UNLOCKING
CONSCIOUSNESS

BRIAN MIND FORUM

Appendix 030

Synapses & Energy

References to Synaptic Tension, and variable width synaptic clefts.

Synapses

Sir Charles Scott Sherrington OM GBE PRS and Nobel Laureate, explored the complex connections between neurons and other organs, and in the early twentieth century named them *synapses* after the Greek word 'to clasp'. In almost all cases the link is affected by a small gap, or cleft thought to be about 20 nano meters wide in its resting state. All electrochemical signal patterns along both dendrites (carrying information to the neural nucleus) and axons (carrying information from the neural nucleus) travel in one direction only. Signals, or action potentials are generated by sodium and potassium ions permeating the membrane in sequence to or from the nucleus. When an action potential arrives at a synapse it generates neurotransmitters which cross the synaptic gap stimulating activity in the target muscle, gland, other organ or, if the target is another neuron, generating an action potential that is then transmitted along that neuron.

We know quite a lot about this actual 'handshake'. How the number of neurotransmitters can vary, how the ambient hormone mix can affect transmission, how other neurotransmitters can bind to the axon to vary activity, and how unused excess neurotransmitters are cleared away after a transmission. The strengthening of synaptic links with use has been observed, suggesting this is part of memory enhancement. It is thought that synapses operate in the Theta band around 4 to 8 HtZ.

Analytical logic suggests that there is an evolutionary reason for this complex process. As a general rule evolution seeks to find simple solutions. Why this complexity? One answer may be that an axon stimulating a dendrite as part of a network to carry out some activity like running, bicycling or gymnastics for instance can move an axon along the target dendrite and so minutely fine tune the timing of the target activity. This seems to be a relatively minor benefit for such a complex process.

In 2008 we published a much more significant hypothesis, suggesting that synapses play a major role in the gradient of being awake; from deep anaesthesia, deep sleep, REM sleep, day dreaming up to full concentration. [The Physical Foundations of Consciousness: Brain Organization: The Role of Synapses. http://arxiv.org/abs/0907.2192. Physical Foundations of Consciousness: Variable Width Synapses'. Neuroquantology Vol 10 no 2 p268-275

http://www.neuroquantology.com/index.php/journal/article/view/544. Biological Systems of the Brain. http://www.troubador.co.uk/book_info.asp?bookid=671]

We asked two questions. What force holds the synaptic clefts in close proximity but apart? Are synaptic gaps fixed or variable? We suggested the implications are significant because, if the forces maintaining the gaps are variable causing the synapses to be in a perpetual state of variable tension, then it follows that the width of the gaps will vary. Lower tension will cause the synaptic clefts to widen, and vice versa.

Variable width synaptic gaps will have a significant impact of the passage of neurotransmitters across the synapses. Maximum tension, shortest gaps will facilitate the most efficient transmission of signals. Conversely minimum tension, widest gaps will enable the passage of signals but at the lowest level of efficiency.

This provides us with an infinitely sensitive process to determine all the stages along the gradient of wakefulness from deep anaesthesia to full concentration.

If the tension across the synapses is determined by the strength of available energy in the neurons then that would explain the well-established observation that as we grow tired either by the normal passage of time in the daily cycle, or as a result of responding to a crisis, we become drowsy (the transmission of signals begins to slow) and we fall asleep (signals are still transmitted, but at a minimum level to enable only 'crisis' messages to pass) As the neurons replenish their energy levels during this period of sleep, so the synaptic tension rises, the synaptic clefts gradually close and on reaching a critical mass, the passage of electrochemical signals is re-established at its normal level and we experience waking up.

There are a number of advantages to this hypothesis. It accounts for the phenomena of the gradual analogue gradient of sleep to wakefulness.

It accounts for the well observed facility that we can focus our attention on a particular activity, a particular group of neural networks, and be able to switch from that activity to another quickly and easily.

Concentration is achieved by generating the maximum tension causing the narrowing of the synaptic clefts and the maximum transmission facility, which again can control the whole brain or just selected parts.

It accounts for the phenomenon where a sharp blow to the head literally knocks apart some or all the synaptic clefts causing an immediate loss of information transmission and awareness.

It contributes to the explanation of dreaming and sleep walking which we describe elsewhere.

Awake, conscious; asleep, unconscious

Over the last couple of years we have been studying the implications of the fact that everyone conflates consciousness with being awake. We suggest we should review this convention.

Therefore, we have carefully used the words 'awake' for the high tension, minimum width gap state, and 'asleep, for the low tension, maximum width gap.

Conventionally the pair of words 'awake' and 'conscious' tend to be used almost as synonyms; similarly 'asleep' and 'unconscious'.

Consciousness is much more complex and is the word we use to cover all the many experiences of being alive, of being hungry, thirty, angry, frightened, aroused……. of being alive, of being a person and aware of everything that is happening around us in the present, and much of what has happened to us in the past.

Being fast asleep – hibernating almost; being drowsy, or concentrating furiously is a fundamentally different process.

Unconscious is not a synonym for being asleep. There is plenty of evidence that the electrochemical activity in the central nervous systems and the brain in particular is continuous and generating glandular activity from very early in the development of the foetus, and ceases only with death. We know for certain that we can be conscious and fast asleep. For example: we can be fast asleep, yet if someone calls our name, even whispers it, we can be instantly fully awake. If someone shouts fire, we 'hear' that warning and respond however fast asleep we were.

The cycles of the awake, asleep gradient and the processes of consciously feeling the emotional sensations of past and present experiences are quite separate. It is equally true that the state of being awake is equally different from the monitoring, timing and feedback processes associated with initiating conscious activity, self-control and decision making. [Brain faster than c'ness Howard Poizner U of C San Diego ASI AM Sep 16 p61]

We discuss the implications of this separation of powers and functions in chapter 5.

What evidence do we have to support the theory of variable width synapses?

Daniel Bushey, Giulio Tononi and Chiara Cirelli (Science. Vol 332, 1576- 1581; June 24, 2011) argue that the chemistry of the brain leads to synaptic weakening. They have identified a group of neuromodulators (currently in Drosophila) possibly including a substance they call Brain Derived Neurotrophic factor (or BDNF). They argue that "the evidence is strong that synaptic strength goes up during wakefulness and down during sleep". They propose that this cycle evolved to provide a resting state to restore energy levels and process signals received in the previous 'waking' period which they call synaptic homeostasis. They do not suggest what initiates and drives this cycle. This hypothesis would be complete if the reciprocal were true, namely if falling energy levels initiated falling synaptic strength. Synaptic homeostasis is the cause of sleep not the result.

Yan Xu and his team at the University of Pittsburgh have developed a model that reproduces an electrical signal arriving at a single neuron and stimulates the resulting signal. They then measure the value that changed the ease of communication

between nodes (across the synapse?) giving this the value of ***p*** between '0' and'1'. By modelling a visual image, they showed it is transmitted across the network at various levels of ***p***. When ***p*** = 0.32 only a barely discernible picture was generated, whereas if ***p*** is raised to 0.38 a clearly recognisable image was perceived. (Phys. Rev. Lett. 115 108103)

In 2013 Lulu Xie and her colleagues at the University of Rochester Medical Center reported that the space between cells in the brain increases during sleep.

Energy

An alternative mechanism other than physical separation has been suggested by Luke Robinson.
He draws attention to the possibility that any variation in the resistance/signal strength across the synapse need not depend on physical separation?
http://www.antanitus.com/hypothesis.
The following is proposed for head trauma:
"Equally mysterious are the mechanisms producing unconsciousness resulting from minor head trauma insufficient in force to cause any detectable injury. Mechanical perturbation has been shown to precipitate calcium waves in vitro (20). A blow to the head results in a mechanical compression wave traveling through the brain. This mechanical force could be sufficient to produce a pattern of widespread sequential calcium waves that reflect the shape and velocity of the mechanical compression wave. The astrocytic calcium waves so produced would be unrelated to, and for a while unresponsive to the influence of, normal sensory input. Meaningful interactions between astrocytes and synapses could be overwhelmed by the disruptively nonsensical mechanically induced calcium wave patterns. Despite the inability of the mechanical force to produce macro or microscopic injury, the brain -- the person -- would be "knocked out" or temporarily unconscious."

Myelin Cannibalism

Roberta Brinton: University of Southern California.

Shortage of glucose initiates a 'starvation response'.

Neurons catabolise the fat rich myelin that insulates axons and dendrites, reducing efficiency of the signal taffic.

Notes

Sir Charles Scott Sherrington OM GBE PRS was an English neurophysiologist, histologist, bacteriologist, and a pathologist, Nobel laureate and president of the Royal Society in the early 1920s. Wikipedia

Born: November 27, 1857, Islington, London: Died: March 4, 1952.

Theta Band 4-8 Htz

4 feet per second

Estimate:

Synaptic gap in resting state 20 nanometers.