A Theoretical Approach to the Late Components of the Event-Related Brain Potential

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Introductory examples of late components

In the mid seventies, Brigitte Rockstroh and myself studied avoidance learning in human subjects. We presumed that brain potentials might be a better predictor of resistance to extinction than, for example, autonomic responses. In one experiment, subjects could escape a highly aversive noise by pressing a button fast enough. As it happens in experimental laboratories, a loose connection in the wire which was attached to the button, only randomly transmitted the subject's response to the computer. Consequently the subject experienced a sudden unexpected change in response contingency, which, of course, we completely denied when reported to us. The complaint about the frequent and unpredictable punishment was, at first, answered only with a request to follow adequately the instruction. When we examined the subject's EEG responses we detected a marked negative wave following many of the single motor responses. This was unusual as generally a positive wave accompanies the termination of an overt response. Systematic studies soon revealed that a pronounced negativity develops, whenever expectations of relevant predictions are not fulfilled, whenever there is a need for ongoing processing (Rockstroh et al. 1979; Elbert et al. 1982; Elbert 1986). Controlled experiments soon revealed that such slow negative variations of the EEG-baseline had little to do with the physical properties of the stimulus as such, but were determined by the psychological context the stimuli were put in. Kutas and Hillyard (1980, 1984) extracted a similar finding from an elegant series

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of experiments: word by word they presented sentences to the subject which were either semantically correct or incongruent: "He drank coffee with milk and shoes". When a subject processes such a sentence, word by word, each one will cause a small deflection of the EEG amplifier's needle towards negativity, quickly followed by a positive return. Not so the one quoted last! In our example, the word "shoes" will trigger a huge negativity (Fig. 1, dashed line). The appearance of this brain wave has nothing to do with the physical aspects of the presented words, but depends exclusively on the context the word has been put in. If, for instance, the sentence had been "She put on her high heeled shoes", the very same physical stimulus "shoes" would have triggered a brain wave opposite in polarity, with smaller amplitude, and longer latency (Fig. 1, solid line). And also, the scalp distribution of both waves would be different. This demonstrates that one and the same pattern on the retina may activate completely different neural generators, depending on the context the stimulus is put in. On the other hand, exactly the same EEG pattern can be evoked irrespective of whether the word is presented visually or acoustically. It is not trivial that some of the brain activity accompanying such high-level

Fig. 1. The EEG averaged over three subjects and a number of presentations of sentences is displayed while subjects process different categories of sentences. The solid line results when the sentences are congruent. A semantically incongruent ending evokes a slow negativity (*dashed line*) while a physical deviation of the stimulus, in this case a change in the letter size, produces a enlarged positivity (P560, *dotted line*). (Extracted from Kutas and Hillyard 1980, with permission)

processing can be recorded from the scalp. Only synchronized neural mass activity generates such large amplitude waves. Information processing should require distinct patterns of neural activity rather than many neurons doing the same thing at the same time.

A physically incongruent stimulus, like in "She put on her high heeled shoes" would have produced even another wave. Figure 1 includes several of the components comprising the event-related brain potentials. The N400 is a negative wave, peaking approximately 400 ms after the stimulus, the P560 is a positive wave with 560 ms latency.

Often, the cognitive operations triggering these waves may occur at other latencies than e.g. at 560 ms. It would not, however, be very convenient to give two different names to very similar neural processes. If comparable steps in the information processing trigger brain waves with similar scalp distributions we will give it the same name even if the latency is different.¹

The concept of a component

Almost any external or internal event may produce a change in the flow of oscillations of the EEG. Patterns which are reproduced with repeated presentation of an event are commonly referred to as '*event-related*' potentials. The term evoked potential is virtually synonymous, but this latter tends to be applied only when the event has little psychological impact, or if relatively short-latency fluctuations are considered. Averaging across repeated trials is commonly assumed to provide a reasonable approximation to the waveform.

Most waveforms are composites of contributions from various functionally separable cerebral processes. It is commonly postulated that the event-related potential results from a sequence of overlapping activities of different assemblies of nerve cells, each such activity representing a component.

The earliest electrical activity a stimulus evokes is the train of exogenous components. It is supposed to reflect the input and transmission of information along the afferent sensory pathway to the primary

¹ The reason we often refer to a distinct positive shift as a P300 or simply P3 and not e.g. P560 has to do more with historical development than with its most common latency. If the potential is filtered using a short time constant, the peak latency will be artificially shortened.

projection areas. A different intensity of the tone, a different pitch or location in space of the evoking sound will change these waveforms, but not a different meaning of the signal, not the context the stimulus is put in. After 100 ms the waveforms of the various event-related potentials to different stimuli become more and more alike, even regardless of the modality of the eliciting stimulus. This suggests that these later or slower components are generated by neuronal assemblies that perform operations on the incoming information such as memory or attention-related operations. Such operations are flexible in the sense that they depend on context, task, instructions and so forth; in other words on *internal representations*, even as to whether they are called for or not. Accordingly, the late components are regarded as endogenous.

In the following I will briefly review the most prominent endogenous components, particularly those with relatively long latencies and discuss their relation to psychological processes. Then I will turn to the neural generation of these electrical waves with sources mostly in cerebral cortex and outline a possible theory underlying the generation of these slow cortical potentials.

A brief tour through the zoo of endogenous components:

Fig. 2 represents a schematic overview of those late components which have been investigated most intensely. The N1 or N100-complex refers to a negative half wave with maximal amplitudes over fronto-central regions. Part of it is generated in auditory cortex from where it projects to the frontal scalp. The N1 is a mixture still, of both, exogenous and endogenous subcomponents. One part of it is related to orienting and the arousing aspects of a stimulus, thus it varies with the physical intensity but also with the novelty of the stimulus. This aspect of the N1, the so-called vertex potential or N1/P2 is widespread and seems to be triggered via an unspecific reticular activation. Another subcomponent is more closely linked to selective attention. A subject can suppress responses to incoming stimuli while attending to others, but only if the two stimulus categories can be discriminated on the basis of their physical properties. When hearing a mixture of voices, we can listen to one speaker, and process information from just this one

Fig. 2. Schematic categorization of endogenous components of the event-related potential in response to a stimulus (left) and prior to a voluntary response (right)

speaker due to the different frequency characteristics of every voice. Probably, the enhancement of N1-amplitude to stimuli in the attended channel does not originate in an enhanced gain of the attended pathway, but results from an independently generated slow negative shift generated in the secondary sensory areas of the cortex, and superimposed on the faster evoked waveform (Hillyard 1981).

When infrequent, physically deviant stimuli are randomly embedded in a train of standard stimuli, a frontal negative wave is elicited, its scalp distribution being modality-specific. This component which reaches its maximal deflection after 200 ms has been termed N2a or mismatch negativity. (MMN). Unlike the N1-enhancement, the wave is insensitive to the direction of attention: N2a can be recorded whether or not the subject is attempting to detect the deviant stimuli and whether or not that detection is successful. That is to say, the underlying discrimination is a pre-attentive response. Its amplitude increases with decreasing probability of the deviant stimulus. For acoustic stimuli the MMN is generated in the primary auditory cortex, having a dipole moment such that it projects to the fronto-central scalp surface. (A recent theoretical account for the MMN can be found in Näätänen 1990).

The MMN characteristics are contrasted by the N4-properties. After hearing "coffee with milk and ..." a subject probably expects the word sugar. When confronted with "shoes", one might think what's wrong? Is the sentence not yet finished, is the coffee consumed with shoe-fly-pie". All kinds of weird hypotheses are screened in rapid succession with the hope to resolve the ambiguity. Only, when presented in a certain context, the word 'shoe' deserves a second thought. Semantic incongruity triggers the N4, but not physical incongruity. Words are not essential, a sequence of cartoons, for example, can elicit a N4 as well. And the N4 obviously is a member of a family of surface-negative waves, as indicated by the introductory example. When a subject perceives a loss of contingency or control the negativity may be quite long-lasting. The post-processing of the stimulus includes prevailing readiness to accept further information or to generate hypotheses which might resolve the incongruity or ambiguity (Elbert et al. 1982; Birbaumer et al. 1986).

If, in the above sentence the word had been 'sugar', - full-stop - the sentence would have been complete. A subject could have closed that part of information processing. And a positive brain wave, a P3 would appear. More precisely, this component would be a P3b.

Characteristics of the P3b are its positive maximum in the latency range between 280 and 600 ms and its posterior scalp distribution. Its amplitude is inversely related to stimulus probability and also to the subjective outcome probability. The more relevant the task, the higher is the P3 amplitude, thereby task relevance and subjective probability affect P3 in a multiplicative manner. The amplitude is also sensitive to the interval between the events, being inversely proportional to the decay of the representation established by preceding occurrences of the eliciting events. As the P3-amplitude is sensitive to the information value of the stimulus and also related to recall, it is tempting to conclude that "the P3 is a correlate of processes that are invoked whenever there is a need to revise the organism's model of the context. We are assuming that the larger the amplitude of the P3, the larger the change in the model". (Donchin and Coles 1988).²

A further example illustrates most clearly that the physical stimulus as such is not important to evoke a P3 but certain steps related to the information processing: Assume you have just purchased an old fashioned Coocoo-Clock from the Black Forest for quite some money

² Available evidence suggests that P3 latency is related to categorization time but unrelated to response execution.

which goes "Tic - tac - tic - tac - tic - - tic - tac - tic ...". Ooops, a tac was missing. A P3b can be recorded each time you are counting such an omitted stimulus.

When the cocoo appears, you are likely to respond with yet another wave, a N2b/P3a followed by a negative afterwave with an amplitude the higher the more crazy you think that these Black Forest people are. Except for its latency around 200 to 300 ms, the N2b/P3a bears close resemblance with the vertex potential. Thus this component also predominates centrally to fronto-centrally. It depends on the attentional set and is sensitive to stimulus novelty. Consequently, it has been associated with a second stage in orienting.

It is interesting to note that we perceive events which arouse a lasting negative brain wave often as positive, they grasp our interest, evoke our curiosity. As sensation seekers we sometimes even look for events which can make our brain potentials more negative.

Slow Cortical Potentials

This leads us to the slow cortical potentials, in particular to the slow negative variations of the EEG-baseline like the Contingent Negative Variation (CNV) and the Bereitschaftspotential (BP) which both are related more to action and output than to input of information (Fig. 2). Although behavior is a continuous process, it can be decomposed into many subunits. Larger subunits are known as 'actions', of which there are many different types. Subunits which uniformly reappear within behavioral sequences are called reactions, with 're'-action indicating that the behavior is elicited as a response to some event. However, reactions can also be emitted without direct relationship to an external event. If the source of action lies within the reacting individual, then the reaction is known as 'voluntary response'.

The onset of a voluntary movement is typically preceded by a ramp-like surface negative shift starting about a second or so prior to the first detectable EMG-response.

Discovered in the sixties by Deecke and Kornhuber, this negative potential became known as the Bereitschaftspotential or readiness potential, referring to the activity of getting ready or becoming prepared for a voluntary response. Psychological parameters largely affect

characteristics of the BP. The complexity of the movement enhances BP-amplitude over the motor strip. Visual attention, like spatio-visual tracking will generate negativity over occipital regions, learning and complex task instructions will activate frontal areas (Rockstroh et al. 1989).

The response as such is not important. By means of EEG auto-correlation it has been uncovered that a BP even develops during intended but not executed movements (Keidel 1983).

In a series of elegant experiments Libet (1985, 1982) has asked subjects as to when they became aware of their intention to act. He demonstrated that the increase in scalp-recorded negativity prior to a self-initiated motor response precedes the subjective awareness of the intention to act. The subject will not become aware of his/her will until a distinct amplitude level is reached. Obviously, a certain *threshold* in negativity must be reached before the response will be executed.

The BP is just one representative of a family of slow negativities. Often external rather than internal processes time the response execution. The term contingent negative variation (CNV) refers to those negative shifts which are elicited by a warning signal (S1), indicating to a subject that a certain response is required a few seconds later, with presentation of a second stimulus (S2). The amplitude varies between -10 and $-20 \mu\text{V}$, reaching extremes of more than $50 \mu\text{V}$. It increases with increasing probability of S2-occurrence, with the aver-siveness of the expected event (S2) or with the high incentive value of the response. It is also higher when a fast response is required, but decreases with task complexity. It is reduced under CNS-depressants. The scalp distribution, though widespread, indicates maximal excitation in brain regions which will perform the anticipated task.

Selfregulation of slow cortical potentials

We have investigated the modifiability of slow cortical potential in a series of studies, and have attempted to determine its behavioural consequences (Elbert 1978, 1986; Elbert et al. 1980, 1984; Rockstroh et al. 1989).

In these studies the slow cortical potentials fed back to the subject, in other words the subject received information about his own potentials which he or she was about to generate. Subjects were asked to generate increased negativity, or to suppress a shift in the EEG-baseline for intervals of six to eight seconds. Healthy human subjects can quickly learn to control their potentials within two training sessions. They even can learn to regulate the shift in an area-specific manner: After the training subjects are confronted with various task situations. For instance, increased negativity shortens the reaction time and enhances solving of arithmetic problems. In one study subjects held a button in each hand and were asked to press one of the buttons after they had shifted their slow negative potential either to the left or to the right hemisphere. Subjects were instructed to respond with the hand that spontaneously occurred to them. The right hand was preferred more often on trials with increased left-central negativity (63% r.hand vs. 33% l.hand), whereas the choice of hand was equally distributed on trials with pronounced right-hemispheric negativity (Rockstroh et al. 1990); (naturally occurring rate in right handers: 42 vs. 58%). For many tasks, however, there seems to be an optimal level of negativity, sometimes within strict limits. This is best exemplified for signal-detection performance. In one of our experimental arrangements minimal changes in the configuration of a visual stimulus had to be detected. The relationship between slow potential shift and signal detection performance followed an inverted u-shaped function: Subjects detected most signals correctly when they also displayed small negativities. Performance decreased with positive and also with large negative shifts (Elbert 1991).

Closer examination showed that excessive negativities are accompanied by increasing false alarms. Obviously, the negative shift can be too large. Zero or positive shifts, on the other hand, decrease the number of detected signals. This and similar other relationships between behavioural variables and slow potentials led us to the assumption that negativity reflects the degree of excitability of neuronal networks (Elbert 1987; Elbert and Rockstroh 1987). If the threshold for excitation is too high, the incoming signal will be missed. If, on the other hand the networks operate close to firing threshold small fluctuations in the input may ignite the cell assembly leading to false alarms.

Slow surface negativity reflects cortical excitability

Neurophysiological evidence complements the view that slow surface negativity reflects cortical excitability. Depth recordings as well as quantitative considerations about volume conduction show that these slow potentials are primarily cortical in origin. We have set up a spherical model of the head with homogeneous layers, modelling scalp, bone, cerebrospinal fluid and the brain (Lutzenberger et al. 1987; Braun et al. 1991; Elbert 1991).³ The computations show that the cross section through an active structure is a most important factor in determining the amplitude on the scalp. Closed fields will not result in surface potentials, and polarizations of gyri opposite in space will partially cancel each other. Possible sizes for planar areas of polarization are small in subcortical structures, hence larger potentials ($>10 \mu\text{V}$, such as P3, CNV) must have their electrical sources distributed mainly ($>80\%$) in extended cortical regions. Subcortical activity may be superimposed but can account for only a small fraction of the potential amplitude. Of course, the generator, triggering synchronous and widespread cortical activity might be located anywhere. The statement that the sources of large-amplitude potentials reside in cortical layers receives support from measurements with electrode arrays in and across the cortex (Mitzdorf 1985; Gemba et al. 1990). Recently electrical activity has been mapped through voltage-sensitive dyes. This method (summarized by Bonhoeffer, this volume) bridges the gap between recordings on a macroscopic and on a microscopic level by staining a section of neuronal tissue with a fluorescent dye. The fluorescence varies with the membrane potential to which the dye attaches and thus the potential can be recorded through optical observation. The results presented by Arieli (this volume) suggest that the macroscopically measured potentials largely result from EPSPs in the den-

³ The following example may demonstrate the conclusion: An active planar area, small in size, with a diameter of 1 cm contributes about 1/25 of an extended layer 10 cm in diameter. Subcortical structures are not only small in size, but also more distant to the surface, diminishing their contributions to surface potentials another 4 to 7 times. Even if a large portion of a subcortical structure is synchronously active and even if we assume that active elements extending over 3 cm^2 have the same orientation in space, their contribution to scalp potentials would still amount to only 1% of that generated by extended cortical sources. Thus an intracranial polarized layer of 1 mV might produce a scalp potential of $200 \mu\text{V}$ when extensive in the cortex, but only $2 \mu\text{V}$ when limited in size and in deeper structures (Elbert 1991)

dritic trees of the cortex. Stellate cells do not produce open extracellular far-fields. What we see on the scalp or the surface of the cortex is primarily the summation of post-synaptic potentials at pyramidal cells (see also Mitzdorf 1985, and this volume). Consequently, a surface-negative potential signifies depolarization of the dendritic tree in vast networks of pyramidal cells and hence indicates an increment in excitability of the underlying cortical tissue.

This suggests the apical dendritic tree as a candidate for the regulation of cortical excitability thresholds. Depolarisation of the apical dendrites causes an efflux of negative charges into extracellular space and back into the neuron in the deeper layers. This results in a polarization of the cortex with the negative pole near the surface. A lowering of thresholds for cortical excitability will hence result in an increase in surface negativity, while a positive wave is generated when thresholds are set high, that means, a positive wave - like the P3 - corresponds to an interruption of ongoing activity, to a dysfacilitation of widespread neural activity.

There is also experimental evidence from macroscopic recordings supporting these statements. For instance, drugs which lower cortical excitability lower, in direct proportion, the slow surface negativity. While various anticonvulsant drugs may exhibit quite different effects on earlier components of visual and auditory evoked potentials, as well as on spontaneous EEG-activity (Rockstroh et al. 1987, 1991) they all lower CNV-amplitude and also the negative DC-shifts obtained during hyperventilation (von Bülow et al. 1989; Rockstroh 1991). Certain stimulant drugs may enhance the CNV although there have been several paradoxical results (for a review see McCallum 1988).

Understanding the link between neurophysiology and psychophysiology of slow potentials thus means understanding the regulation of excitability in neuronal networks.

Valentin Braitenberg, Günther Palm and Almut Schüz developed the important bases and ideas regarding this regulation. The following theoretical outline relies on their thoughts, many of them expressed in discussions and personal communications.

The concept of cell assemblies and aspects of memory storage

The concept of cell assemblies (Hebb 1949) has become fundamental to models concerned with the functioning of the brain. Hebb (1949, 1961) postulated that short-term memory is represented in reverberatory circuits as described earlier by Lorento de Nó (1943). Once activated, these circuits can maintain excitation as they are formed by a set of neurons, each of which receives excitation from, and gives excitation to, other members of the same set. If a sufficiently large number of neurons in one such cell assembly is activated, the whole set will become active. The trick to memory storage is that the structure of these cell assemblies is flexible and can be changed rapidly. This requires the strengthening of connections between simultaneously active neurons (Hebb's rule), an assumption which has long been considered the physiological basis of learning and memory storage (Hebb 1949). It is thought that within neural networks the ability of an active synapse to depolarize the postsynaptic membrane is increased, once a certain level of postsynaptic activity is present (based on Hebb's rule, models of the memory have been suggested by Willshaw et al. 1969; Palm 1982, and others). Generally it is assumed that plasticity is realized through NMDA-receptors. (For evidence and models of synaptic plasticity see e.g. Collinridge 1987; Gustafson et al. 1987; Bear et al. 1987). Probably all the synapses on spines are subject to modification in their strength, which would mean that 3 out of four cortical synapses are plastic (Schüz, this volume). Therefore, the build-up and strengthening of a cell assembly requires that a large portion of cells not relevant for the concept of the incoming event be shut off. Otherwise connections would form randomly. From the *in vivo* experiments with single cells, we can infer that a tetanic burst of say 100 stimulations with 100 Hz frequency is sufficient to alter the strength of synapses. Moreover, even weaker activity will be sufficient, particularly when similar firing patterns are repeated several times. Consequently, cortical excitability must be reduced for a fraction of a second or so before a relevant event can be stored in long-term memory. Based on the considerations of the previous section, we conclude that the widespread reduction in cortical excitability or inhibition respectively will show up in a widespread positive wave on the scalp (Birbaumer and Elbert 1988). There is evidence that the P300, and in part the positive slow wave, reflect exactly this process. P300 has been

linked to the updating of memory (Donchin and Coles 1988) and is thought to reflect processes associated with the maintenance of a model of the environment.

The larger the information transmission, the more cells should be involved in the network. As all these must be isolated from background activity, the dysfacilitation must be more widespread, and positivity should increase in expansion and hence in amplitude. An interesting example of the experimental evidence confirming the inhibitory/dysfacilitatory character of the P3 is provided by Woodward et al. (1989). Probe clicks to which subjects had to respond were delivered during the time-course of (an auditory-oddball) P3. Reaction times were significantly slowed during the time course of the P3, a finding which has been recently replicated in our laboratory and which is consistent with the proposed dysfacilitatory process.

Threshold regulation of cortical excitability (TRE-model) and the direction of attention

Pyramidal cells constitute 85% of the cortical neurons (Schüz and Palm 1989; Braitenberg and Schüz 1990)⁴. The connections among them are excitatory and constitute over 70% of all cortical synapses. The strength of synapses between neurons depend to a large extent upon experience. There is, as Braitenberg argued, a danger intrinsic to such interconnected networks that they may become activated beyond control (Braitenberg 1978, 1984). If the number of active networks reaches a certain critical level, there is a high probability that many of the remaining ones will become activated too. A final stage of extreme activation may be seen in an epileptic seizure. Indeed, the few DC-records taken prior to epileptic attacks (summarized in Rockstroh 1991) suggest that extreme negative shifts precede seizure activity. Furthermore, the regulation of slow potentials is impaired in epileptic patients (Elbert et al. 1990). Even sub-epileptic high levels of activation disable information processing, for instance, signal detection.

⁴ This figure may seem surprisingly high given the illustrations in many publications. This bias may arise from man's tendency to give greater attention to more exotic animals, i.e. when we see a rare species of interneurons it may evoke a larger P3 than when we are confronted with another illustration of the common pyramidal cell.

Over-activation might be prevented by a threshold control that could rapidly detect an explosive chain reaction among cell assemblies, and that would then regulate cortical excitability by raising thresholds (Braitenberg 1984; Elbert and Rockstroh 1987).

Furthermore, such a threshold control would provide the brain with the ability to interrupt ongoing activity, when relevant information is received. If thresholds are set high consequent upon the presentation of relevant information, ongoing activity will drop to a low level instantaneously, and activity will survive only in the elements pertaining to the concept of the incoming stimulus. Such interruptions will generate positive waves with amplitudes in the microvolt range, like the P300.

If, on the other hand, the total activity were low, further lowering thresholds would encourage circulation of activity throughout the brain. This would avoid the possibility that ongoing subsequent activations become interrupted. One set of cell assemblies would activate the next one and then another in turn, and so forth. As the activation of cell assemblies might correspond to thoughts, such a process would thereby enable a 'train of thoughts', as Braitenberg has put it. In other words, it would enable processing of related information in a sequential manner. Each thought would be related to the next one.

The feedback process inherent in the threshold regulation process will give rise to permanent fluctuations in the depolarization of the dendritic tree and thus will contribute to the fluctuations observed in the EEG.

If thresholds are not adjusted adequately, the process of connected thoughts would be disrupted. Thoughts might be more loosely related which to some extent could contribute to creativity. In more severe cases it may favour thought disorders leading to erroneously activated networks, giving rise to hallucinatory phenomena. False activation of cell assemblies, would result in massive attentional problems, and it is possible that it might favour a schizophrenic breakdown (Elbert et al. 1983). We have tackled this question by investigating the ability to regulate one's own slow potentials in a sample of subjects believed to be at risk for schizophrenic development (Elbert et al. 1983) and more recently in hospitalized patients (Schneider et al. 1991). Compared to the respective controls, we have indeed observed altered or impaired abilities in the regulation of slow potentials in both groups. Too low an activation will bring the train of thoughts to a halt and might make it difficult to initiate it smoothly. While akinesia and rigidity in the motor system are easily observed, corresponding cognitive disruptions

Fig. 3. Superimposed are six single EEG traces recorded prior to a voluntary self-administration of an electric shock. Over central areas, a negative variation of the EEG-baseline occurs prior to the onset of the EMG for every response (From Elbert 1985)

require more sophisticated techniques to be detected (Canavan et al. 1989).

Finally, if the brain were able to adjust thresholds in advance, threshold control could be considered a mechanism for directing attention to future action. Thereby cortical tuning would improve the processing of an expected event. Examination of slow negativities like the CNV or the Bereitschaftspotential (Fig. 2,3) suggest that such processes do indeed exist! As negativity is generated whenever firing thresholds are lowered, a negative shift prior to an expected event might correspond to a state of preparedness in the underlying networks which are to become activated. This view fits well with the one expressed by Deecke (1976) purely on grounds of experimental observation of slow potentials: "We can then interpret the two very slow potentials, BP and CNV, as reflecting a general facilitation process, preactivating those brain regions which will be needed in the special experimental situation under study".

This leads us to the scenario illustrated in Fig. 4, which provides partial explanation for the flow of event-related potentials: Consider an external event or stimulus producing additional input to the cortical networks. Consequently the momentary activity will be changed. Apart from its specific information transmission, the feedback control will

also change subsequent firing patterns: The additional activation will call for a compensatory increase in thresholds immediately, generating a transient positive EEG-wave. Indeed, a transient positivity (P2, P3) develops in response to more or less every enhancement of cortical activity. This reflexive limiting of cortical excitability (P2) interrupts weaker ongoing activity, while incoming activity, (particularly, when fed strongly enough through subcortical afferents) is maintained. A pronounced dampening of cortical activity may be required further, if the transmission of information to long-term memory has to be extensive, giving rise to a P3. The scenario is illustrated in Fig. 4. Whenever an event is anticipated - being signalled as in the two-stimulus paradigm - or is self-induced, it is advantageous to the organism to adjust thresholds in advance. Thresholds should be lowered to facilitate processing of the incoming information, but they should not become too low, in order to avoid a 'false start' or premature assumptions.

Support for the functional significance of threshold regulation for attentive behaviour can be derived from the relationship between surface negative slow potentials and behavioural responses. For example,

Fig. 4. Explanation of the genesis of the major event-related potential components in the TRE-model. Investigation of these event-related potential components may serve to study threshold regulation and its psychological consequences

response latency is shorter when a reaction time task is presented with high CNV amplitudes than when following decreased negativity (Rockstroh et al. 1982). Spontaneous slow potential shifts have also been found to covary with performance efficiency in various tasks (Stamm 1984; Bauer 1984). When subjects learned to modify their slow potentials systematically within an operant feedback procedure, their performance in various tasks was efficient when negativity was large in those cortical regions related to task processing, whereas subjects performed more poorly if negativity remained suppressed in the 'task-relevant' areas but not elsewhere (Rockstroh et al. 1989).

These data are open to the following interpretation: Tasks presented during increases in negativity are presented when activity was previously low and consequently thresholds have been lowered. Hence, the task is presented to an easily excitable brain region with little ongoing activity, and is consequently processed efficiently.

There is currently no clear evidence which physiological structures participate in the threshold regulation. One candidate is the inhibitory interneurons. About 13% of all cortical synapses are pyramidal synapses which connect to non-pyramidal cells (type I synapses, Schüz, this volume; Braitenberg and Schüz, 1990). There is an equal number of non-pyramidal to pyramidal cell synapses, but they are inhibitory. However, probably less than 2% of all synapses constitute connections among the non-pyramidal cells themselves, and these are type II. It is possible that these inhibitory interneurons help to maintain partial activation but it is unlikely that these cells (with their local range and their limited, inhibitory interconnections) can synchronize larger portions of cortical tissue. Therefore the activity of these cells might not be detectable in surface recordings.

It is more likely that excitability of larger cortical regions can be tuned by non-specific thalamic fibres which synapse on the apical dendrites. This process might be considered a candidate for the regulation of thresholds of neuronal excitability. However such a tuning of nonspecific thalamocortical afferents can only be effective if information about ongoing activity in the networks to be regulated is fed into the system adjusting new thresholds. We must therefore assume the existence of a 'measurement device' that receives information about ongoing cortical activity, and which transmits this information to the thalamus. As suggested by Braitenberg (personal communication) and Palm (1982) the neuroanatomical structures that might fulfill such

requirements are the basal ganglia.⁵ Their role for the generation of slow potentials has been discussed elsewhere (Elbert and Rockstroh 1987; Rockstroh et al. 1989; Canavan and Sartory 1990; Elbert 1991). Further experimental evidence is needed to elucidate the role of sub-cortical structures for threshold regulation and in the TRE-model of event-related potentials as outlined in Fig. 4.

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References

- Arieli A (this volume) Novel strategies to unravel mechanisms of cortical function: From macro- to micro recordings
- Bauer H (1984) Regulation of slow potentials affects task performance. In: Elbert T, Rockstroh B, Lutzenberger W, Birbaumer N (eds) *Self-Regulation of the Brain and Behavior*. Springer, Heidelberg Berlin, pp 216-226
- Bear MF, Cooper LN, Ebner FF (1987) A physiological basis of a theory of synapse modification. *Science* 237:42-48
- Bienenstock EL, Cooper LN, Munro, PW (1982) Theory for the development of neuron selectivity: Orientation specificity and binocular interaction in visual cortex. *J Neurosci* 2:32-48
- Birbaumer N, Elbert T, Rockstroh B, Lutzenberger W (1986) On the dynamics of the post-imperative negative variation (PINV). In: McCallum WC, Zappoli R, Denoth F (eds) *Cerebral Psychophysiology: Studies in Event-Related Potentials*. *Journal of Electroenceph. Clin. Neurophysiol. Suppl* 38:212-219
- Birbaumer N, Elbert T (1988) P3: By-product of a by-product? *Behav Brain Sci* 11:375-376
- Birbaumer N, Elbert T, Canavan A, Rockstroh B (1990) Slow potentials of the cerebral cortex and behaviour. *Physiol Rev* 70:1-41

⁵ Every area of the cortex sends projections to the neostriatum where information is preserved in a roughly topographic fashion (Kemp and Powell 1970, 1971; Yeterian and Van Hoesen 1978) thereby 'lateral' inhibition may serve to focus activity.

- Braitenberg V (1984) *Vehicles - Experiments in Synthetic Psychology*. MIT-Press, Cambridge, Mass.
- Braitenberg V (1978) Cell assemblies in the cerebral cortex. In: Heim R, Palm G (eds) *Theoretical Approach to Complex Systems*. Springer, Berlin Heidelberg, pp 171-188
- Braitenberg V, Schüz A (1991) *Anatomy of the Cortex. Statistics and Geometry*. Springer, Berlin Heidelberg
- Braun Ch, Lutzenberger W, Miltner W, Elbert T (1990) Can subcortical structures generate potentials large in amplitude? In: Brunia CHM, Gaillard AWK, Kok A (eds) *Psychophysiological Brain Research*. Tilburg University Press, Tilburg pp 31-35
- Canavan AGM, Passingham RE, Marsden CD, Quinn N, Wyke M, Polkey CE (1989) Performance on learning tasks of patients in the early stages of Parkinson's disease. *Neuropsychologia* 27:141-156
- Canavan AGM, Sartory G (1990) *Klinische Neuropsychologie*. Enke-Verlag, Stuttgart
- Collinridge GL, Bliss TVP (1987) NMDA receptors - their role in long term potentiation. *TINS* 10 (7):288-293
- Deecke L (1976) Opening remarks on motor aspects. In: McCallum, Knott (eds) *The Responsive Brain*. Wright & Sons, Bristol
- Donchin E, Coles MGH (1988) Is the P300 component a manifestation of context updating? *Behav Brain Sci* 11:357-374
- Elbert T (1978) Biofeedback langsamer kortikaler Potentiale. Minerva, München
- Elbert T (1985) Langsame Hirnpotentiale und Verhalten. Habilitationsschrift, Universität Tübingen
- Elbert T (1986) Externally and self-induced CNV patterns of the brain hemispheres - A sign of task-specific preparation. *Human Neurobiol* 5:67-69
- Elbert T (1987) Regulation kortikaler Erregbarkeit: Im EEG ein deterministisches Chaos? In: Weinmann HM (ed) *Zugang zum Verständnis höherer Hirnfunktionen durch das EEG*. Zuckschwerdt Verlag, München, pp 93-107
- Elbert T (1991) Slow cortical potentials reflect the regulation of cortical excitability. In: McCallum (ed) *Plenum Publishing Corp.*, New York London
- Elbert T, Rockstroh B, Lutzenberger W, Birbaumer N (1980) Biofeedback of slow cortical potentials. *J Electroenc Clin Neurophysiol* 48:293-301
- Elbert T, Rockstroh B, Lutzenberger W, Birbaumer N (1982) Slow brain potentials after withdrawal of control. *Archiv für Psychiatrie und Nervenkrankheiten* 232:201-214
- Elbert T, Lutzenberger W, Rockstroh B, Birbaumer N (1983) When regulation of slow brain potentials fails - A contribution to the psychophysiology of perceptual aberration and anhedonia. In: Mendlewicz J, van Praag HM (eds) *Advances in Biological Psychiatry*, Vol. 13, Karger, Basel, pp 98-106
- Elbert T, Rockstroh B (1987) Threshold regulation - a key to the understanding of the combined dynamics of EEG and event-related potentials. *J Psychophysiol* 4:317-333
- Elbert T, Rockstroh B, Canavan A, Birbaumer N, Lutzenberger W, von Bülow I, Linden A (1990) Self-regulation of slow cortical potentials and its role in epileptogenesis. In: Carlson J, Seifert R (eds) *Biobehavioral Self-Regulation and Health*. Plenum Press, New York pp 65-94

- Gemba H, Sasaki K, Tsujimoto T (1990) No-go potential in monkeys and human subjects. In: Brunia CHM, Gaillard AWK, Kok A (eds) *Psychophysiological Brain Research*, Tilburg University Press, Tilburg pp 133-136
- Gustafson B, Wigström H, Abraham WC, Huang YY (1987) Long term potentiation in the hippocampus using depolarizing current pulses as the conditioning stimulus to single volley synaptic potentials. *J Neurosci* 7 (3):774-780
- Hebb DO (1961) Distinctive features of learning in the higher animal. In: Delafresnaye JF (ed) *Brain Mechanisms and Learning*. Oxford University Press, New York
- Hebb DO (1949) *The Organization of Behavior*. Wiley, New York
- Hillyard SA (1981) Selective auditory attention and early event-related potentials: A rejoinder. *Canad J Psychol* 203-204
- Keidel M (1983) The motor intention potential, an electrophysiological correlate to a volitional intention of a non-performed voluntary movement. *J Electroenc Clin Neurophysiol* 56:1-224
- Kemp JM, Powell TP (1970) The cortico-striate projection in the monkey. *Brain* 93:525-546
- Kemp JM, Powell TP (1971) The connections of the striatum and globus pallidus: Synthesis and speculation. *Phil Trans Roy Soc Lond B* 262:441-457
- Kutas M, Hillyard SA (1980) Reading senseless sentences: Brain potentials reflect semantic incongruity. *Science* 207:203-204
- Libet B (1985) Unconscious cerebral initiative and the role of conscious will in voluntary action. *Behav Brain Sci* 8:529-566
- Libet B, Wright E, Gleason C (1982) Readiness-potential preceding unrestricted spontaneous vs. pre-planned voluntary acts. *Electroenc Clin Physiol* 54:322-335
- Lorente de Nó, R (1943) Cerebral cortex: Architecture, intracortical connections, motor projections. In: Fulton FJ (ed) *Physiology of the Nervous System*. Oxford University Press, New York
- Lutzenberger W, Elbert T, Rockstroh B (1987) A brief tutorial on the implications of volume conduction for the interpretation of the EEG. *J Psychophysiol* 1:81-89
- McCallum WC (1988) Potentials related to expectancy, preparation and motor activity. In: Picton TW (ed) *Human Event-Related Potentials, EEG Handbook* (revised series vol. 3) Elsevier, Amsterdam, pp 427-534
- Mitzdorf U (1985) Current source-density method and application in cat cerebral cortex: Investigation of evoked potentials and EEG phenomena. *Physiol Rev* 65:37-99
- Näätänen R (1990) The role of attention in auditory information processing as revealed by event-related potentials and other brain measures of cognitive function. *Behav Brain Sci* 13:201-233
- Palm G (1982) *Neural assemblies: An alternative approach to artificial intelligence*. Springer, Berlin
- Rockstroh B (1990) Effects of Clonazepam on Hyperventilation-Induced EEG-Changes in Man. *Epilepsy Res* 7:146-154
- Rockstroh B, Elbert T, Lutzenberger W, Birbaumer N (1979) Slow cortical potentials under conditions of uncontrollability. *Psychophysiology* 16:374-380. PINV
- Rockstroh B, Elbert T, Lutzenberger W, Birbaumer N (1982) The effects of slow cortical potentials on response speed. *Psychophysiology* 19:211-217

- Rockstroh B, Elbert T, Canavan A, Lutzenberger W, Birbaumer N (1989) *Slow Brain Potentials and Behaviour*. (second, completely revised edition). Urban & Schwarzenberg, München
- Rockstroh B, Elbert T, Lutzenberger W, Altenmüller E (1991) Effects of the anticonvulsatory benzodiazepine Clonazepam on event-related brain potentials in humans. *J Electroenc Clin Neurophysiol* 78:142-149
- Rockstroh B, Elbert T, Lutzenberger W, Altenmüller E, Birbaumer N, Diener H-C, Dichgans J (1987) Effects of the anticonvulsant Carbamazepine on event-related brain potentials in humans. In: Barber R, Blum T (eds) *Evoked Potentials III*. Butterworths, Boston pp 361-369
- Rockstroh B, Elbert T, Lutzenberger W, Birbaumer N (1990) Biofeedback produced hemispheric asymmetry of slow cortical potentials and its behavioural effects. *Int J Psychophysiol* 9:151-165
- Schneider F, Rockstroh B, Heimann H, Lutzenberger W, Mattes R, Elbert T, Birbaumer N, Basrtels M (1991) Selfregulation of slow cortical potentials in psychiatric patients I: Schizophrenia. *Biofeedback & Selfregulation*, (in press)
- Schüz A, Palm G (1989) Density of neurons and synapses in the cerebral cortex of the mouse. *J Comp Neurol* 286:442-455
- Schüz A (this volume)
- Stamm JS (1984) Performance enhancements with cortical negative slow potential shifts in monkey and man. In: Elbert T, Rockstroh B, Lutzenberger W, Birbaumer N (eds) *Self-Regulation of the Brain and Behavior*. Springer, Heidelberg Berlin, pp 199-215
- von Bülow I, Elbert T, Rockstroh B, Lutzenberger W, Canavan A (1989) Effects of hyperventilation on EEG frequency and slow cortical potentials in relation to an anticonvulsant and epilepsy. *J Psychophysiol* 3:147-154
- Willshaw DJ, Buneman OP, Longuet-Higgins HC (1969) Non-holographic associative memory. *Nature* 222:960-962
- Woodward SH, Brown WS, Marsh JT, Dawson ME Probing P3 with secondary reaction time. (submitted)
- Yeterian EH, Van Hoesen GW (1978) Corticostriate projections in the rhesus monkey: The organization of certain cortico-caudate connections. *Brain Res* 139:43-63