Timer Role of Blood Circulation when Brain Processing Information

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ABSTRACT: Since the year 2006, I have posted evidences and papers on magazine, webs, and conference. (I)Those posts and papers discuss relationship among origin of EEGs, information processing in brain, and blood circulation. (II)Those posts and papers suggest that blood circulation plays the role of basic timer when brain processing information; Suggest one possible molecular mechanism of this 'blood cycle timer'. In the molecular mechanism, H^+, O_2, working mechanism of microcirculation system, H+ seats of NMDA receptor are key factors. (III)Those posts and papers suggest that the sense of 'time passes' is the sense of 'press' that comes of blood flowing in brain. The sense of 'a physical continuous process' is constituted by two senses: (i) Sense of 'press' (ii) Sense come from 'a sequence of active metadata'. This paper is a review of those evidences and papers. Section 1 gives a description of the model. Section 2~5 give the evidences supporting the model, and clarify some problems. Section 6~8 use the model to explain some phenomena of neuroscience (e.g. Origin of alpha wave, EEGs from kinds of sleep phases, etc.).

Keyword: Model of Process storing and recalling; Blood cycle timer; Electroencephalogram (EEG); CNS; Time Cognition; Process of Cognition

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Introduction

Since the year 2006, I have posted evidences and papers on magazine, webs, and conference [1][2-3]. (I)Those posts and papers discuss relationship among origin of EEGs, information processing in brain, and blood circulation. (II)Those posts and papers suggest that blood circulation plays the role of basic timer when brain processing information; Suggest one possible molecular mechanism of this 'blood cycle timer'. In the molecular mechanism, H^+ and O_2 are two key factors. (III)Those posts and papers suggest that the sense of 'time passes' is the sense of 'press' that comes of blood flowing in brain. The sense of 'a physical continuous process' is constituted by two senses: (i) Sense of 'press' (ii) Sense come from 'a sequence of active metadata'. This paper is a review of those evidences and papers. Section 1 gives a description of the model. Evidences supporting the model given in section 2~5, and clarifying some problems. Section 6~8 use the model to explain some phenomena of neuroscience (e.g. Origin of alpha wave, EEGs from kinds of sleep phases, etc.).

1 Viewpoints about origin and meaning of EEGs in Model of Process storing and recalling

This section gives a description of the EEG model and expounds the viewpoint: blood circulation plays the role of basic timer when brain processing information. Introduce the result of modelling at the beginning of this paper is good for improving the understandability of other sections that describe evidences of the model.

1.1 Basic timer function of blood circulation

In every cardiac cycle, artery blood perfusion arrive different small partial groups of nerve cells on different time points. Because the existence of microcirculation system and period of arteriole vasomotion is 5~6 times of cardiac cycle, there are partial groups of nerve cells that didn't get artery blood supply in a single cardiac cycle. For every 5~6 cardiac cycles, statistical viewpoint considers every small partial groups of nerve cells gets artery blood perfusion at least once(Name it Patulous-Blood circulation). In a Patulous -Blood circulation, arrange groups of never cells into a sequence by the order of artery blood perfuse arrival (Figure 1).

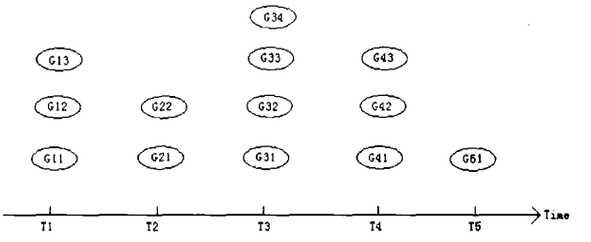

Fig.1 Arrange groups of never cells into a sequence by the order of artery blood perfuse arrival

Sequence of Fig.1 repeats in every Patulous-Blood circulation (Some factors may adjust the sequence in a certain extent).The cyclical variation of biochemical environment in cerebra, which is

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determined by the blood circulation, is the basic timer for the cooperation of large quantities of nerve cells when brain processing complex information. One example of artery blood's timing control's function: nerve cells that get artery blood supply at the same time have larger probability to store an integrated unit of information (name it a 'metadata', e.g. a frame of image) basing on plasticity of synapses. Each metadata mapping to one group of nerve cells who storing it. In Fig. 1, tag a group with 'G'.

1.2 Description of EEG model built

Concentration wave of matters brought by artery blood, and the trigger signals (come from perfusion of artery blood/other ways), cause the naissance of δ wave. δ wave reflects concentration wave of matters that brought by artery blood (e.g. O2 etc.). Activities of metadatas (or input information) have chopping effect on δ wave —— Amplitude of wave decreases and a δ wave is chopped into a sequence of lesser waves. If number of active metadatas is small in a time slice (α wave recorded on EEG), silhouette of concentration wave is still observable: amplitude modulation of α wave. When number of active metadatas (or input information) in a time slice becomes larger, silhouette of concentration wave is no longer observable; β wave is recorded on EEG at this time (Fig. 2).

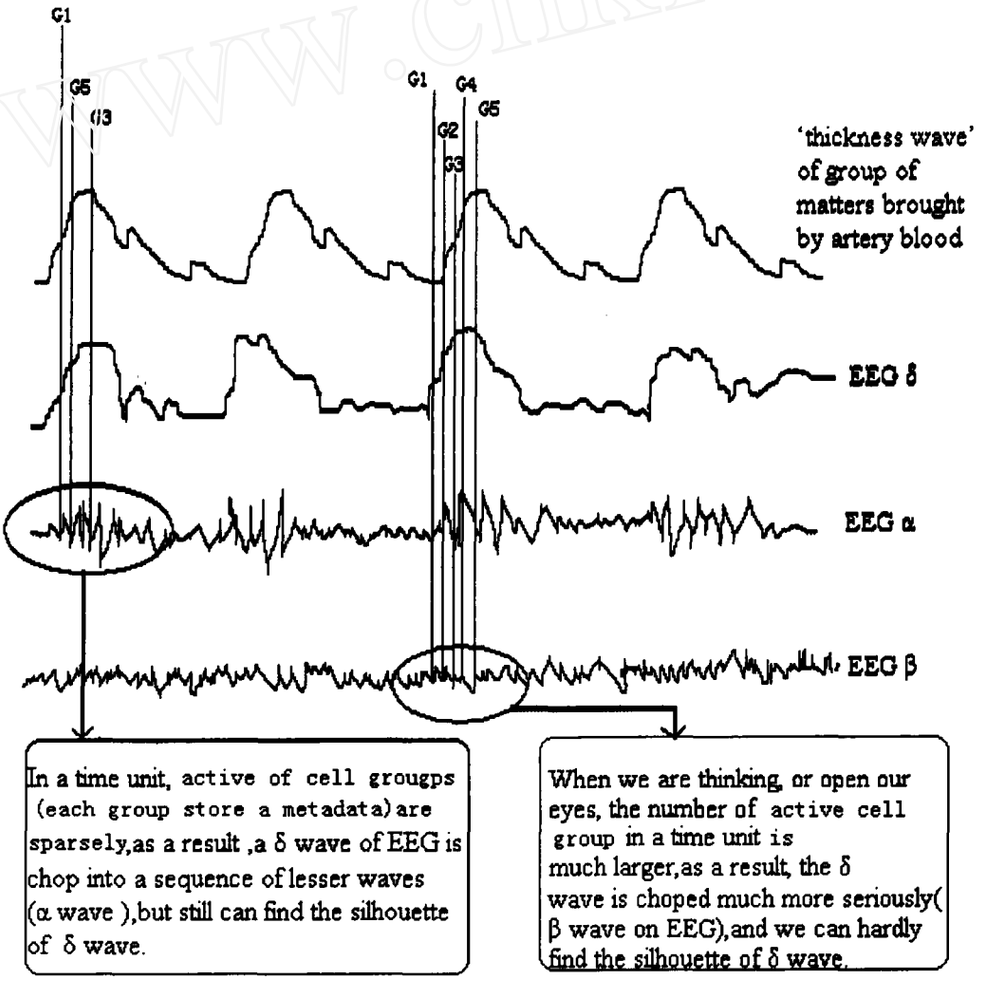

Fig.2 Relationship among 'Concentration wave' of matters brought by artery blood (e.g. O_2 etc.), 3 kinds of EEG waves, and active of metadatas

2 Evidences from clinical EEG phenomena

This section expounds clinical EEG phenomena that support the model and viewpoints of section 1. Sinusoids "C (t) = A1 * sin (w1*t + q1), δ (t) = A2* sin (w2*t + q2), AMSα (t) = A3* sin (w3*t +q3)" are used to describe 'Concentration wave' of matters brought by artery blood (e.g. O_2 etc.), δ wave, AMSα" in Fig. 2 of section 1. Clinical evidences that prove these sinusoids have relationships shown in Fig. 3 are needed when proving the EEG model in Fig 2.

Mapping relationships among the 3 Sinusoids in Figure 3 are described by following points: (1) A2>A3 >amplitude ofβ wave, A2 and A3 decrease when A1 decreases. (2) 3 Sinusoids have same periods: w1=w2=w3. (3) 3 Sinusoids have same phase: q1=q2=q3. (4) 3 Sinusoids have same domain. 2.1 verifies (1) and (2); 2.2 verifies (3); 2.3 verifies (4); 2.4 verifies 'chopping effect'; 2.5 discusses about whether the model conflicts against the experiments that support 'EEGs is origin in summation of PSP'.

These verifications and discussion have following preconditions: (1) Intensity of 'chopping effect' is controlled (e.g. close eyes etc.). High 'chopping intensity' will makes silhouette of 'concentration wave' invisible: only fast waves are recorded on EEG. (2) Brain tissue is not serious injured. (3) Waveforms of EEG are steady and able to endure for a period of time. (4) Origin of δ waves in band 2~3HZ are discussed after the discussion about microcirculation mechanism.

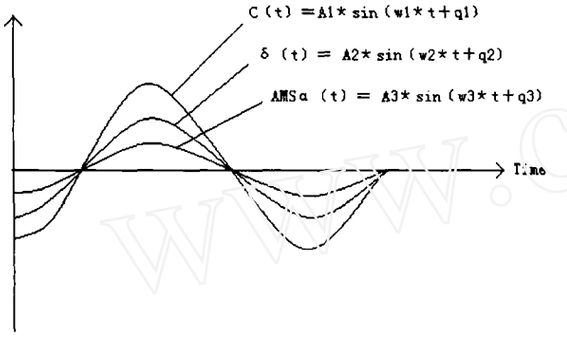

Fig. 3 Mapping relationships among 'Concentration wave' of matters brought by artery blood (e.g. O_2 etc.), δ wave, and AMSα (Amplitude modulation silhouette)

2.1 Discussion about amplitudes and periods

2.1.1 Parameter comparison of α wave, βwave, δ wave From Table 1, amplitude modulation period of α wave is near equals to period of δ wave, and amplitude of α AMS is comparable with amplitude of δ wave. Data of Table1 comes from textbook [4]. After analysis more EEG data, we found many cases of α AMS whose period on small side of 1second is common, and mean period of several sequential α AMSs is about 0.8 second - near equals to mean period of cardiac cycles (heartbeat frequency of healthy adult is about 75 times/minute). From δ wave, α wave to β wave, the amplitude is degressive. From above facts, an intuitionistic concept of the model can be got.

Table 1 Compare parameters of four kinds of EEGs

Eeg Kind	Frequence(Hz)	Period(S)	AMPLITUDE(μV)
α wave	8~13	0.0769~0.125	20~100
AMS of α wave	0.5~ 1	1~2	20~100
β wave	14~30	0.0333~0.07	5~20
δ wave	0.5~3	0.33~2	20~200

* AMS means amplitude modulation silhouette. Data from [4]

2.1.2 Blood supply obstacle and "disappeared" of α Amplitude Modulation In the EEG model, amplitude modulation of α wave reflects 'concentration wave' of matters brought by artery blood. When blood supply becomes poor (but doesn't trigger 'balance traces switching', a phenomena discussed in later sections), amplitude of δ (t)/AMSα (t) becomes smaller. There are clinical evidences (Figure 4). More cases about this problem were found in research reports about EEG change when blood supply obstacle happens (Table 12-1 of reference [5]): 32 of 72 cases (44.4%, highest percent in change styles) have lazy wave on artery blocked side. These cases show that A2 and A3 decrease when A1 decreases.

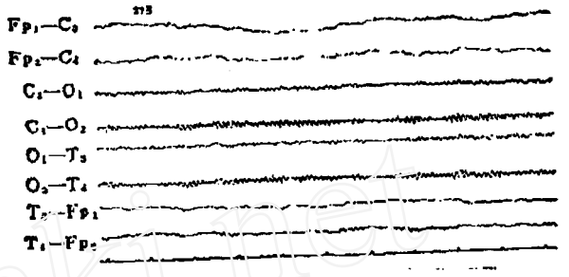

Fig. 4 Blood supply obstacle and "disappeared" of Amplitude Modulation Phenomena

* Male, 37, left MCA blocked, 4 months' ill history, awake. EEG: Left lazy wave, the exponent of α wave decrease markedly. Difference between left and right above 50 %.(Data comes from [5].Figure 12-8)

2.1.3 Clarify a question about amplitude changing. In Table12-1 of literature [5], there are cases that have high-amplitude theta waves and high-amplitude δ waves. Here are reasons why their amplitude is higher than normal α wave /βwave: (1) In the EEG model of section1, activities of metadatas (or input information) have chopping effect on δ wave. Smaller number of active metadatas in a time slice ends in higher amplitude of EEG wave. (2) When blood perfusion obstacle happened, because raise of Ca^{2+} in cells /other factors, phenomenon of 'Balance trace switching' appeared. Section 5 will expound this phenomenon in greater details.

2.2 Phasic relationship between α AMS and cyclical perfusion of artery blood.

The following research conclusions are helpful to solve this question. (1) Inbreathe air containing 10% of CO_2, PCO_2 of blood becomes higher; pH value of blood becomes lower. At the same time, frequency of EEG becomes higher; amplitude of EEG be comes lower [6-7]. (2) After stop of blood flow, change of EEG is: disappear of alpha wave →appear of fast wave →appear theta wave. After stop of blood flow, O_2 monotonously decrease, H^+ monotonously increase in the brain tissue. (3)Change of EEG when inbreathe air of low O_2 is: EEG doesn't change obviously→ decrease in amplitude and periods → amplitude increases and waveform becomes regular→amplitude and periods increase obviously [6].(4) There is an interesting sentence in some book: β wave mingles with α wave, and these two kinds of waves form the phenomena of α amplitude modulation [5]. In clinical cases of α amplitude modulation, it's common that the lower-amplitude part of wave has higher frequency. (5) Transport of CO_2/O_2 and cerebral blood flow. Arrived of artery blood bring abundant O_2. With time passing by, H^+ increases with CO_2's increase. H^+ increases until reaches a gate value of triggering the diastole of blood vessel. Then, with the arrival of artery blood, CO_2 and H^+ are cleaned, and O_2 becomes abundant again. The process described goes round

and round, again and again. From (1) (2) (3) (4) (5), we draw this conclusion: Wave crests of α AMS mapping to time point when corresponding area is abundant in O_2. With time passing by, Frequency of EEG becomes higher because increasing of PCO_2. Then, with arrival of artery blood, Frequency of EEG becomes slower again. This conclusion opens out the phasic relationship between α AMS and cyclical perfusion of artery blood. This conclusion is able to explain phenomena of (1) (2) (3) (4).On the other side, conclusion of "Wave crests of α AMS mapping to time point when corresponding area is lack of O_2." has this deduction: After stop of blood flow, change of EEG is "EEG frequency becomes lower, high amplitude alpha wave without α beating recorded → high amplitude slow wave",this deduction conflicts against facts in (2) (3).

2.3 Relationship between appearance of δ wave/ AMSα and blood perfusion.

2.3.1 Adams-Stokes syndrome Attention should be paid to an Adams-Stokes case, Fig. 11-11 of literature [6] (Jung, 1952) .One minute after stop of systole, with the recovery of systole, high-amplitude δ wave appeared on EEG. They are synchronous on time, and at the beginning, they have same periods.

2.3.2 Domain relationship between δ wave/ AMSα and blood perfusion. In model of section 1, under preconditions that have been described in senction2, domains of δ (t), AMSα (t) are determined by domain of C (t). (1) In cases of vasodepressorsyncope or stop of blood flow, changes on EEG are: disappear of alpha wave→appear of fast wave →appear theta wave. Domain of AMSα (t) is determined by domain of C (t).One more problem need to been clarified is: "disappear of AMSα" is talking about the steady AMSαs that are able to repeat in aptotic frequency and to endure for a period of time. "Disappear of AMSα" doesn't imply "absolutely without AMS". In fig. 11-13 of reference[6](EEG record of Aschner experiment), after systole stop, before EEG become flat, in the 10 seconds' period when fast wave switching into theta wave, there are several times of amplitude modulation on EEG. This phenomenon may origin in active of microcirculation system, and it need more research. (2) From description in 2.3.1 shows that domain of δ (t) are determined by domain of C (t).

2.4 Active metadatas' effects on EEG

2.4.1 About chopping effect Description "a δ wave is chopped into a sequence of lesser waves" is macroscopical. This description doesn't imply constrain that "active of metadata mapping to trough of α wave /βwave on EEG". That's the reason why human have different kinds of α wave /βwave waveforms, but same normal psychic function.

2.4.2 Evidences that support chopping model Examples that support chopping model and the concept of 'metadata'.

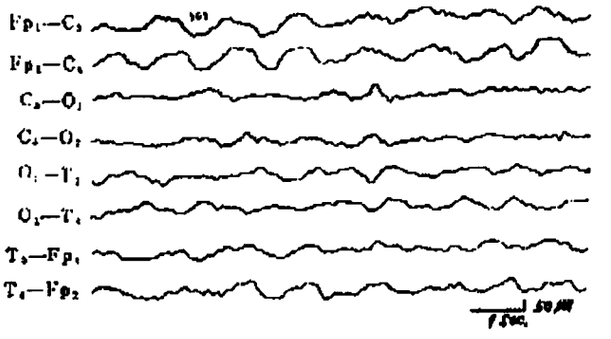

Fig. 5 Waves of Higher Frequency Superpose on Waves of Big-Slow Waves

* Female, 8, Epidemic cerebrospinal meningitis (Recovering Stage), awake. EEG: Exponent of δwave high. Theta wave (4~5Hz) superpose on big slow wave. (Data comes from [5].Figure 14-4)

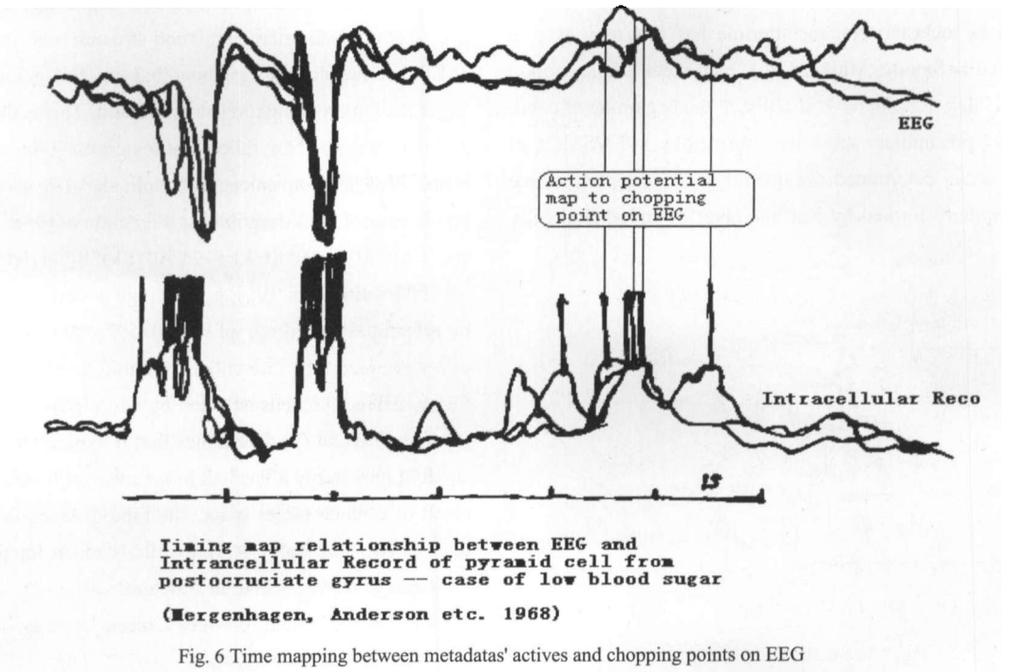

Fig. 6 Time mapping between metadatas' actives and chopping points on EEG

Other clinical EEG examples: On EEGs from patient suffering different kinds of diseases, time points when 'metadatas' are not active are usually mapping to slow wave (e.g. For typical absence seizure, it's accepted that time points when psychic obstacles happen mapping to slow-wave phrases of spike-and-slow waves on EEG).When people concentrate on 'after image', α wave is recorded on EEG, this is an evidence for the concept of 'metadata' and chopping effect.

This model didn't conflict against experiments that support EEGs is origin in summation of PSP.

3 Evidences from Anatomy and Microcirculation

3.1 introduce anatomical evidence for EEG model of section 1.3.2 introduce the structural bases for timer role of blood circulation. These bases come from anatomy and microcirculation research. 3.3.1 gives a brief introduction of microcirculation structure. 3.3 collects the discussion about EEG phenomena that relate to microcirculation.

3.1 Newborn cats have slow wave and few branches of top dendrites, EEGs of different frequency appeared with increase of synaptic on top dendrites after10~12 weeks.

3.2 Structural bases for timer role of blood circulation

(1) The density of capillary distributing has close parallel relationship with the quantity of synapses and neuropil this provide a structural probability for the EEG model of this paper. (2) In brain tissue, blood supply radius of each capillary is about 20 micro meters. Cell body of pyramid cell is about 0.01~0.02 milli meters [4]. This granularity matching provides a structural probability for timing control function of blood. (3) One critical working of microcirculation structure is that capillaries open rotationally.

3.3 EEG phenomena that relate to microcirculation

3.3.1 Structure of Microcirculation unit and Granularity Arteriole dispatched into metarterioles, metarteriole has just one layer of VSMC (Vascular Smooth Muscle Cell), each metarteriole support 1~several capillary (capillaries). Usually, at the beginning of capillary, there is a precapillary sphincter (formed by 1~2 VSMC), its vasomotion status determined the quantity of blood perfuse into capillary. Capillary formed by just one layer of endothelial cells,

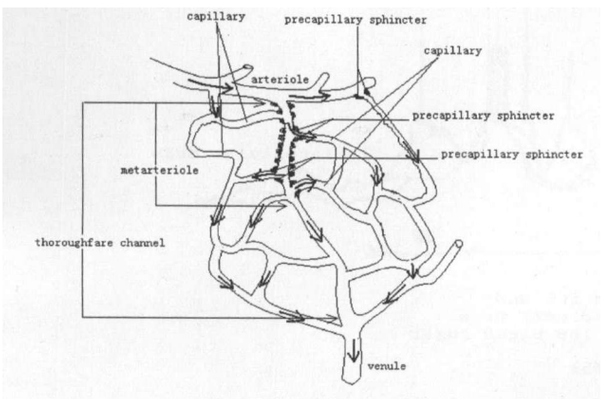

Fig. 7 Microcirculation mode

the length is 0.5~1mm. This granularity is same as pyramid cell. A pyramid cell (including axon and dendrites) is about 1mm. The matching of granularity supplies portability and structural base for the timer function of blood circulation. (Figure 7, from literature[4])

3.3.2 Microcirculation mode and EEGs of 3HZ In EEG phenomenon, 3HZ wave is a common frequency. It is accepted that 3HZ wave origin in thalamencephalon, however, origin of 3HZ waveforms is another thing. Under the framework of this model, active of microcirculation structure is one of the causes of the 3HZ waveforms. For example, waveforms of Triphasic waves (Patients suffer hepatic encephalopathy, frequency range 1.2-2.7 HZ) and Spike-and-slow Waves (Patients suffer typical absence seizure, main frequency 3HZ, frequency range 2.5-4 HZ) have following similar mechanism:

(1)The origin rhythm from thalamencephalon 3HZ is enhanced morbidly by some reason (e.g. abnormal GABA), and forming the first phrase of triphasic waves/the spike phrase of Spike-and-slow Waves. And makes the nerve cell silent for a period.

(2)For the reason of granularity matching that described in 3.2 and 3.3.1, enhanced trigger signal get nearby capillary, opens the sphincter or causes vasomotion. Blood flow into the corresponding area.

(3)Matters brought by artery blood affect the summation of PSP. And at this time, there is no 'chopping efforts' because the reason described in 3.3.2 (1). At this time, 2nd and 3rd phrases of triphasic waves and slow wave phrase of Spike-and-slow Waves recorded on EEG.

(4)Comparing against Spike-and-slow Waves, triphasic waves has lower amplitude and longer period. High GABA maybe the reason.

3.3.3 Granularity differences between inspecting range of EEG active electrode and microcirculation structure unit Inspecting range of EEG active electrode is about 2~3 cm, this granularity is much larger than microcirculation structure unit. This is the reason of the problem described in following sentences: Use sinusoid to describe EEG for convenience. Amplitude of δ wave recorded by bipolar recording is described as a function of time:

$$\delta(t) = A0*f(t) - A0*f(t+k) = -2* A0*f(k)*f[(p/4)+(t+k/2)]\cdots(1)$$

f (t) = sin[(2*pi*t)/p], p = average period of δ wave recorded by referential recording, pi = 3.141592, A0 = average amplitude of δ wave record by referential recording, k = time distance of perfusion arrived the cells detected by two electrodes. Existence of f (k) in expression (1) determines that if experiments found δ (t) is small, it may imply k is small too. However, k reflects a statistical result of a much larger space, the time distance that artery blood reaches two metadatas' storing cells is much longer because the existence of microcirculation structure.

3.3.4 Period differences between concentrate wave and cardiac cy-

cle Microcirculation system makes the difference of periods between concentrate wave and cardiac cycle in a certain extent. However, for a granularity larger than 2~3cm, the change is small. The blood flow modulating capability of microcirculation system (e.g. thoroughfare channel) and the system structural reason that maintain encephalic pressure buffer the affects of heartbeat rhythm change and control the blood flow. They make the timer function of blood circulation more reliable.

4 Evidences from molecular mechanism

4.1 NMDA Receptor

(1) Inhibit function H^+ seat on NMDA receptor: Inhibit function of H^+ seat on NMDA receptor is independent from the membrane potential. Part of NMDA receptor action has been inhibited when pH value is 7.4, when pH value is 6.6, 50% of NMDA receptors' action is inhibited.

(2) Character of NMDA Receptor: when NMDA Receptor actives, channels open, PNa^+, PK^+, PCa^{2+} increase. Inflow of Na^+, Ca^{2+} and leak of K^+ engender slow EPSP. Opening of NMDA channels and inflow of Ca^{2+} are key steps when forming memory.

(3) 80 % of excitatory synaptic have NMDA receptors; this means that an element which able to modulates action of NMDA receptors will affect 80% of excitatory synaptic, forming of memory, and summation of PSP.

Deduction 1: From (1) and 2.2 (5), when O2 is abundant, H+ low (pH value High), inhibit of NMDA receptor is weaken, end in reinforce of EPSP. Then with the increase of H^+, inhibit of NMDA becomes stronger, EPSP becomes weaker on time axis. H^+ continues increases until reach a gate value of triggering the diastole of blood vessel. Arrival of artery blood then cleans the CO_2 and H+, environment back to O_2 abundant state, inhibit of NMDA receptor released. The process described continues from one end to another begin, again and again.

Deduction 2: From (3), whether biochemical environment is advantageous for forming memory changes with the cyclical process described in deduction 1. This talent blood circulation the timing control capability when information stored in to the brain.

Deduction 3: (i) Inflow of Ca^{2+} modulate active of K^+ channel by modulating the mechanism of phosphorization, engender depolarize. (ii) NMDA receptor distribute widely in cerebra. Its state affects summation of PSP.

4.2 Molecular mechanism of forming memory

The biochemical reaction path of early LTP model is: Ca^{2+}inflow through NMDA channel →Ca^{2+}/CaM →Ca^{2+}/CaM kinase → phosphorization of AMPA receptor. In the described reaction path, the precondition (O_2, ATP, etc) for the first step and last step is depending on matters brought by artery blood. For this reason early-LTP reaction becomes much stronger by the arrival of artery blood.

4.3 Activity of GABA system

In area where artery blood is abundant, because reasons described in 4.1, biochemical environment in this area is advantageous for active of nerve cells. Active of M-Ach/GLU end in release of NO, then NOcGMP system depresses the GABA system in that area. Active nerve cells in this area will release more GABA transmitter to farside cells. This mechanism enhances the timing control capability of blood circulation.

5 Discussions of some problems

5.1 How concentrate wave changed into δ wave

This paper suggests two possible ways. Case1 Origin of EEG is summation of PSP. Concentrate wave modulates the timing and quantity of release, storing, functioning, and invalidation of ions, transmitter, buffering proteins; Concentrate wave modulates the working of membrane transport proteins and ionophores; these modulations result in macroscopical δ wave directly. Case2 Two elements (I) what have been described in Case 1 only provides an 'environment base' (determined the amplitude and the periods silently), (II) To 'develop' concentrate wave into δ wave, trigger elements is need. For example, arrival of artery blood releases the constraint of NMDA receptors. However, this effect isn't 'developed' until some trigger signals arrive at the cell and open NMDA channels. Trigger elements come from kinds of ways: thalamencephalon, blood perfusion, input information from environment, etc.

5.2 Balance traces switching

5.2.1 In model of this paper, when a healthy adult awake, balance point of biochemical reaction network in cerebra fluctuates with the 'concentrate wave' determined by blood circulation, and forms macroscopical δ wave. Activities of metadatas make 'chopping effect' on δ wave, result in α wave /β wave. The waveform differences among these 3 kinds of waves are mainly determined by number of active metadatas. They belong to a same balance trace.

5.2.2 There are many situations in that number of active metadatas is not the only reason why waveform changes. For example, when perfusion obstacle happens, abnormal inflow of Ca2+ causes the prevalent rise of voltage in cell. When balance point of biochemical reaction network in cerebra fluctuates with the 'concentrate wave' determined by blood circulation, it is on another balance trace. EEGs recorded when numbers of active metadatas are different belong to this same new trace.

5.2.3 Base what have been described in 5.2.1 and 5.2.2, a one-to-one mapping can be build between an EEG group and a balance trace. Process that balance point of biochemical reaction network in cerebra switches from one balance trace to another balance trace is defined as 'balance traces switching'.

6 Application of model——Balance traces switching of EEG groups and active of Reticular formation of brain steam

6.1 Phenomena analysis

(1) Divide the process of falling into sleep by characters of EEGs: stage of suppressed waves, stage of ripple waves, stage of hump waves, stage of mixture of humps and spindle, stage of spindle, stage of sleep hills.

(2) Awake-EEG-group: α wave (8-13HZ), β wave, δ wave when awake

(3) Deep-sleep-EEG-group: waves in stage of spindle (contain but not only sigma wave), big slow wave in stage of sleep hills. Wave in stage of spindle is mapping to α wave in awake-EEG-group; big slow wave is mapping to δ wave when awake. (Figure 8 [5])

(4) EEGs in other stages (stage of suppressed waves, stage of ripple waves, stage of hump waves, stage of mixture of humps and spindle) reflect the switching process from awake-EEG-group to deep-sleep-EEG-group.

(5) Comparing two groups: (I) the amplitudes become higher (II) α wave (awake-EEG -group) much more regular than wave in stage of spindle (deep-sleep-EEG-group).

(6) According to 5.2, δ wave (awake-EEG-group) reflects the balance trace of biochemical reaction network. It is determined by the 'concentrate wave' when people awake. - Name it C1. Big slow wave (deep-sleep-EEG-group) reflects the balance trace of biochemical reaction network It is determined by the 'concentrate wave' when people sleep deeply. - Name it C2.What have described in (4) reflect the switching process from C1 to C2. Parti tion of reticular formation of brain steam is pre-requested for the sleep-awake mode (a proved fact).

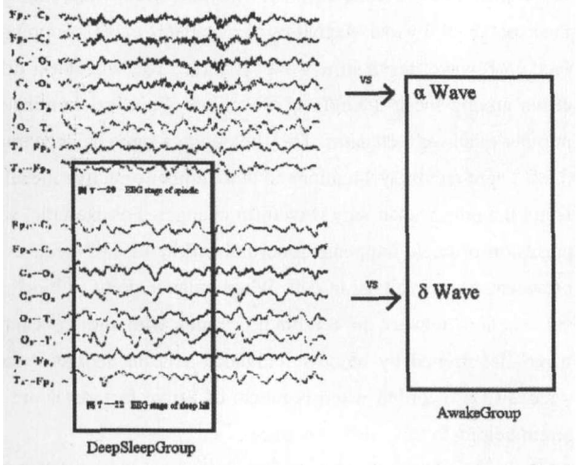

Fig. 8 EEG groups' partition

6.2 Infers and viewpoints under the framework of the model

(1) The fading out of reticular formation activity is a reason why biochemical network switching from balance trace C1 to balance trace C2. Finding out activity of reticular formation→thalamus→cortex path/reticular formation→cortex path bring what biochemical events in cortex is helpful for clarifing the biochemical mechanism of EEG model that have been described.

(2) In balance trace C2, the timing control capability of blood flow has been weakened (Evidences: when in balance traces switching process, dream is chaos; when in stage of spindle, the information processing is so chaos that there is no dream at all). A visual result of timing control capability changing: α wave (awake-EEG-group) is much more regular than wave in stage of spindle (deep-sleep EEG group). This is one molecular reason why "Reticular System is pre-requested for consciousness and awake".

7 Application of model —— Origin of alpha wave

7.1 Phenomena analysis

Base on researches of alpha wave of dog (Lopesda Silva etc 1973,1977,1978,1980), it's known that: (1) alpha wave recorded on optical cortex, and optical-related parts of thalamus (2) alpha wave origin in an equivalent dipole layer. Centre of the layer is base dendrites of pyramid cells from IV, V layer of cortex. (3) For alpha wave recorded in cortex, alpha wave from nearby area (within diameter 2 mm) is more related than alpha wave from thalamus.[6]

7.2 Infers and viewpoints under the framework of the model

Explanation for (2) and (3) of 7.1: Alpha rhythm origin in thalamencephalon. Arrival of artery blood helps conducting alpha rhythm onto the cortex. Because reasons that has been described in 3.3.1, cells in range 2mm are supplied by same capillaries. Getting artery blood at the same time helps synchronous active of these cells, and results in phenomenon (3) of 7.1.

8 Application of model —— Forming of nerve circuits

Biochemical environment differences determined by time difference of blood perfusion arrivals, affect the properties, classes and quantities of synapses when forming circuits, and determined the properties and timing characteristic of kinds of circuits. This implies that there is a new timing control mechanism when brain processing information [1].

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血液循环在大脑信息处理过程中的时序控制作用

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摘要:自 2006 年以来,作者已陆续在一些杂志,网站和学术会议[1-3]上发表一些文章和证据,主要探究如下几个问题:(1)讨论各种脑电波现象的起源,大脑信息处理过程,血液循环三者之间关系。(2)提出了血液循环在大脑处理信息的过程中起到了基础时钟的作用;提出其可能的一个分子机制,其中 H+浓度,O_2浓度,微循环系统的工作机制,NMDA 受体的 H+位点是几个关键因素。(3)大脑如何感知时间的生理机制。提出时间感(或者时间流逝感)其实是一种压感,来源于当血液流经大脑的血管网的时候,造成的压感;而大脑对连续物理过程的感知则是大脑中记录情景(照片)的一个细胞群序列兴奋的信号和压感通过"捆绑效应"整合给人的感觉(即有点像放电影)。在本文中,将对这些观点和证据进行回顾:文章第 1 节在整体上叙述所建立的脑电波模型,和"血液循环在大脑处理信息上具有时序控制作用"的观点。第 2~5 节将从各方面给出支持第 1 节中所叙述模型和观点的证据,并对一些问题进行澄清。第 6~8 节将该模型应用于解释一些脑电波和神经生理现象,解决目前一些问题(例如 α 波的起源问题,睡眠的脑电现象等)。

关键词:过程存储和重组模型;血液循环;脑电波;中枢神经系统;时间认知;时序控制

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热烈祝贺我刊被俄罗斯《文摘杂志》(Abstract Journal,缩写 AJ)收录

2008 年 04 月 18 日,我刊编辑部收到俄罗斯《文摘杂志》(Abstract Journal,缩写 AJ)数据库 Elena Raevskaya 博士的邮件,经过专家评估,我刊适合 VINITI 数据库要求,决定正式收录(通知网址 http://www.cessp.org.cn/u/data100.htm)。

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上述成绩的获得是广大作者和编委会全体成员共同努力的结果,是对我刊质量的一种肯定也是对我们的一种鞭策,编辑部将再接再厉,努力提高工作效率,为广大科研工作者提供一个良好的学术传媒,为提高我国科研水平和科技实力添砖加瓦!

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