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Threshold regulation—a key to the understanding of the combined dynamics of EEG and event-related potentials

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ABSTRACT Neural networks with mutual excitatory connections, forming the substrate of information processing in the cerebral cortex, might activate each other beyond control. It is suggested that this possibility is avoided by a threshold control of cortical excitability. The tuning of cortical excitability is realized via control of the depolarisation in the apical dendrites, which gives rise to surface negative potentials in the EEG. We suggest that the information about the amount of ongoing cortical activity is projected via the basal ganglia to the thalamus. This in turn projects back to the apical dendrites. The thalamocortical afferent system is controlled by the mediothalamic-frontocortical system. This paper illustrates how such a threshold regulation could provide a key to the understanding of the EEG and event-related potentials.

I. Introduction

Event-related potential (ERP) research is a perfect example of the way in which variation of electrical brain activity may be shown to depend upon psychological variables. The converse, however, that psychological phenomena such as thoughts, feelings or awareness might be dependent upon the physiological processes within the brain which generate the EEG waves, is not easily proven. Indeed such an issue has long been the province of that branch of philosophy concerned with the 'mind-body' problem. Within this context, the concept of 'free will' as an autonomous decision-making force beyond the realm of the natural sciences presents a further obstacle to the development of simple mechanistic approaches to the brain-behavior relationship. In contrast, it has been argued that if we were able to specify completely the workings of the brain, the behavior would become fully predictable. Thus, we are left with a dilemma. Either one denies the concept of 'free will' or one concedes that we cannot go beyond heuristic descriptions of the relationship between brain waves and behavior. (The former choice is not only in conflict with the pride of mankind but also with our inability to predict what will happen in one's own brain in the next moment.)

However, the assumption that the behavior of even simple mechanistic systems must be predictable, is wrong. This was shown nearly a hundred years ago by the mathematician Henri Poincaré (1892) for the three-body problem of classical mechanics. Poincaré's pioneering work was ignored until recently when it was acknowledged as the forerunner of the mathematical description of what has been termed 'deterministic chaos' (for a summary see Schuster, 1984; Holden, 1986; for a non-mathematical introduction see Crutchfield, Farmer, Packard, and Shaw, 1986). The analysis of EEG time series employing the newly developed mathematics has revealed deterministic properties inherent in EEG generation (Babloyantz, Salazar, and Nicolis, 1985; Babloyantz & Destexhe, 1986; Elbert, 1986; Mayer-Kress & Layne, 1987). Nevertheless, while an estimate of the extent to which stochastic and deterministic properties may have generated a time series is possible, in most cases mathematics alone cannot uncover the nature of these deterministic processes. In order to reach this goal, we have to develop models in a deductive manner and to evaluate

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their goodness of fit to the data. This is the adopted approach in the present article.

The model to be described is based on the biocybernetical ideas of Braitenberg (1977, 1978, 1984) suggesting that threshold regulation of neural activity is at the basis of neural processing. Evidence will also be cited that thresholds of cortical excitability have a definite neuroanatomical basis. They are regulated via a loop comprising the basal ganglia and other structures; within this loop the basal ganglia transfer information from the cortex back into the thalamo-cortical system. The latter system, which has been elaborated by Skinner and associates (Skinner & Yingling, 1976, 1977; Skinner, 1978, 1984; Skinner & King, 1980) plays an important role in the regulation of attentive behavior and its related EEG events. Furthermore, it will be shown that such regulatory circuits exhibit features of deterministic chaos and may contribute to the generation of electrical activity in the cortex. In accord with the general approach outlined above (that models have to be evaluated with respect to their goodness of fit with experimental data) the final sections will discuss in which way this contribution may underly EEG synchronization and explain certain components of the event-related potential of the brain. These topics will be dealt with in the subsequent sections in this order. First the basic biocybernetical concepts—cell assemblies, neuronal networks, and threshold regulation (section II)—and their neuroanatomical and neurophysiological bases—thalamocortical circuits and basal ganglia (section III)—will be outlined. Next the question of representation of thresholds in the EEG (section IV) is discussed, which leads to the inclusion of principles of nonlinear systems theory or 'deterministic chaos' (section V). In the final sections (VI and VII) the model is discussed from the point of view of experimental findings on EEG and ERPs. EEG desynchronization consequent upon stimulus input as well as components of the ERP are interpreted as manifestations of excitability threshold regulation.

II. Cell assemblies and threshold regulation

Following an early idea of Hebb (1949), Braitenberg (1978, 1984) assumes that the events relevant to the execution and control of behavior correspond neither to events at the level of single neurons nor to abstract, diffuse properties of the brain, but instead to a network of interconnected neurons called a 'cell assembly': 'A cell assembly is a set of neurons, each of which receives excitation from and gives excitation to some other members of the same set. However, a cell assembly is a collection of neurons which cannot be cut into two separate collections without severing at least two excitatory fibers, one for each direction' (Braitenberg, 1978, p. 173). Afferent neurons which are members of a cell assembly but do not receive excitation in return, do not form part of these assemblies, nor do those on the efferent side, which are excited by a cell assembly without contributing excitation to it.

The strengthening of the connections between neurons is considered to be the basis of learning. The ability of simultaneously active synapses to depolarize the postsynaptic membrane tends to be increased, while mutual excitatory connections might degenerate when not being activated for a long time period. As Braitenberg points out, there is a danger intrinsic to such an interconnected network, namely that it might become activated beyond control: If the number of active cell assemblies reached a certain critical level, there is a high probability that the remaining ones would become activated too. Maximal activation, as seen in an epileptic seizure, however, would impair information processing, since it is certain that the brain processes information through distinct patterns of cortical activation. We know that neurons act like threshold devices and that their firing thresholds are changed by a variation in dendritic depolarization (a component of the EEG). In order to prevent a network of cell assemblies with mutually excitatory connections becoming overaroused, a threshold control is needed that can rapidly detect an explosive ignition among the cell assemblies. If the total number of active elements were monitored, an epileptic attack could be prevented by raised thresholds. Were thresholds of cortical excitability set high enough, cell assemblies would become active only when the afferent input was very strong. If, on the other hand, the total activity were low, lowering thresholds would encourage

circulation of activity throughout the brain. Then, one set of cell assemblies would activate the next one, which would result in a 'train of thoughts' (Braitenberg, 1984).

Such a threshold control would also provide the brain with an ability to interrupt ongoing activity, when relevant information is received. During such an additional input, thresholds are set so high that the previous activity will drop to a low level instantaneously. Elements that were previously active will be switched off, and eventually activity will survive only in elements pertaining to the concept of the incoming stimulus. This illustrates that threshold control can be viewed as a mechanism for focussing attention: If the brain might foresee events and adjust thresholds in advance, attention can be directed to future action. Such a cortical tuning would improve processing of an expected event.

III. Neuroanatomical structures related to attention and threshold control

Skinner & Yingling (1977) presented evidence that both the *m*edio-*t*halamic—*f*rontocortical *s*ystem (MTFCS), and the *m*esencephalic *r*eticular *f*ormation (MRF) regulate bioelectric activity related to attentive behavior. Both systems converge upon the Nc. Reticularis Thalami (R), which 'gates' thalamo-cortical activity (see Fig. 1). The regulation is realized from excitation by the MTFCS and inhibition by the MRF on R-cells, which have inhibitory control over the transmission through sensory relay nuclei.

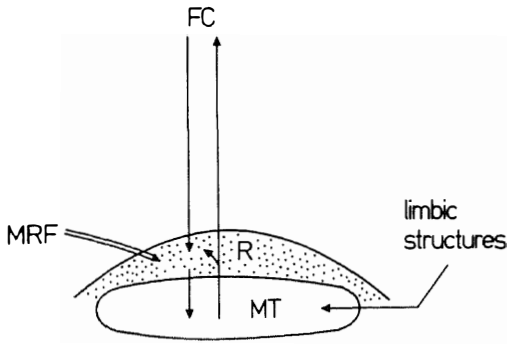

Figure 1 Schematic illustration of brain structures that interact in the MTFCS. MRF: mesencephalic reticular formation; R: nucleus reticularis thalami which surrounds the thalamus like a shell; MT: nucleus medialis dorsalis and unspecific medial nuclei of the thalamus; FC: frontal cortex. If R is inhibited by MRF input, MT is disinhibited and activates FC, which in turn can remove inhibition from R. Limbic afferents modulate MT activity.

Stimulation of the MRF increases ERP amplitudes, blocks EEG synchronization (Skinner & Yingling, 1977), and increases frontal negativity (Arduini, Mancia, and Mechelse, 1957), presumably as a result of the removal of inhibition on R-neurons. On the other hand, a reduction of tonic MRF input to R cells results in positive potential shifts (Caspers, 1963). MTFCS activation reduces sensory EPs and slow cortical negativity, an effect which can be reversed by cryogenic blockade of the inferior thalamic peduncle, i.e., the connection between MT and FC. An event which inhibits R through MRF activation, thereby opening the thalamic 'gates', will lead to a general readiness for information intake. Excitatory influence is exerted on the frontal cortex (FC) via MT which generates a slow negative wave (as the initial CNV, see Rockstroh, Elbert, Birbaumer, and Lutzenberger, 1982; Components of the CNV are also summarized by Rohrbaugh & Gaillard, 1983). If the event is irrelevant, the FC will activate R in turn, thereby interrupting its own excitation. Thus, the ability to focus attention occurs by way of inhibition in the thalamocortical circuits carrying irrelevant information (Skinner & Yingling, 1977). Otherwise a distinct pattern of R activation throughout the FC will release thalamic activation to specific cortical regions.

Our model assumes that excitability of cortical regions can be tuned by nonspecific thalamocortical fibers which synapse with the apical dendrites; by this process the *thresholds* of neuronal excitability are *regulated*. Such a tuning of nonspecific thalamocortical afferents can only be effective if information about ongoing activity in the networks to be regulated is taken into account. Therefore, every grouping of cortical neurons must send information about its ongoing activity to the thalamus; in fact it may do so in a manner which preserves the topographical information. Braitenberg (1984) suggests that this information is transmitted to the thalamus via the paleostriatum. We end up with a loop as illustrated in Fig. 2.

The neuroanatomical connections of such a loop are well documented (DeLong, 1974;

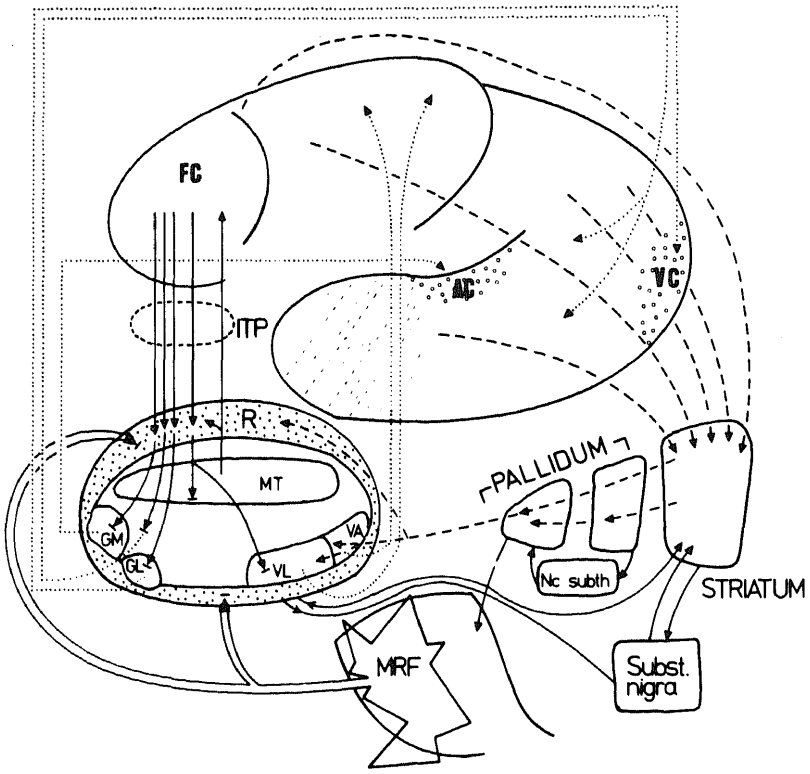

Figure 2 Schematic illustration of brain structures assumed to be involved in threshold regulation: All cortical regions send information about the actual excitation via the basal ganglia (striatum, pallidum) to the thalamus (dashed lines). From there, thalamocortical afferents project back to all cortical regions (dotted lines) being modulated by the MTFCS (solid lines). ITP: inferior thalamic peduncle. The pole of the temporal lobe (hatched area) receives no thalamocortical afferents and, hence, does not participate in the described regulatory loop (this may account for the fact that ERP-amplitudes are generally small over temporal sites).

Scheibel & Scheibel, 1966; for a review of basal ganglia functions see, e.g., Marsden, 1982; Canavan, 1986; Brooks, 1986).

The four major sources projecting to the striatum (caudate nucleus and putamen) are (a) most areas of the cerebral cortex, (b) the midline intra-laminar thalamic nuclei, (c) the substantia nigra and—not shown in Figure 2—(d) the raphe nuclei of the midbrain. The striatum sends its efferents to the pallidum, the major output center of the strio-pallidal complex, which in turn sends afferents to the thalamus and brainstem from where influence can pass back to the cortex. Stimulation of the Nc caudatus activates cortical inhibition. There is also ample evidence indicating the importance of the basal ganglia for mechanisms of attentive behavior and intention. Marsden outlines three lines of evidence from which it derives that the basal ganglia play a role in focussing attention: '1) extensive anatomical inputs into the strio-pallidal complex and the discrete electro-physiological responses of single striatal neurons to environmental events, 2) the striking capacity of electrical stimulation of the striatum to deviate the head and eyes towards the opposite side or, conversely, of striatal ablation to lead to posturing of the head and body in the opposite direction, and 3) the electrophysiological evidence that striatal activation causes widespread behavioral and neuronal inhibition in other areas of the brain' (1982, p. 5). According to Hassler (1978, 1980) the pallidum is an important link in the regulation of goal-directed and attentive behavior. The pallidum transmits the information

about the activity of cortical elements collected by the striatum to the thalamus (Fig. 2). As pointed out by Hassler (1978) each cortical region receives two sets of impulses during perception, conception and intentional motor action: 'One set comes through the specific thalamo-cortical projection from the different sensory or other integrative pathways and one through the non-specific trunco-thalamic projection, which travel the loop through the basal ganglia and which determine the degree of vigilance and awareness... Only if both sets of impulses arrive at the distinct cortical region, do the neuronal electro-physiological processes fulfill the preconditions for a conscious perception or realization'. From his research on lesions in the basal ganglia, Hassler (1980) concluded that 'the global directing of attention as a function of the pallidum becomes a circumscribed focussing of attention of a single event by the action of the striatum' (p. 597). To a large extent the striatum exerts an inhibitory influence on the pallidum, preventing an explosive ignition of the threshold regulatory circuit between thalamus, cortex, striatum, and pallidum. Consequently, destruction of the pallidum results in a loss of consciousness (Cairus, 1952), while pallidum stimulation can compensate for a loss of MRF activation (Hassler, Dalle Ore, Dieckman, Bricolo, and Polce, 1969).

The increase in thresholds regulated through this loop restricts activity in the cortical networks pertaining to a single event, suppressing and attenuating all other activities. This notion is reminiscent of the conclusion reached by Denny Brown, and Yanagisawa (1976) that the basal ganglia may function as a 'clearing house' that accumulates samples of ongoing cortically projected activity and, on a competitive basis, can facilitate one sample and suppress all others. The inputs from the pallidum to the thalamus, especially to R, are tuned by an adaptive system, the MTFCS. This has the advantage that certain features of the threshold regulatory loop can be set in advance. A failure of the paleo-striatal loop to transmit adequate information to the threshold regulator, however, cannot be fully compensated for by MTFCS activity. This results in poor regulation of those cortical areas which project to disturbed parts of the striatum. Indeed, it has been found that restricted lesions of the striatum will lead to behavioral deficits similar to those produced by damage of the areas of the cerebral cortex that project to that region of the striatum (Rosvold & Szwarcbart, 1964; Öberg & Divac, 1979; Marsden, 1982).

Further evidence on the relevance of the basal ganglia stems from investigation of patients suffering from lesions in this system. An impaired dopaminergic projection from the substantia nigra to the striatum is considered to play an important role in Parkinson's disease[1]. In Parkinsonian patients, Deecke, Kornhuber, and Schmitt (1976) observed a marked reduction of the Bereitschaftspotential (BP) contralateral to the akinesic limb, or even a complete absence of the BP over both motor cortices in bilateral Parkinsonism. Since output from the motor cortex depends upon subcortical input from the basal ganglia which project via the ventrolateral thalamus and SMA to the MC, the reduction in BP amplitude can be taken as indicator of striatofugal overactivity. The dopamine deficit results in a failure to adequately dampen striatal excitation which, in turn, would inhibit cortical activity. Higher thresholds would extinguish the internal ongoing activity. Animals become akinetic and their behavior becomes dependent upon external stimuli, i.e. 'stimulus-bound' after blocking of dopaminergic activity (Schmidt, 1987) or after lesions in the ventral lateral thalamic nuclei (Canavan, 1986). Shibasaki, Shima, and Kuroiwa (1978) found that the BP was abnormal in 18 out of 20 patients with Parkinson's disease. The amplitude was reduced and the movement was delayed relative to the potential's onset. Nonetheless, the latter was evident in nearly all patients. These results have been confirmed for 10 patients by Obeso *et al.* (unpublished, cf. Marsden, 1982). Similarly, Tsuda (1982) observed a reduced CNV in 12 out of 14 Parkinsonian patients. These results were supported by intracranial recordings (Tsuda, 1984; Groll-Knapp, Ganglberger, and Haider, 1977). As we will argue, slow surface negativity such as the BP or the CNV

[1]Despite providing information on the consequences of loss of normal functioning of the basal ganglia, it should be noted that this disease is likely to also involve degeneration of dopaminergic neurons in other regions such as the retina, which contains 30 per cent of neurons (Bodis-Wollner, 1988).

indicate changes in neuronal excitability, i.e., in threshold regulation,—and the latter might be impaired in Parkinsonian patients. While akinesic patients may display higher BP amplitudes prior to a voluntary response, the BP may be lower in patients when rigidity or tremor are the primary clinical features. Papakostopoulos (pers. comm.) derived this suggestion from his recent investigations employing the skilled performance paradigm (Papakostopoulos, 1980). Libet (1985) and Libet, Wright, and Gleason (1982) argued that the conscious awareness that one is willing to make a voluntary response is related to a critical level of negativity, i.e., a certain amplitude of the BP. Therefore, Marsden's argument that the changes observed in Parkinsonian patients may 'reflect the inability of patients with Parkinson's disease to execute a discrete fast movement (1982, p. 524) should be reversed: these patients may suffer from inadequate threshold regulation. Their inability to generate fast and efficient rises in cortical excitability may impair their ability to activate motor plans or to execute motor programs (see also Brooks, 1986).

IV. Representation of thresholds in the EEG

The connections between cortical pyramidal cells can be roughly divided into two systems (Braitenberg, 1978). The A-(*a*pical) system connects distant cells, whereas the B-(*b*asal) system connects proximal ones. As indicated by the schematic drawing in Fig. 3, the axons of the A-system (dashed) cross the white matter and project to distant apical dendrites. In contrast, the axon collaterals of the B-system remain in the grey matter and synapse with pyramidal cells located within a distance of 1 mm or less.

EPSPs within the B-system generate a surface positivity, while activation of A-fibers contributes to a negative potential shift on the scalp.

Activation of cell assemblies, which in general include A- and B-fibers, therefore, is not likely to lead to consistent changes in the EEG. PSPs produced by thalamic afferents, however, are known to contribute to the EEG waves. At least one part of this contribution originates in the nonspecific fibers which synapse with apical dendrites relatively distant from their cell bodies. The reason for this separation is that it renders the EPSPs susceptible to an inhibitory 'veto' delivered closer to their cell bodies. This suggests the *apical dendritic* system as a candidate for the regulation of cortical excitability. We assume that the thresholds are realized via thalamocortical afferents projecting to the apical dendrites (Creutzfeldt, 1983; Elbert, 1986). Therefore, a lowering of thresholds for cortical excitability will generally result in an increase in surface negativity, while a positive wave will be generated when thresholds are set high. An anticipatory response, including lowered thresholds, will therefore show up as a negative shift in the EEG.

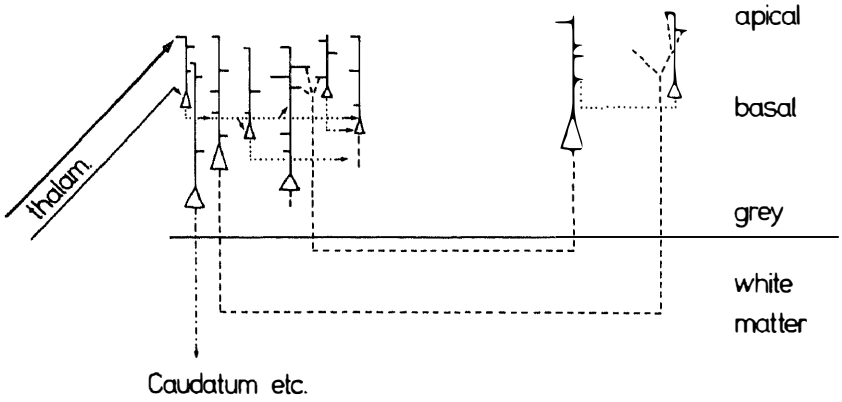

Figure 3 Schematic illustration of connections of cortical pyramidal tract (PT) cells. Dashed lines represent axons of the A-system which cross the white matter and project to distant places; dotted lines represent axons of the B-system which synapse within small distances. One PT cell has about 10^3 to 10^4 neighbours, i.e., it is re-connected to itself after about two synapses. 75 per cent of the cortical neurons are PT cells; 80 per cent of the synapses are synapses between PT cells.

Let us illustrate these basic statements, firstly for an 'automatic' control of thresholds, largely unaffected by incoming stimuli. For very high levels of excitation, and consequently a large number of active cells A(t), the thresholds would be set so high automatically that the activity will drop to a lower level in the next moment. Thus, A(t) predicts the activity in the very next moment $A(t + \tau)$. τ denotes the amount of time required for the regulatory loop cortex → striatum → pallidum → thalamus → cortex to transform the information received from the cortex and to transmit it back readjusting its threshold. The total nonspecific thalamocortical input to the cortex, Th(A), should be inverse to the threshold, thus, large for small A but small, when much activity is circulating. We do not know the exact function which describes Th(A). It may even differ between different cortical regions. But we can consider principal features of the dynamics of this threshold regulation if we assume any simple function which vanishes for large A but is maximal where A approaches zero. A simple function fulfilling these requirements is

$$\mathrm{Th}(A) = r \cdot e^{-A} \quad \text{with } r = \text{constant} \quad (1)$$

The activity in the next moment $A(t + \tau)$ is a result of the previous activity, amplified or dampened through Th(A). Thus,

$$A(t + \tau) = A(t) \cdot \mathrm{Th}(A(t)) = r \cdot A(t) \cdot e^{-A(t)} \quad (2)$$

using expression (1) for Th(A). Equations (1) and (2) describe a recursion formula which determines the time series of the threshold as well as that of the activity, A(t).

V. The EEG—a deterministic chaos?

Equation (2) is illustrated in Fig. 4. (It is similar to the quadratic map that has been described by, e.g. Feigenbaum, 1978; Schuster, 1984, or Holden, 1986). Depending upon the constant r, the time series is not regular but has chaotic features, as is illustrated in Fig. 5.

Such time series have been described by mathematicians as 'deterministic chaos' (Schuster, 1984; Feigenbaum, 1978, 1979). The chaotic behavior is a consequence neither of external noise nor of an infinite number of degrees of freedom. The irregularities are a consequence of the property of non-linear systems which results in an exponential separation of originally close trajectories in state space[2]. Since the initial values as well as the

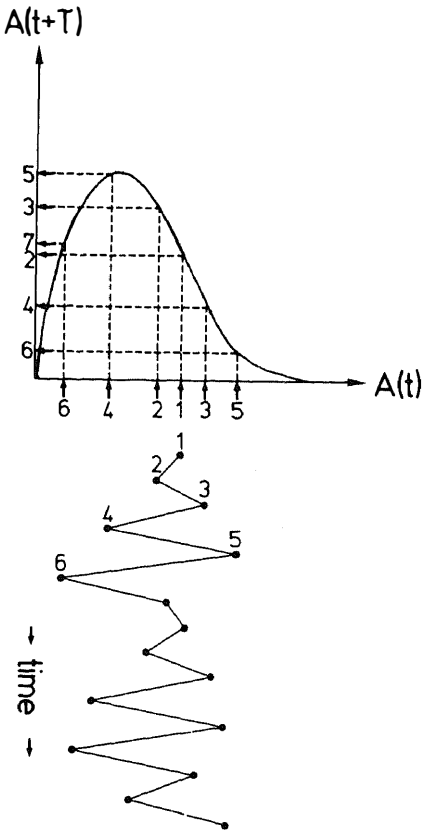

Figure 4 Relationship between A(t) and $A(t + \tau)$ and example of a time series of A(t) generated by the successive iterates according to equation (2): starting with the arbitrary point '1', the next iterate is '2', then '3', etc.

[2]The state space is a useful concept for visualizing the behavior of a dynamic system. It is an abstract space whose coordinates are the degrees of freedom of the system's motion. Since the degrees of freedom and the coordinates, which govern, for example, the generation of a physiological time series A(t) are generally unknown, it is necessary to make use of a technique to reconstruct an 'equivalent' state space. The values measured at fixed time delays A(t), $A(t + \tau)$, $A(t + 2\tau)$ . . are treated as though they were new dimensions. The delayed values then define a single point in a multidimensional state space. Repeating the procedure for different times 't' generates trajectories in the state space. If these trajectories are attracted to a smaller region of the state space with lower dimension, the region is called an 'attractor'. (A time series can be reconstructed from an attractor, as is illustrated in Fig. 4.) For an illustrative introduction into chaos and chaotic attractors see, e.g., Crutchfield *et al.*, 1986.

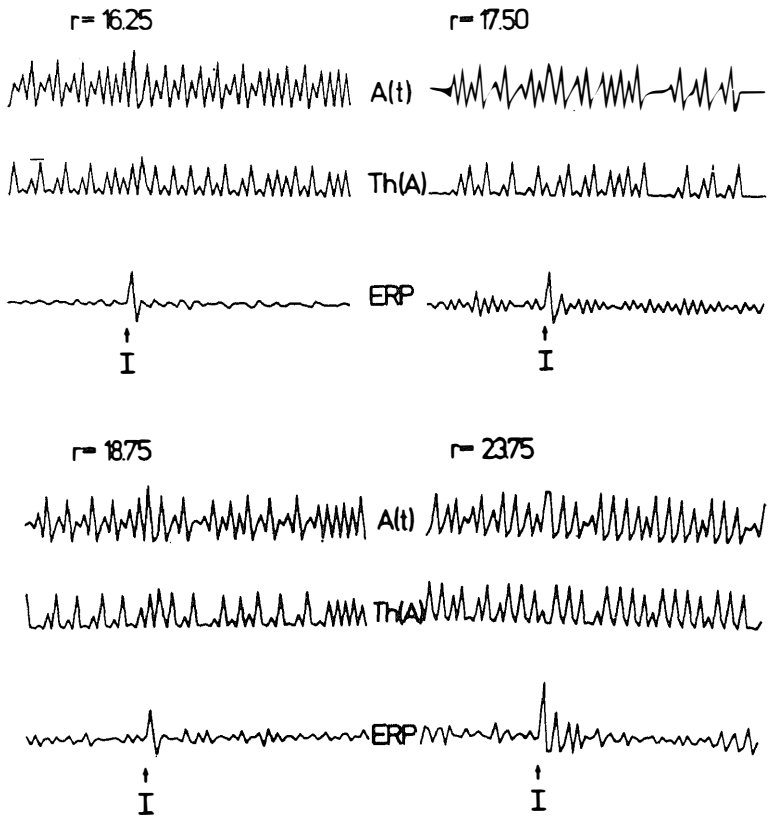

Figure 5 Time series computed on the basis of the iterative regulation as described by equation (2) and illustrated in Fig. 4. It is thought that such time series (when smoothed) represent EEG activity. At 'I' an additional input contributes to A(t). While this is hardly detectable in the raw A(t), it clearly shows up as an input related change when averaged over 10 traces (ERP): A biphasic wave is generated reflexively. The four different panels result from computations with different coupling constants 'r'. For higher coupling, there is a tendency for slowing of the spontaneous frequency and for a more pronounced ERP, resembling a K-complex for $r > 20$ (lower right graph). (We may assume that higher 'r' refer to drowsiness or slow wave sleep.)

successive iterations are limited and since errors increase exponentially, it is actually impossible to predict the behavior of the time series for a long period of time. It may seem that a time series like the EEG offers only a one-dimensional view of brain activity. However, it can be shown that it may provide information about a large number of pertinent variables characterizing the system's dynamics (Takens, 1981; Schuster, 1984; Holden, 1986; Graf, 1986). The primary merit of recent progress in the theory of non-linear dynamics is the possibility to discriminate between the random and deterministic aspects of the system generating a time series. Moreover, the number of variables necessary to describe the dynamics can be determined (Takens, 1981; Grassberger & Procaccia, 1983; Graf, 1986).

Recently, Babloyantz and coworkers (1985, Babloyantz & Destexhe, 1986), Mayer-Kress & Layne (1987) as well as computer simulations from our own laboratory provided evidence of 'strange attractors' for the EEG, i.e., that features of deterministic chaos as suggested by the iterative process, control the EEG, at least during slow wave sleep (SWS). Fractal dimensions of 4.05 and 4.27 were obtained for sleep stage 4 and 4.99–5.03 for stage 2. The results suggest a quasi periodic motion in the state space $EEG(t)$, $EEG(t + \tau)$. This means that a deterministic rather than a stochastic process governs the EEG during SWS, as suggested by

the iterative process indicated in equation (2). Babloyantz obtained chaotic attractors for the EEG in the adult during slow wave sleep but failed to detect chaotic attractors in a space of low embedding dimension (<10) during the waking state or in REM sleep. On the other hand, Mayer-Kress & Layne (1987), as well as Albano, Abraham, de Guzman, Tarroja, Bandy, Gioggia, Rapp, Zimmerman, Greenbaum, and Bashore (1986) suggest a finite dimensionality during the relaxed waking state. Our own computations, however, showed that in most cases the dimensionality remained above 10 even for a relaxed waking state with eyes closed. Cognitive activity in general tends to increase the dimensionality and to blur the fine structure of the attractor.

The achievement of stationarity certainly requires further elaboration. The existence of a chaotic attractor is a consequence of the deterministic nature of the electrical brain activity during SWS. The finite dimensionalities in the range above 4 confirm that equation (2) can serve only as a crude idea. Actually, many loops as that described by equation (2) may be interconnected. The feedback is then continuous rather than discrete, as suggested by Figure 4. Lower dimensionalities of the attractor, however, can be observed in certain disturbed states. Babloyantz & Destexhe (1986) report values around 2 during petit mal seizures. If such properties contributed to the EEG, τ could be estimated by determining the dominant frequencies in A(t) and Th(t). From our EEG analysis it follows that $\tau \sim 30$–50 ms in the awake adult. Antiepileptic drugs may prolong τ by a factor of 2 (and also result in a slowing of the EEG and an increase in EP latencies; Rockstroh, Elbert, Lutzenberger, Altenmüller, Diener, Birbaumer, and Dichgans, 1988). According to Babloyantz (1985) the maximal Lyapounov exponents range between 0.3 and 0.6 (stage 4), 0.4 and 0.8 (stage 2 SWS sleep). It follows that the limits of predictability of the longterm behavior of the system amount from 1 to 3 s.

It is unsurprising that the detection of a chaotic attractor during the waking state is very difficult. Firstly, continuous input and processing of stimuli modifies the activity of the brain presumably in a manner which is not predictable on the basis of the intrinsic properties of the brain alone. Secondly, the modulation through the MTFCS may come into play. The latter regulates the system according to previously learned expectancies being conditionable to a large extent. Therefore, we have to consider event-related—especially anticipatory—responses in the EEG.

Before we turn to these changes, however, let us speculate why regulatory feedback loops—such as the one in equation (2)—should be driven in the chaotic regimen. To put it in another way; why doesn't the brain try to maintain as constant a level of excitability as possible? There are several alternative answers: a general rule guiding the regulation of physiological quantities seems to be that they are not held at a distinct level but rather oscillate within a given range. One simple reason for this is that interoceptors generally fire in response to changes. Therefore, a constant level could slowly drift. In this respect chaos may serve functional roles (Conrad, 1986), such as to generate diversity. Permanently changing the excitability of the brain could for example enable search processes in which a variety of possible operations—thoughts, ideas—are generated and tested. In this way exploratory behavior is enhanced, new solutions to problems can be found, the adaptability is increased, and the behavior becomes unpredictable, which favours also the defense of the organism. Another possible role of neuronal chaos is the prevention of entrainment. As it was pointed out by Conrad (1986) 'it is conceivable that, in the absence of chaos, either very dull pacemaker activity would develop or highly explosive global neural firing patterns would emerge' (p. 10).

VI. Event-related changes in the EEG

Let us now turn to the last topic, in which way changes in both EEG frequency (event-related desynchronization, ERD, or alpha-blocking response) and event-related changes in EEG potential (ERP) as they are observed in response to a meaningful event, can be explained from the point of view of the model. These two event-related changes, ERD and ERP, have rarely been related either to each other or to a common process.

Event-related desynchronization (ERD) in response to stimulation was originally described by Berger (1930) as blocking of alpha

waves. It is reliably observed in response to individual stimuli (in the orienting paradigm, for example), as well as in response to the signal stimulus in the two-stimulus paradigm (e.g., Pfurtscheller & Aranibar, 1977) (see Fig. 6). Considering the former first, we ourselves have described a correspondence between ERD (alpha-blocking), ERP (vertex potential), and slow potential shifts during a typical OR-paradigm (Rockstroh, Elbert, Lutzenberger, Birbaumer, Johnen, and Schnitzler, 1986; Rockstroh & Elbert, 1987; Simons, Rockstroh, Elbert, Fiorito, Lutzenberger, and Birbaumer, 1987). Comparing spatial and temporal patterns of power in the alpha frequency range and averaged ERPs to tone-flash sequences, Pfurtscheller & Aranibar (1977, 1980; Pfurtscheller, Maresch and Schuy, 1977; Pfurtscheller, Buser, Lopes da Silva, and Petsche, 1980; see also Aranibar, Pfurtscheller, and Maresch, 1980) observed time-dependent decreases in rhythmic alpha activity during both visual and acoustic stimulation. ERD turned out to be larger for visual than for acoustic stimulation.

A series of studies by Basar and coworkers with both animals and humans (Basar, 1980; Basar, Demir, Gönder, and Ungan, 1979, Basar, Durusan, Gönder, and Ungan, 1979, Basar, Gönder, and Ungan, 1980, Basar, Basar-Eroglu, Rosen, and Schütt, 1984, Basar & Basar-Eroglu, 1984; Basar & Stampfer, 1985; Stampfer & Basar, 1985) investigated the functional relationship between pre-stimulus EEG changes and post-stimulus ERP. By analyzing the frequency components of spontaneous EEG and ERPs, Basar and coworkers observed coupling and synchrony in the amplitude-frequency characteristics of the ERP. They concluded that a combination of frequency stabilization, time-locking and amplification of spontaneous activity upon stimulation determines the ERP. Furthermore, the evoked response magnitudes could be predicted from certain features of the pre-stimulus spontaneous activity. In more recent human studies these relationships were investigated within an odd ball paradigm and an omitted stimulus paradigm. The EEG prior to an expected event showed reordering or synchronization into 'preparation rhythms'. The P3 then appeared as a continuation of the reordered, stationary prestimulus EEG. The omission of stimuli triggered an EEG enhancement in some cases.

Fig. 6 illustrates the time-courses of the ERP and ERD as they can be observed within two-stimulus paradigms. An ERD is evident in response to S1 parallel to the ERP up to the development of an initial CNV peak. (The temporary increase in alpha power is caused by the vertex potential, N1/P2). During the course of the interstimulus interval, the ERD recovers parallel to the development of the terminal CNV. Hillyard, Picton, and Regan (1978)

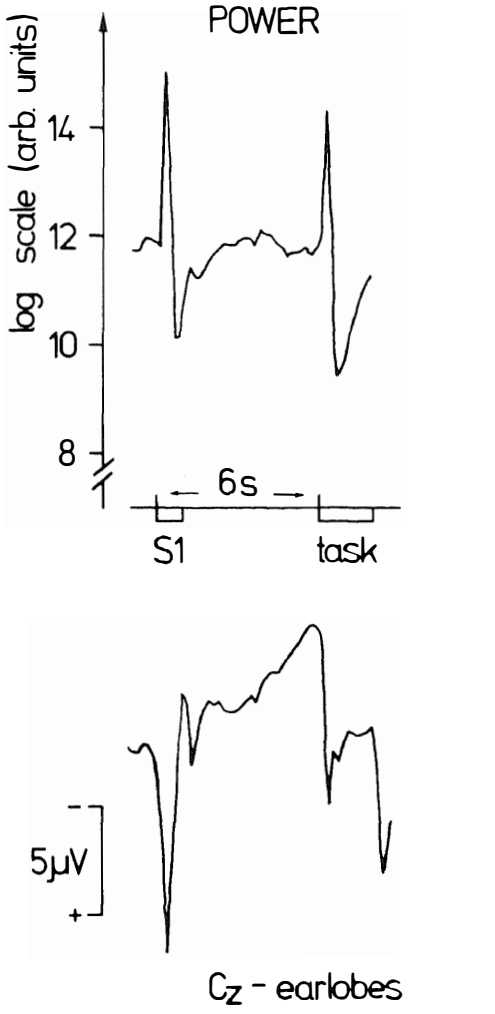

Figure 6 Typical waveforms of event-related desynchronization (power) and event-related slow potentials (Cz referred to earlobes) obtained from a two-stimulus reaction time paradigm with 6 s anticipation interval and 2 s task interval. A tactile S1 of 1 s duration signals a sensorimotor task to be presented 6 s later.

pointed out that late rhythmic components of ERPs were often attributed to a regeneration of rhythmic background activity following transient attenuation during stimulation, while an increase in alpha activity at 500 to 800 ms following stimulation was considered to be specifically evoked by the stimulus. 'It is probable, therefore, that the EP can represent, in addition to specific evoked waveforms, a reorganization or enhancement of background activity' (Hillyard *et al.*, 1978, p. 232). On the other hand, the decrease in ERD parallel to an increase in CNV late in the anticipatory interval led many authors to conclude that there is no consistent relationship between ERD and CNV (see, for example, Grünewald, Grünewald-Zuberbier, and Netz, 1980; Storm van Leeuwen & Kamp, 1980; Denoth, Zappoli, Navona, and Ragazzoni, 1984; Zappoli, Denoth, and Navona, 1980; Lutzenberger, Elbert, Rockstroh, and Birbaumer, 1985; Maresch, Pfurtscheller, Aranibar, and Schuy, 1980). Pfurtscheller & Aranibar (1977), however, report that the two phenomena exhibit different topographical characteristics, from which they deduce different generating processes to be at work. Furthermore, Pfurtscheller & Aranibar (1980) and Pocock (1980) investigated the relationship between ERD and ERP in the 'voluntary response' paradigm: They found parallels in ERD and BP in that both gradually increase prior to the voluntary movement and predominate around the vertex. Obvious dissociations at the point of movement onset—the BP terminates, while ERD reaches its maximum—may be a consequence of (reafferent) potentials.

Let us consider these results from the point of view of the threshold regulation model: An external event produces additional stimulus input I(t). Let $I(t) \neq 0$ in the case of a brief time period. Then A(t) and all subsequent activity will be changed:

$$A(\tau) = A(0) \cdot Th(A) + I$$

By applying equation (2) all further values of A(t) can be computed. Stimulus input, which activates the primary projection areas (possibly generating the N1, for example), will be automatically followed by a compensatory reduction of the thalamocortical input, Th(A), or a compensatory increase in thresholds which should show up in the EEG as a transient positive shift. Indeed, modality nonspecific positive EP components develop after a latency of 100–150ms (P2). The consequence of such a reflexive regulation will be a reflexive EP. This tendency for reflexive enhancement in thresholds has the advantage that ongoing activity may be interrupted, more so with higher values of 'I'. In contrast, the incoming activity fed through *specific* afferents is maintained. Consequently, a reflexive increase in threshold permits the distribution of excitation within a network and enables initiation of activity within distinct networks. If we set the latency of the computer simulated ERP (as shown in Fig. 5) to that of the true vertex potential to an irrelevant stimulus, we once again produce an estimate of $\tau \sim 50$ ms.

If the event is *relevant*, it may enhance 'I' through additional MRF and limbic input to the cortex. This makes ERPs to relevant events different. Whenever attention is directed towards a stimulus, the feedback loop comprising the MTFCS is involved. Information about A(t) integrated by the basal ganglia is not only directly fed back to the cortex but is also transmitted via collaterals to the MTFCS. A dampening of ongoing activity can then be achieved by a lasting increase in thresholds and thereby reduction of Th(A). A 'controlled' P3 instead of an 'automatic' or 'categoric' P2 would be the outcome[3]. Were the stimulus to elicit the activation of additional networks for additional processing, lasting positivity (processing positivity, as evinced in the slow wave within the late positive complex) would result (Rockstroh et al., 1982). This conclusion has also been reached by Rösler (1986) on the basis of his experimental results.

VII. Slow potentials of the brain

Whenever an event is *anticipated* or is self-induced, it is advantageous to the organism that thresholds should be reduced. This process would be manifested as an increasing surface negativity. In order to prevent a 'false start', i.e., a premature activation, the threshold regulation should start late in the anticipatory interval. This fits the view recently expressed by Ruchkin, Sutton, Mahaffey and

[3]The use of terminology is derived from Shiffrin & Schneider, 1977, and Cooper *et al.*, 1979.

Glaser, 1986: 'The timing of the E-wave is related to when in time the process it reflects is needed' (p. 445). This trade-off between efficient preparation—enabled by threshold reduction—and the avoidance of 'false starts'—increase in thresholds—leads to the 'minimax' principle of slow potential (CNV) regulation which has been proposed earlier (Rockstroh *et al.*, 1982) and which manifests itself in the biphasic SP in longer anticipatory intervals.

When the event had been *signalled*, as is the case in the CNV-eliciting two-stimulus paradigm, the input from S1 activates the MTFCS. However, the contingency between S1 presentation and MTFCS activity is favoured when the excitation reverberating in the MTFCS is maintained. This is facilitated by the presentation of S2 within an adequate time interval. Consequent upon this contingency the neuronal presentation of S1 acquires the capacity to elicit threshold regulation in networks that might be involved in S2-elicited processing, i.e., it is conditioned. This threshold regulation is reflected in the terminal CNV.

The behavioral significance of *spontaneous slow potential* shifts, as investigated by Stamm (1984) or Bauer (1984) in the 'potential related event' paradigm, can also be explained within the framework of the threshold regulation model: in this paradigm, task onset is triggered by the detection of slow negative or positive shifts in the EEG. Tasks are performed more efficiently when triggered by increases in negativity than when triggered by positive SP-shifts, this relationship being area-specific. This is open to the following interpretation. Tasks presented during increases in negativity are presented when activity is low and consequently thresholds have been lowered. Hence, the task is presented to an easily excitable brain with little ongoing activity and is consequently processed efficiently.

This interpretation is also supported by evidence obtained from the systematic variation of SPs within an operant feedback paradigm (Elbert, Birbaumer, Lutzenberger and Rockstroh, 1979; Elbert, Rockstroh, Lutzenberger, and Birbaumer, 1980), see also Rockstroh, Birbaumer, Elbert, and Lutzenberger, 1984 for a summary of studies on EEG-biofeedback). When increases in SP negativity were induced by the feedback procedure for 6 s intervals, a larger and longer lasting ERD showed up simultaneously as compared to conditions of reduced negativity. This relationship between self-induced SPs and ERD turned out to be hemisphere-specific when hemisphere-specific SP-polarization was required (Birbaumer, Elbert, Rockstroh, and Lutzenberger, 1988, see Fig. 7). During one type of trial subjects were rewarded for a simultaneous increase in left-central and sup-

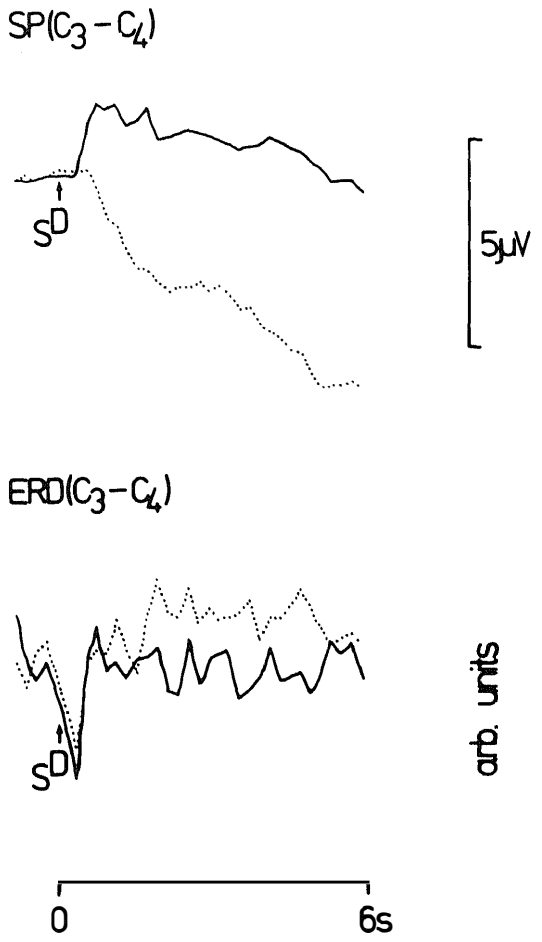

Figure 7 Bipolar SPs (C3–C4) and bipolar ERD (C3–C4) averaged for 6 s SP-regulation intervals with required left-precentral (C3) increase (solid lines) and required right-precentral (C4) increase (dotted lines) in SP-negativity. For ERD the solid line represents less alpha activity over C3 than over C4, i.e., larger and longer lasting ERD, corresponding to increased negativity in SPs at C3; dotted lines represent the reverse, i.e., less alpha activity at C4 than at C3 corresponding to increased negativity at C4.

pression of right-central negativity, while the reverse was required in another type of trial. ERD also showed lateral asymmetry corresponding to the SP-polarization. Alpha activity at C3 was reduced parallel to increased C3- and reduced C4-negativity, while for the reverse SP-polarization the reverse bipolar ERD is evident.

VIII. Concluding remarks

This leads us to the conclusion that in one 'mode' of brain functioning the EEG reflects brain functioning under the control of MTFCS activity. During this mode event-related slow potentials are generated, and EEG synchronization vanishes. In the other 'mode' threshold regulation is automatic, and the EEG tends to oscillate with a frequency in the alpha band. 'There has long been a distinction between states of involvement about which the individual is aware and those in which he performs a task in an automatic and 'unthinking' manner...' (Cooper, McCallum, and Papakostopoulos, 1979, p. 185). In their 'bimodal theory of cerebral processing' Cooper *et al.* (1979) suggest that the latter mode of processing, the 'categoric mode' is associated with the reduction or absence of slow potential changes. The mode of action, which is adopted when 'the subject enters a selective state of dynamic involvement with the environment', which is 'directed towards the execution of a planned action' (p. 184) has been termed 'scopeutic mode' by these authors. A central feature of processing in this mode is the development of SPs. According to our model, the scopeutic mode is the one under control of the MTFCS. Similarly, Norman & Shallice (1985) differentiate two levels of control for action sequences, namely 'deliberate conscious control' and 'automatic contention'. The activation of a network or, as Norman & Shallice expressed it, 'a schema, may not be available that can achieve control of the desired behavior, especially when the task is novel or complex. In these cases, some additional control structure is required' (p. 6). Hence, Norman & Shallice propose a '*S*upervisory *A*ttentional *S*ystem' (SAS) which provides one source of control upon the selection of schemas. The functions which these authors attribute to the SAS 'correspond closely with those ascribed by Luria to prefrontal regions of the brain' (p. 8), i.e., SAS exertion requires MTFCS functioning. Furthermore, Posner & Snyder (1975a, b) have previously proposed a conceptual distinction between 'automatic processes' and 'conscious attention'. Their operational criteria for a process to be classified as 'automatic' were: (1) it must occur without intention, (2) it should not give rise to any conscious awareness, and (3) it should not interfere with other ongoing mental activity. In an attempt to integrate data and theories in the related areas of selective attention, short-term memory search, and visual search, Schneider & Shiffrin (1977; Shiffrin & Schneider, 1977) proposed a theory resembling the one of Posner & Snyder (1975a, b, see also Eysenck, 1982): 'controlled processes' are of limited capacity, require attention, and their constraints are those of short-term memory. 'The typical pattern of findings in studies of visual search and short-term memory search is attributable to controlled processes making serial comparisons at a rate of 40 ms per comparison' (Eysenck, 1982, p. 20). In the 'automatic mode' the EEG oscillations should display deterministic features—but this remains to be confirmed by future research aimed at detecting attractors in the resting EEG. The results illustrated in Fig. 7 suggest that both modes may be present at the same time in different brain regions (as it might also be concluded from Pfurtscheller & Aranibar's suggestions mentioned above). The normal state, then, will reflect an integration of both modes modulated by the effects of incoming stimuli and consequent activities in reafferent pathways. Experimentally, however, it is possible to evoke either of the modes and to test the predictions for EEG and slow potentials of the brain arising from the present work.

'Even the process of intellectual progress relies on the injection of new ideas and on new ways of connecting old ideas. Innate creativity may have an underlying chaotic process that selectively amplifies small fluctuations and molds them into macroscopic coherent mental states that are experienced as thoughts. In some cases the thoughts may be decisions, or what are perceived to be the exercise of will. In this light, chaos provides a mechanism that allows for free will within a world governed by deterministic laws' (Crutchfield *et al.*, 1986).

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