several 'alphas' — from idling to cognition

[E. Başar] Thank you very much. From the cognitive science, I will ask Dr Polich to make some comments. What can we learn from the 10-Hz activity and P300 and from the combination — what is your feeling about it?

[Polich] Something that has not been discussed very much at this meeting is the relative success that ERP research has obtained using patients with lesions to assess different parts of the cortex: one can determine reasonably well where a cognitive component may originate if you have a lesion in a specific location and the size of the potential is diminished. Knight (1990) has demonstrated quite elegantly that the parieto–temporal junction contributes to P300 generation using this technique. Shibasaki's group (Ikeda et al., 1994) has published a paper recently on the CNV with the same sort of techniques. The interesting thing for me is that this sort of approach can be applied for EEG–ERP associations (we've heard a little bit about some lesion effects on the EEG). Thus, it may be possible to isolate different aspects of the EEG and ERPs by using lesions to affect either EEG or ERPs or both. For example, if a particular background EEG band could be diminished that would affect P300 measures, the association between the background EEG and the ERP would be illuminated.

[E. Başar] Some more questions from the audience?

[von Stein] If you talk about alpha, at least for me the main interest in coming to this conference was the discrepancy between those who say that alpha blocking is the correlate of cerebral processing and those who say that alpha synchronization is correlated to processing. I am very happy because this conference indeed revealed a kind of solution to the problem, namely that there are different types of alpha.

Many contributions in the last few days reported about discriminations between two 'alphas' (Klimesch, Lopes da Silva, Lutzenberger, Pritchard). And some of the results even nicely fit together (e.g. there is the Lopes da Silva distinction between linear alpha and the non-linear alpha). Yesterday he mentioned that there is some tendency for the non-linear alpha to be found in the higher frequency ranges, and we heard from Klimesch that his behavioural studies revealed the higher alpha as the 'cognitive alpha'. Therefore it seems probable that both, the non-linear alpha and the higher alpha, reflect a new phenomenon, a cognitive alpha. Whereas the classical alpha reflects a resting state or a state of expectancy, and is blocked during cortical processing, this second type of alpha might reflect a cerebral activation, analogous to the induced oscillations in other frequency ranges which have been described e.g. during visual processing. This alpha would therefore be induced by stimulation. In this way during processing we would find both, alpha block and alpha synchronization, each concerning a different 'alpha'. Further distinctions of alpha subtypes obtained with dimensionality analysis (Lutzenberger, Pritchard) and phase resetting analysis (Brandt) may hint at the same point and should be correlated. Two other distinctions, between alpha as a resting phenomenon (idling) and as an expectancy/preparatory phase (Buser) to me do not contradict, both reflecting the same alpha: the network is just there, ready, resting, and not processing input.

Thus, distinguishing two subtypes of alpha is a
way to solve the discrepancy between the different views, the blocking and the synchronization view of processing.

[E. Başar] Would you also agree with the term 'alphas' or 'alpha processes' — how does it sound?

[von Stein] Yes, I definitely would agree; with 'alpha processes' thereby intending the second alpha, the non-linear alpha. As Lopes da Silva has demonstrated, alpha might be either correlated to oscillations in the cortical network (non-linear alpha), or to the activity of thalamocortical loops (linear alpha). This shows that there is a different functional meaning in the different alphas. Resting alpha may mean activity of the thalamocortical loop, a thalamocortically driven synchronization signifying the system's readiness for input to enter it. If input then does enter the system, this driven oscillation is replaced by the intrinsic oscillation of the cortical network, an oscillation reflecting the specific network properties plus the stimulus properties. The dynamics of these processes therefore determine whether we measure oscillations in certain frequencies or not; like in every frequency range they also might occur in the alpha range. According to the complexity of cortical networks these dynamics are expected to be non-linear. These induced oscillations or dynamics is what I would refer to with the term 'alpha processes'.

[E. Başar] Thank you very much. Dietrich, maybe a short comment?

[Lehmann] I would like to comment on what Dr Brandt said about the tools. The point is well taken, we should always use the most adequate tool. However, since that what we want to examine is not exactly known, often we don't know yet which tools are most adequate.

I think in research at the end there is interpretation — of course, everybody wants to know what his results mean. And I think our goal should be to use completely different, independent tools and come up with results that lead to an identical interpretation. I think that would be an ideal situation.

As an example: as you know, if a thing is non-linear, you might as well use a linear analysis, and sort-of decipher the main lines, and a non-linear process will survive a linear analysis to quite an extent, if it is strong enough. Thus, converging evidence originating from different approaches is the most valuable result. And there is something to be seen for instance in the convergence of PET studies and MEG studies, I think. Unfortunately, nothing much is being done in the EEG world in parallel.

[E. Başar] Thank you very much. I think that we had a long and important discussion session and I would suggest, if you agree, to close the conference.

References


