Consciousness During Sleep: Alcohol Intake Experiment Proposal

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Introduction

For a couple thousands years, the questions surrounding human consciousness – the very essence of our being – have been the field of philosophers from Plato to Descartes to Spinoza. Scientists have studied the evolution, the mechanisms and function of the brain, but have difficulty playing apart the complex processes that give rise to the human consciousness mostly because it is difficult to measure and evaluate individual subjective experience, but it is however possible to do so. Consciousness can be viewed and defined as simple awareness that includes perception and emotion (Johnson-Laird, 1983).

It is the arena of self-knowledge, the ground of our individual perspective, and the realm of our private thoughts and emotions. Until recently, most brain-based theories of consciousness have engrossed on how neurons make a magic internal experience and how the magic emerges from the neurons. It has commonly been proposed that mind and consciousness are mixtures of neuronal activity and arise from brain activity. This is similar to how light arises from a light bulb, but is not the same as the underlying processes taking place within the light bulb (Penfield, 1937).

A number of different theories have been proposed to make up for this phenomenon. Precisely, it has been proposed that consciousness may arise where brain cells connect together, through a synchronous activity of brain cell networks in the brain, and as a novel property of computational complexity among brain cells. Evidence to support these theories have come from the observation that specific changes in function such as personality and memory are assosciated with specific brain injury, for instance people with specific head injuries or head tumors may lose some of their consciousness. Moreover, scanning devices, such as MRI, and PET scanning have also revealed a relationship between activity of brain cells and different mental states. All of this has required investigators to relate brain events to reports of experience and to research on cognitive processes (Flanagan, 1992). In truth, the facts of consciousness are all around us. All psychological findings involve conscious experience. For example, dreaming has captivated humankind for a long time. For most of the twentieth century, scientific dream theories were mainly psychological. Ever since the discovery of rapid eye movement (REM) sleep, the neural underpinnings of dreaming have become more and more well understood, and we are now able to match the details of the brain mechanisms with a theory of consciousness that is derived from the study of sleep and dreaming (Hobson, 2009). It has always seemed remarkable to me that the ingestion of a single substance can have such complex effects on consciousness. Alcohol does it, in part, by promoting the effects of inhibitory neurotransmitters and suppressing the effects of excitatory ones, while also devastating a nice surge of dopamine. This ruins the coordination and can lead to loss of memory, and indeed consciousness (Han, 2012).

Summary of Extended Paper

A study I found completed by J. Allan Hobson presents a critical overview of conceptions of consciousness, relating it to specific areas of the brain and their chemical and physical states. Hobson, Professor of Psychiatry Emeritus at Harvard University and author of several books, is the undisputed celebrity of this scientific outlook. In his 30 years of work, Hobson is also perhaps the greatest provocateur in the field of dream studies, stirring up old philosophical conflicts such as the value of objective science over experience, and mechanism over meaning. He is most known for his break out 1977 paper where he declared victory over Freud by reporting his discovery that dreams in the REM state form by grace of neurochemical changes in the brain. In the study I found, his theory emphasizes data that suggests REM sleep is set up from a conscious state, giving a virtual reality model of the world that is of functional use to the development and maintenance of consciousness. In this study, the brain states underlying waking and dreaming work together in consciousness showing that their functional interaction is important to the best functioning of both (Hobson, 2009).

Looking at previous relevant work explained in this study, Giuseppe Moruzzi was the first to suggest that sleep facilitates processes in the brain. Many studies showed that REM sleep duration is increased when humans learn a new task and that REM sleep deprivation obstructs with learning. Therefore, sleep is crucial to memory, and to act as a unified system, neurons need to be coupled to each other and activated in temporal and chemical unity. This is what REM sleep does, through its functions, on which consciousness depends. When awake, our brains have access to information about the external space and time, however, these are not obtainable in sleep, so they have to be stimulated while dreaming. From this and the study, I found, Hobson proposes that the REM-sleeping brain has built-in predictions of external space and time that are later adjusted from experiences of the outside world with the act of three different factors (Hobson, 2009).

Hobson's methods for his study included three factors. To begin with first however, the control of waking, REM sleep, and NREM sleep states lies in the brainstem. This composes of events in the forebrain and in the spinal cord. This system of consciousness states has been cut down into three factors: activation (A), input-output gating (I), and modulation (M). He then describes these factors in detail. Input-output gating (I) signals are an important part of REM sleep and are also called PGO signal waves. The activation of this system in sleep supports the idea that the brain simulates its interactions with the external world. Moreover, during dreaming, PGO signals are used for visual imagery so this is also an important part of consciousness. Modulation (M) are neuromodulators that release chemicals on the brain, and the shift of activity of two neuromodulatory cell populations determines the shift from predominantly external input to internal input. Lastly, activation (A), are inactive in NREM sleep and reactivated in REM sleep. The level and quality of consciousness are functions of internal brain activation and deactivation, which are controlled by the neurons in the brainstem (Hobson, 2009).

With these factors, he used the AIM model. It described different states of the brain and showed how they fluctuate over a course of a night in normal control adult humans. By choosing activation, input source, and mode of neuromodulation as his three dimensions, Hodson selected how much information is being processed by the brain (A), what information is being processed (I), and how it is being processed

(M). With brain imaging technology, his results were obtained for activation as it included the electrical output of the brain's surface. It was discovered that the normal control had an overall deactivation of the brain during NREM sleep, and a reactivation during REM sleep. The cortex was activated when the electroencephalogram (EEG) showed a low-voltage fast brainwave pattern. This means that it denoted a good rate of information processing. The strength of input-output gating was measured by quantifying postural muscle tone (EMG) and eye-movement activity (EOG) and these showed high strengths too where sensory input and motor output are slowed down during REM sleep. This means that signals from the brain to the body are cut off and we are paralyzed during REM sleep. The last factor, modulation, determines the way that information is processed, whatever its source. The strength of this determines whether the mind keeps a record of its conscious experience, or in other terms, memory. Hobson measured this in the ratio of cholinergic to aminergic neuromodulator release using functional MRI. These two systems switched in waking and dreaming, with aminergic systems dominant in waking, and the cholinergic system dominant in dreaming. In other words, in REM, the aminergic (serotonin) inhibition is shut off and the cholinergic (acetylcholine) system is at its highest peak (Hobson, 2009).

With this in mind, activation of the normal control brains are at its highest during waking, and lowest during non-dream sleep. In other words, the gates for sensing the outside world, and the gates that allow input for the body to move from the brain to the body are the highest during wake and the lowest during dream sleep. Normal controls are able to process external data from the open sensory input and motor output gates and the suppressed internal stimuli. In dreaming, input-output gates are closed, and the internal stimulus generation is increased. The PGO system is therefore turned-on, producing input of fictive visual and motor data from the bodies to the occipital cortex. Therefore, the parts of the brain that deal with motor movements and sense data are switched on, thus normal controls are able to dream about movement and sensorial scenes. The third change, the neurochemistry of the brain changes from wake to sleep to dream-sleep. As we go to sleep, the aminergic inhibition loosens its control slowly and the cholinergic system slowly gains in strength. Consciousness depends on all these factors to act normally (Hobson, 2009).

Proposed Experiment

The experiment I am extending is from Hobson's study on all three factors relating to sleep and consciousness. I hypothesize that humans who consume alcohol before going to sleep will have different results for each factor affecting consciousness, compared to the normal control group in his study. The design of my experiment will be a group of people who are exposed to alcohol and then go to sleep. It will show how alcohol affects brain function by interacting with multiple systems thereby affecting consciousness. The methods will be that once the group has fallen asleep, brain imaging technology will take place and using Hobson's AIM model, I will compare the normal control with the alcohol intake group. The AIM state-space model will show transitions within the AIM state space from waking to non-rapid eye movement and then to rapid eye movement. Alcohol intake will hopefully show that there are low activation, low input-output gating, and reversed modulation values affecting consciousness in the brain.

Damage or dampening of activation to a variety of particular brain areas will cause dampening of conscious activity (Hobson, 2009). In general, during REM, the normal control brain shows waves similar to

waking; low-voltage, and fast pattern. Specific brain areas that have been shown to be active from brain imaging techniques on normal control are in the upper part of the brain during dreaming which is part of information processing. Large parts of the brain, such as subcortical and neocortical areas, that are active during waking, are inactive during NREM sleep and are reactivated during REM sleep (Gann, 2004). Evidence to show that there will be a low activation in the cortex when consuming alcohol before sleeping can be found from a study performed by Roehrs and Roth. When they compared EEG readings of various sleep stages, they evaluated the frequency of brain waves and the amplitude of brain waves from intoxicated subjects. During REM sleep, cortical EEG readings show slow-wave sleep with a frequency of 0.5 to 2.0 Hz which is what is usually seen in NREM sleep (Roehrs, Roth, 2004). This evidence and EEG readings will show low activation values for my experiment with intoxication while sleeping. This would affect consciousness because hardly any information would be processed as REM sleep would be acting like NREM sleep.

The second factor, input-output gating will be less in value when humans are exposed to alcohol than the normal control. This factor in my experiment will be measured by EMG postural muscle tone and EOG, eye-movement activity. Sensory isolation during REM in normal control groups is what is seen. The source of this is in the brain stem, the pontomedullary reticular formation that hyperpolarizies the motorneurons. This means that the body is less responsive to instructions from the brain to act. Loss of muscle control is also seen which is from tonic postsynaptive inhibition of spinal anterior horn cells by the pontomedullary reticular formation (Hobson, 2009). When there is an imbalance in the brain stem due to intoxication, people will still experience REM sleep, but it is however shortened. In the same study as previously mentioned, the EMG during REM sleep was reduced to its lowest level for the night. Therefore, it would be evident that most voluntary muscle groups are paralyzed when intoxicated as well, because certain nerve cells in the spinal cord, or motor neurons, do not respond to nerve signals. In one study, the nature of the signals internally that arise as PGO signals during REM sleep show how the system brings up sensorimotor integration. The activation of this in sleep tells us that the brain simulates in interaction with the external world. In their alcoholic patients, PGO waves did not encode eye movement that are commanded by the brain stem (Roehrs, Roth, 2003). In my experiment, EOG will show little or no PGO signals used in the construction of hallucinoid visual imagery of dreams which is an important element of consciousness. Moreover, REM sleep would be shortened, so there is less time for information to even be processed.

The last factor is modulation, which is the strength of chemical systems modulating the brain. For Hobson, this was the ratio of cholinergic to aminergic neuromodulator release. In REM, these two switch in waking and dreaming. The aminergic system is dominant in waking and the cholinergic system is dominant in dreaming. These two systems modulate cognition, attention, decision and insight. It is this function that helps the activated brain to select, hold, and evaluate its representations when we are awake (Hobson, 2009). I hypothesized that alcohol intake before going to sleep would lower the strength of modulation or memory. The dreaming brain has its own neuromodulatory system that involves the shutdown of the aminergic system and the activation of the other system. The thalamus and amygdala are cholingerically modulated and the cortex is aminergically demodulated, in terms of dampening recent memory and orientation (Valenzuela, 1997). Evidence shows that aminergically demodulated and

cholinergically hypermodulated is the same as what occurs in a shutdown brain. In one study, alcohol dependent participants were examined in a sleep laboratory. They found that their REM sleep pressure was increased due to an impaired aminergic. Their data shows that alcoholic patients exhibited an increase in REM pressure. The pronounced abnormalities in sleep parameters in alcoholics were due to an enhanced activity of cholinergic neurons, and impaired aminergic neurotransmission (Davis, Fiebre, 2006). In another study, alcohol-induced sleep in mice was performed to see the effect of cholinergic alteration. They saw that the apparent central cholinergic antagonism of alcohol sleeping time suggested that a part of alcohol's central depressant action is to do with the inhibition of acetylcholine function, which is opposite of normal controls (Erickson, Burnam, 1971). Therefore, this evidence and more brain imaging technology would show the ratio of aminergic to cholinergic release opposite that of the normal control group in Hobson's study. Both the brain stem reticular activating system and cerebral cortex implicate as sites of action of alcohol, and alcohol as a depressant would block the function of central neurons in these systems (Hobson, 2009). My experiment would show how alcohol in the modulation factor inhibits acetylcholine release at central cholinergic synapses. This would decrease consciousness since this affects how information is being processed. Evidence again for this would be looking at the blood flow changes measured with positron emission tomography and functional MRI.

With this being said, these are all possible outcomes of my experiment based on the evidence found from studies and other research. The evidence required to prove or disprove my hypothesis that intoxication affects the brain consciousness differently from no intoxication would be from brain imagining technology as mentioned above for each factor.

Summary and Discussion

Consciousness is the great, and confusing crucial point of cognitive science. We are all conscious beings, but consciousness is not something we can look at directly, so scientists wish to collect objective knowledge even about subjectivity itself. In the study I found from Hobson, the major point of consciousness while dreaming and sleeping are that there is more to the dreaming brain than just activation of areas, as there is also information gating, and neuromodulatory control. From his study, when we go to sleep, the brain becomes deactivated to the outside world and sensations and switches over from an aminergic neurochemical system that keeps us alert and focused on the outer world to a cholinergic system that allows for relaxation. Then, the aminergic system stops and the cholinergic system becomes hyperactive. During this time, many parts of the brain become active, the body becomes rigid, and we begin to dream, and while dreaming, it is functioning in the same way as waking, but the inputs and outputs to normal feedback are missing or dampened (Hobson, 2009).

In my proposed experiment, I hypothesized that intoxicated patients during sleep would have different factor values in the brain compared to the normal control group in Hobson's study. The activation in the brain would be missing or dampened, the input and output gating would be lower in value with REM sleep shortened, and the ratio of aminergic and cholinergic system would be reversed. All evidence to back up my hypothesis would come from brain imaging techniques mentioned earlier. Again, activation values would be measured by electrocephalography. It would record the electrical activity along the brain measuring voltage fluctuations and frequencies. Since consciousness relies on communication between different brain areas, EEG would show the distinctive brain wave patterns when con-

sciousness is lost, offering easily identifiable markers for this impairment. Electromyogram signals would detect muscle tone and electrooculogram signals would detect eye movements for the next factor. If both of these are low in value, then it would support my hypothesis of lower input-output gating. For modulation, I hypothesized a low value of modulation where the acetylcholine is dominant. The evidence to support this hypothesis remains in previous research and to be seen whether the blood flow changes measured with positron emission tomography and functional MRI reduce to the changes of value M in the AIM model or not.

To end, my experiment would be informative to both scientists and to other people outside of science. Current research suggests that alcohol affects multiple neurotransmitter systems in the brain affecting in the long term, consciousness. Virtually all brain functions depend on a delicate balance between systems. In my experiment, scientists will find my findings informative on how alcohol affects which system. More importantly, a detailed understanding of alcohol's mechanism of action in the brain is a prerequisite to discovering effective treatment for both alcohol abuse and alcoholism in the long run. Such an understanding could also lead to more effective treatments for alcohol dependence, if they know which parts of the brain consciousness are being affected.

Alcohol use is laden with a myriad of mixed judgments, creating such confusion over its use that most people summarily dismiss this lesson. People drink alcohol for many reasons, but inevitably want to feel better. They want to avoid or deny experiences, they drink to relax, socialize, to forget. They drink in order to reduce some sort of pain. Drinking is always an attempt to feel better emotions than the ones they are feeling. Consciousness, however, is gained through each emotion, good, bad, pleasant, and unpleasant. If one is drinking, in order to feel better, they are trying to manipulate their emotions, thereby the process of growth is thwarted. If people knew how alcohol affects the brain consciousness, they might be less likely to drink since there would be evidence. Showing which parts of the brain are affected would be informative to them as losing consciousness can lead to brain damage.

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