Heading: Sensory Deprivation in Humans, Mice, and History

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Abstract

This paper will highlight a study done in lab mice, to observe the effects of sensory deprivation on spinal dendrites. Light will be given to the history of sensory deprivation chambers, focusing on John C. Lilly and his work, as well as his findings in the effects of these chambers on people. The writer’s own theory will be proposed, based off of an analyzed case study, centered around the hearing loss of an older woman. This case study involves the observation and treatment of a woman who claimed to hear voices after hearing loss due to a car accident. Time is given to defining the use and structure of vibrissae, in order to highlight their importance in the spinal dendrite study.
History of Sensory Deprivation Chambers

Little was done in the study of sensory deprivation, through the means of actual tests in human subjects, until a means in which to test people, and theories, was developed by John C Lilly. For a long time, people theorised outlandish possibilities of what would happen when someone was deprived of nearly all their senses. Some believed you could contact other worlds, while more grounded thinkers assumed you would simply be put into a higher state of concentration, able to theorize, think, and ponder more efficiently. Many people were tested, and while few spoke of traveling to other worlds, most stated experiencing what many know as an, “out of body experience”. Feeling disconnected from their bodies due to an absence of sensory input to let the brain know we are even still alive, we begin to feel detached. Studies done by Lilly resulted in findings that showed just 15 minutes of near-total sensory deprivation was enough to trigger vivid hallucinations in many of its test subjects.

"Somewhere, deep within the brain, was a mechanism capable of generating internal experiences completely independent of the outside world, and this settled the issue of what happens in profound physical isolation. The mind does not pass into unconsciousness, the brain does not shut down. Instead, it constructs experience out of stored impressions and memories. The isolated mind becomes highly active and creative. This was the principal discovery that Dr. Lilly reported in his first three scientific papers on the isolation tank research, published in 1956, 1957, and 1958."
Defining Sensory deprivation

It is necessary, that when discussing or reading a topic of interest, a person should begin with a basic and concrete understanding regarding the foundations of the subject matter. Sensory deprivation is defined by Dictionary.com as, “The experimental or natural reduction of environmental stimuli, as by physical isolation or loss of eyesight, often leading to cognitive, perceptual, or behavioral changes, as disorientation, delusions, or panic.” This definition would not be found to be all inclusive, however, one would do well to use it as a cornerstone from which to build off. Firstly, when reconstructing this definition for purposes of application, I would like to start off on the absence of recognition in regards to auditory deprivation. In this paper you will learn about a woman who experienced some effects of what is presumed to be sensory deprivation, due to the loss of her hearing.

Following this same path of definitory reasoning, the other senses need to be included in a fully working definition as well, as this is “sensory” deprivation; sensory being an all inclusive word. In a study that will be included in this paper, and will comprise an extensive amount of this writing, mice were forced into a form of sensory deprivation by the removing of their whiskers or, “feelers.” With this in mind, the definition should also be sure to include, whether directly or indirectly, the loss of tactile sensory.

The Vibrissae

The aforementioned whiskers are also known as “vibrissae”, and are a type of mammalian hair that are typically characterised anatomically, by their large size, as a well-
innervated hair follicle, and by having an identifiable representation in the somatosensory cortex of the brain.

They are specialized for tactile sensing acting much like skin or feelers. What however makes them different from other hairs and are they different at all? There are numerous differences. The vibrissal hair is usually thicker and stiffer than other types of pelagic hair, but, like other hairs, the shaft consists of an inert material called keratin, and contains no nerves. Contrastly, if these vibrissae have no nerves, how can they be used for tactile sensory? The answer is that they grow from a special hair follicle, incorporating a capsule of blood called a “blood sinus,” which is heavily innervated by sensory nerves.

Vibrissae in Application Studies.

Now that we know their function, let us look at why these whiskers are so pivotal. It is found that rats and mice have approximately 30 macro vibrissae on each side of the face, with whisker lengths up to around 50 mm in laboratory rats, 30 mm in laboratory mice. Thus, an estimate for the total number of sensory nerve cells serving the mystacial vibrissal array on the face of a rat or mouse might be 25,000. This number in relation to the number of sensory neurons in the body of a mouse or rat is astonishing. In order to better understand the extent of the deprivation the mice were put under, you can imagine this as the equivalent to cutting off both of your hands. Now with a more comprehensive and operational understanding of the premise of the research portrayed through this medium of writing, we will begin looking deeper into sensory deprivation in lab rats and mice, and eventually, in people.
Prevention of Dendritic Spine Loss in Mice via Sensory Deprivation

To begin, an understanding must be had of the spinal dendrite, and thereby the loss of synapses that is experienced by mice as they age. Experience plays a key role in the modification of synaptic activity, however, it is not currently understood how it relates to the nearly life long synapse loss in mice (the continued loss of synapses occurring naturally throughout the aging process.). What is known for sure by scientists however, is that synaptic loss is apparent throughout the life of laboratory mice. It has been discovered that sensory deprivation through the medium of whisker trimming, decreases the number of synapses lost throughout the life of the mouse, an inquisitive and seemingly contradictory discovery.

The typical percentage of spinal synapses lost in humans and most mammals is on average 50%, and takes place throughout the entire lifespan of the animal, primary in the adolescent stages. When the vibrissae are trimmed, causing near complete tactile sense deprivation, the loss of synapses is greatly decreased, almost to a stop. However, if a mouse is put through adolescence in a deprived state, and then has its senses restored in adulthood, the synaptic loss is sped up incredibly. This indicates that the role of experience in synapse loss is one of importance, but not yet clearly defined. It is also found that Piaget's idea of learning rebound is found to be present, and seemingly related to the occurrence of a rapid loss in dendritic spines, soon after sensory recover.
Experiment Results and Findings

It is found in the experiment, *Long-term sensory deprivation prevents dendritic spine loss in primary somatosensory cortex*, in control mice one month of age, that the number of spines eliminated over a two week period was significantly higher than the percentage formed. To determine the effects of the sensory deprivation on this spinal dendrite loss rate, mice from four weeks of age to six weeks of age had their whiskers trimmed on one side of the facial pad. The number of spines generated and eliminated compared to the "non-deprived constant growth vs, non-deprived dendrite loss ratio", was far different; not speeding up the growth, but simply slowing the loss. There was no significant change in the number of spines formed. As mentioned previously, once adulthood is reached, (which in this case is defined as 4 months and beyond), the loss of dendritic spines is significantly reduced. However, when tests on adults went from only two weeks with little results and were bumped up to 2 months, a significant reduction in spine reduction was found.

To determine whether spine loss would rebound after reintroduction to tactile sensory, whiskers of two month old mice were trimmed for two weeks, and allowed to regrow the next two weeks. It was found that during these two weeks, dendritic spine loss exceeded that of the control group rate (natural spinal loss rate). Similar results were found when trimming was lengthened to four weeks, and grown and tested over the next two.

-Below are three charts, two concerning the elimination of the Filopodia, which will not be included in this paper, while the first shows the ratio between sensory deprived mice and non-deprived, and their percentage of spine loss.
Effects of Sensory Deprivation in Humans

The Case

A 63 year old woman complained that she was hearing voices for the past 1 1/2, to 2 years, and that the presence of voices have been increasing recently, especially at night. Initially, the voices frightened her, and she thought she was going mad; then she realized they were not real. She had hearing loss in her left ear following a car accident 5 years ago, and it was recommended that she use a hearing aid. Due to the fact that the aid disturbed her ear, she used it irregularly for 3 years and not at all thereafter.

Five to six months later, she started to hear voices. Everything was normal in the patient's psychiatric examination except for auditory hallucinations and anxious mood. There was no substance use or psychiatric illness in her history, and a complete blood count, biochemical tests, thyroid function test, EEG, cranial computed tomography scan,
and neurological examinations of the patient were all normal. The, “Minnesota Multiphasic Personality Inventory and Beier Sentence Completion” tests were evaluated as normal.

**Treatment and Procedure**

“Treatment was started with olanzapine 10 mg daily and diazepam 10 mg daily. Later, diazepam was stopped and continued by olanzapine 5 mg daily only. On the tenth day of treatment, the auditory hallucinations disappeared. The patient was observed for 10 months at regular intervals; she took olanzapine 5 mg daily and used a hearing aid for 7 months. During this time, no psychopathology had been detected.” (Canadian Journal of Psychiatry pg. 3)

**Phantom Extremity Phenomenon**

An important question must be asked here. How does a loss of hearing in only one ear, create such a case as hearing voices? There have been countless cases like this one, and the majority do not lead to something as fantasy like as hearing voices. There is however, another phenomenon in concurrence with sensory deprivation, and that is known as the, “Phantom Extremity Phenomenon.” Many have heard of phantom pains, left behind with the loss of a limb either through amputation or other means. Damage to the sensory nerve can affect reuptake, and other components of the sensory neurons, creating a feeling that the limb is still there, throbbing in pain. This operation is similar to what is happening in this case of lost sense of hearing in one ear. In both cases the stimuli going to the cortex is
blocked, but it is still unknown as to how this perceptual disorder occurs. There is a theory that this is caused by receptor hypersensitivity.

“This could be explained by examining the effective mechanisms of hallucinogens and atypical antipsychotics. Hallucinogens and serotonin-dopamine antagonists (SDAs) act through the same receptors but create different effects. Hallucinogens activate $5HT^{2A}$, $5HT^{2C}$, and $5HT^{6}$ receptors and cause hallucinations. In contrast, SDAs block these receptors and prevent the formation of hallucinations.” The parameters for the Charles Bonnet Syndrome are all met with this specific case study. However, it was initially discarded as a possible answer, simply because CBS deals, or has only dealt in the past, with visual hallucinations, not auditory. However, because it was proven through multiple tests that the woman was psychologically sound, carried no disorders, and recognized the hallucinations as imaginary among other pivotal criteria, the case may prove to be what is needed to expand the working definition of CBS, to include auditory hallucinations as well, and potentially even other sensically based hallucinations.
Bringing it Back to Lilly

When we look at this case, as we looked at the results produced from sensory deprivation chamber tests done by Lilly, we can begin to theorise on our own, using Lilly's data as a basis. It can be reasonably inferred, though not scientifically prove, that perhaps the cause of the woman's imaginary voices was much like the hallucinations having been reported in S.D.C tests. Due to a lack of sensory input to the cortex for an extended length of time from the damaged ear, the brain created it's own sensory input in an attempt to fill the void. Much like hallucinations caused in the deprivation chambers, brought on by a lack of sensory stimulus, the mind could then create its own stimulus, causing voices to be, "heard," in the damaged ear,

It is is scientific knowledge that the brain is the control center of the body, making sure all systems operate efficiently, and even sacrificing organs to save itself. The brain can be called a control freak, as it dominates all other organs, assuring the body runs the way the brain intends it to; mending errors when needed, and maintaining homeostasis. For these reasons, in an attempt to, "restore balance" to the woman's damaged body, the brain created auditory stimulus that seemed to come from the damaged ear, in order to, as stated, "fill the void".

This is, of course, merely a personal hypothesis only loosely supported by case study evidence. However, one could find there to be merit to this theory, and there may already be research available to substantiate this claim. Many opposing facts are present, one of the most looming being cases of the blind. If there were to be a case of hallucination due to the sensory deprived, it certainly would have been heard more of in those who are blind. This
however does not include those born blind, nor perhaps even those who went blind at a young age as the spinal study in mice may support; but those who lost their eyesight later in their life will be an interesting place to start in search for validation, or destruction of said theory.
References


