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Functional and Neural Mechanisms of Out-of-Body Experiences: Importance of Retinogeniculo-Cortical Oscillations

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Abstract

Current research on the various forms of autoscopic phenomena addresses the clinical and neurological correlates of out-of-body experiences, autoscopic hallucinations, and heautoscopy. Yet most of this research is based on functional magnetic resonance imaging results and focuses predominantly on abnormal cortical activity. Previously we proposed that visual consciousness resulted from the dynamic retinogeniculo-cortical oscillations, such that the photoreceptors dynamically integrated with visual and other vision-associated cortices, and was theorized to be mapped out by photoreceptor discs and rich retinal networks which synchronized with the retinotopic mapping and the associated cortex. The feedback from neural input that is received from the thalamus and cortex via retinogeniculo-cortical oscillations and sent to the retina is multifold higher than feed-forward input to the cortex. This can effectively translate into out-of-body experiences projected onto the screen formed by the retina as it is perceived via feedback and feed-forward oscillations from the reticular thalamic nucleus, or "internal searchlight". This article explores the role of the reticular thalamic nucleus and the retinogeniculo-cortical oscillations as pivotal internal components in vision and various autoscopic phenomena.

1. Introduction

Consciousness is a term used widely throughout neuroscience and philosophy, yet there

lacks fundamental accord on the meaning of consciousness within and between the two. Francis Crick suggested that the brain had an internal "attentional searchlight" that intensified what was already partly visible [1]. Another accepted explanation is that consciousness is the observation of the intrapersonal space—represented by a first person perspective identified as the "self", created through "entanglement" of "sensory information with the current mind-state within an inner mental space" [2]. We contend that the intrapersonal space is not created from entangled sensory information, but rather it is created by information being received and processed in an organized manner.

Previously, we proposed that retinogeniculo-cortical oscillations continuously communicated with the retina and the brain [3]. This constant communication brings the faculties of the brain to the retina, allowing images to be processed in the brain immediately upon entering the eyes, and forms the infrastructure of the visual consciousness space [3]. This process creates the final images we perceive as vision, and creates the final images when we all have grown up believing it is the external world.

The theory presented in this article is that the cornerstones of this internal infrastructure of the intrapersonal space are the retinogeniculo-cortical oscillations (e.g. delta, theta, alpha, beta, and gamma waves) and corticothalamic activity [4]. Together, they form an internal visual space that replicates highly processed information that has been received from the periphery about external events and stimuli. This in turn provides us with an internal representation of the external world [5].

Our review of literature included searches for the visual etiology of autoscopic phenomena, but was unable to find any literature explaining how these hallucinations were projected into the visual field. In our review of literature, we found that studies primarily focused on the "what" but not the "how" in hallucinations experienced in cases of autoscopic phenomena. Current literature uses evidence from MRI and EEG studies to explain that damage to certain brain regions prevented multi-sensory signal integration which may be at the root of these hallucinations, but there was no mention of the roles thalamic nuclei played in autoscopic phenomena, or how abnormal cortical activity could transmit incorrect information into the retinogeniculo-cortical oscillations resulting in false information reaching the retina and become re-projected into the external visual field

2. Lateral Geniculate Nucleus

The brain's subcortex is an important component in information exchange throughout the cortex. Regulation of visual information between the retina and cortex is managed via the lateral geniculate nucleus, pulvinar nuclei, and superior colliculus [6]. The retina provides the geniculate relay cells with only 10% - 20% of presynaptic connections, with the remaining input coming from other feedback inputs, including the reticular formation and cortex [7]. Since the retina is concave, the area of the periphery of the retina is greater than the central area, and the retinal ganglion cells are densely packed in and near the fovea; however, there is an even distribution of neurons in the lateral geniculate nucleus and primary visual cortex [7]. The lateral geniculate nucleus is responsible

for sending information to the cortex, and is also the place where 90% of retinal axons terminate [7].

In humans, spontaneous retinal activity is generated by retinal neurons before vision takes place, creating functional maps in higher visual areas. Retinal cells oscillate at alpha rhythms and synchronize with the visual and parietal cortices, which form the infrastructure for visual consciousness that is formed through an "internal" neural space. This "internal" neural space is created by retinal development and subsequent retinal waves that link together the lateral geniculate nucleus and the cortex [3].

3. Visual Space Formation

The retina is an extension of the brain, and during embryogenesis the retinogeniculocortical oscillations create a pre-existing oscillatory network enabling the brain and the retina to communicate as one organ. This network forms the visual space, and during times when the eyes are closed, the pathways synchronize [8]. After birth as the baby develops, the images that form on the retina are immediately projected into the brain through fast oscillations. These fast oscillations between the brain and retina are termed "alpha" oscillations, which allow the brain and retina to communicate in milliseconds, forming the visual consciousness space [3].

Since we see our world from an internal point of view at the thalamus, the retinogeniculo-cortical synchronization provides the final image that is seen from the perspective of the lateral geniculate nucleus. When we say that we observe our external world from an internal point-of-view of the lateral geniculate nucleus, it means that the thalamus integrates the cortical faculties with external sensory stimuli, amplifying and intensifying sensory stimuli at the points of contact. This integration of cortical faculties with the retina includes information received from the retina, the retinogeniculo-cortical oscillations, and the oscillations from the executive cortex [3]. The relay cells that innervate the lateral geniculate nucleus receive and supply feedback inhibition to the reticular thalamic nucleus, and this influences the information the retina sends to the primary visual cortex [9].

4. Visual Processing

When light enters the cornea and the image is displayed on the retina, it produces graded electrical potentials by inducing electro-chemical changes on the discs. When these changes are transferred to ON-center bipolar cells, it stimulates ganglion cells. Lateral inhibition returns these potentials to the photoreceptors in addition to synchronized thalamic and cortical input. Graded potentials are the electrical version of the original image, with rods and cones serving as the receptors of the final perceived image. It is through retinogeniculo-cortical oscillatory synchronization with the visual cortex that produces this electrical image, which is what we perceive as the external image [3].

This conversion of signals from light to chemical to electrical is known as transduction and occurs in microseconds [10]-[12]. Additionally, transduction requires thalam-

ic and cortical loop processing [13] [14] in order for consciousness to arise [4]. The supposition is that the information coded within the electrical signals is processed through corticothalamic feedback loops, and that information is sent to the thalamus. Once the information reaches the thalamus, it is inserted into the internal visual space forming a recreated internal picture of the external environment [5]. The visual pathway processes up to 80% of the information it receives from the external environment [15], which may help explain how vision defines the internal visual and non-visual spaces.

Through this pre-existing visual network, images formed on the retina are automatically projected into the brain through retinogeniculo-cortical oscillations. The brain projections oscillate with the retina and visual and association cortices causing the faculties of the brain and the retina to communicate with each other in milliseconds. This communication is thought to occur among neurons located in the horizontal cells, amacrine cells, and the inner and external plexuses within the eye [3].

Furthermore, synchronization between the retinogeniculo-cortical oscillations and the visual space creates an internal grid that functions in unison with the discs on the photoreceptors to accurately code distance. The internal grid created by synchronization of retinogeniculo-cortical pathways accurately codes for distance and is generated with discs from the photoreceptors [5]. The individual photoreceptors act in a similar way to tiny eyes (like pixels on a computer screen), and convert light to graded potentials. These tiny images combine in the OFF-center cones, forming the final larger image perceived in vision. Photoreceptors integrate with the brain through the retinal plexuses because they oscillate with the brain, forming a complete image that appears external to us [3]. If integrated information is inaccurate, this perceived external image can contain images that do not exist, i.e. hallucinations.

Functional magnetic resonance imaging (fMRI) experiments have confirmed that detailed maps exist in the lateral geniculate nucleus [6]. A map of the contralateral visual field is formed by retinal ganglion cells, and an indirect pathway from the retina to the cerebral cortex is formed via projections through the superficial layer cells through the pulvinar nucleus to the cerebral cortex [7]. Both deep and superficial layers have the same visual field map, but deep layer cells respond to auditory and somatosensory stimuli. The spatial locations are aligned in such a way that neurons within the contralateral visual field respond to both the object and the sound of the object [7], supporting our theory that the brain receives multisensory input and places it on a map within the neural space that serves to orient us to our external world [4].

5. Visual Formation in the Brain

It was previously proposed that vision occurs when images from the left and right eyes are merged through corticothalamic feedback loops, and processed by the thalamus [5]. To better understand this concept, consider that it takes approximately 300 ms for the retina to process visual stimuli, but the volley of information is transmitted from the thalamus to the cortex in a single, static synapse [12]. This cortical processing illustrates

that our experiences only are perceived *after* our brain has had time to convert stimuli to neural signals *before* we can become conscious of it. Further, we only *interpret* visual processing as instantaneous because we are incapable of detecting time intervals that occur in less than 40 milliseconds [16]. This series of processing events creates a lag time between the time it takes for the brain to receive and process the visual images and the time when conscious visual perception occurs [17]. This suggests that vision, as we understand it, is not immediate—although it appears to be, but instead can only give rise to consciousness *after* reception, transduction, and re-projection into the visual field.

To further explain this delay between visual processing and conscious visual perception, an experiment using a flash stimulus followed by a backward mask determined that a minimum of 50 ms was needed to perceive the stimulus, suggesting that the delay was conceivably related to cortical loops processing [14]. Additionally, motion processing has been shown to occur in area V5 prior to vision activation in V1 (striate cortex) [18], while it can take evoked responses between 100 - 135 ms before visual information is processed by V4 (color processing area) [10]. This further supports the theory that vision is not instantaneous, because what is perceived as images in the external world has first undergone several stages of processing *before* images can rise to consciousness. What we have come to believe are external images are really only internal recreations of those images that have been re-projected outward, giving the perception of the external environment. If there is abnormal activity within the cortical areas in the brain, it is possible that real information may become entangled with unreal information, become processed together, and re-projected outward into the external visual field information, creating a hallucination of being outside of the body. Gaining a better understanding of how the reticular thalamic nucleus functions could provide a better understanding of the etiology of visual hallucinatory events.

6. Thalamic Nuclei and Cortical Activity

Although the thalamocortical system modulates alertness during information processing, it is the corticocortical system that is responsible for conscious experience content and its expression. However, consciousness can be lost or altered if distinct and distant neurons in the cortex lose synchronization and coherence [7]. We contend that if abnormal processing occurs in the cortex, then that activity could potentially cause visual or auditory hallucinations that are projected into the external visual field.

The neural network identified with consciousness is the corticothalamic system, and it has been proposed that the thalamic nuclei, including the reticular thalamic nucleus, play an important role in consciousness [7]. The reticular thalamic nucleus helps to regulate cortical activity, and is a vital component in communication between the thalamus and cortex [19]. Many areas of the brain project into the reticular thalamic nucleus, including the cortex and thalamus [7].

7. Corticothalamic Oscillations and Visual Processing

Sensory processing is intrinsically oriented to external space, so what is visually per-

ceived as the external world is actually only an internal representation of external space, arising from the translated electrical impulses from the thalamus [20]. During waking states, the corticothalamic oscillations actively link all parts of the intrapersonal space with the senses, creating a template for the external space through electric signals [11] [21]. Additionally, corticothalamic activity processes this information, sending the processed information to the thalamus. The thalamus continually integrates this information and sensory information with the cerebral cortex, allowing us a unified consciousness experience [3]. This integration automatically recreates an internal representation of the external environment from the information it has received [4], allowing us to experience events in the present moment, granting us the capability to respond to our environment in real time. During this internal recreation of the external world, if cellular hyper- or hypo-activity or abnormal cortical activity occurs then thalamus can recreate a representation that includes inaccurate information that results in hallucinations experienced in various forms of autoscopic phenomena.

8. Autoscopic Phenomena

The forms of autoscopic phenomena include out-of-body experiences (OBE), autoscopic hallucinations, and heautoscopy. All have been referenced in literature as far back as Aristotle [22]. These phenomena have been experienced by healthy people, as well as patients with epilepsy, seizures [23], migraines [24], depression, psychological conditions [22] [25]-[27], or resulting from anesthesia or drug abuse [28]. The forms of autoscopic phenomena consist of visual hallucinations that occur during the waking state, and create an illusion of disembodiment and perception of existing outside of the physical body. Each form creates a varying and unique perspective. **Figure 1** illustrates the location where various autoscopic hallucinations occur in the brain, and shows that all these areas communicate with the reticular thalamic nucleus, which was previously proposed as a central hub of consciousness.

In a "simple" OBE, the physical body is usually supine but the person may feel disembodied, and reports feeling as if he/she is outside the physical body and looking down from a superimposed position. During this experience, the person feels as if feelings, thoughts, and emotions are experienced from this parasomatic-visuospatial perspective—not from the actual physical body [26] (see **Figure 2**).

In an experience of autoscopic hallucination, a person may see his or her body doubled in external space in a position similar to the physical body, but continues to identify as the owner of the physical body. Those who have experienced this form of autoscopic phenomenon describe the experience as having seen a replica of themselves—as if looking at a reflection—but having retained a first person point-of-view, sense of "self", and body ownership [25]. This type of autoscopic phenomenon is considered rare, but has been reported in patients with neurological disorders such as migraine, focal epilepsy, or damage to the occipital or occipitoparietal lobe [23] [25].

Heautoscopy is described as experiencing shared similarities of disembodiment re-

ported in OBE and autoscopic hallucination, but differs in that the body ownership is indeterminable. To explain, when a person has a heautoscopic experience, he or she sees the physical body and a body double, but is unable to identify whether the sense of "self" belongs to the physical body or the body double. This causes uncertainty about the embodiment of the "self" because the body *and* the sense of "self" are doubled, resulting in the person's inability to determine which body is real [22] [23]. Those who have had heautoscopic experiences reported feeling as if they had the ability to see the world from two concurring and coexisting visuo-spatial perspectives [29], perceiving the world simultaneously from the real body and the body double [22]. Some patients who had heautoscopic experiences attempted suicide explaining that the experience was extremely terrifying [22] [23]. Although there is an abundance of research regarding the various autoscopic phenomena, these experiences are considered rare [26].

Corticothalamic Networking in Autoscopic Phenomena
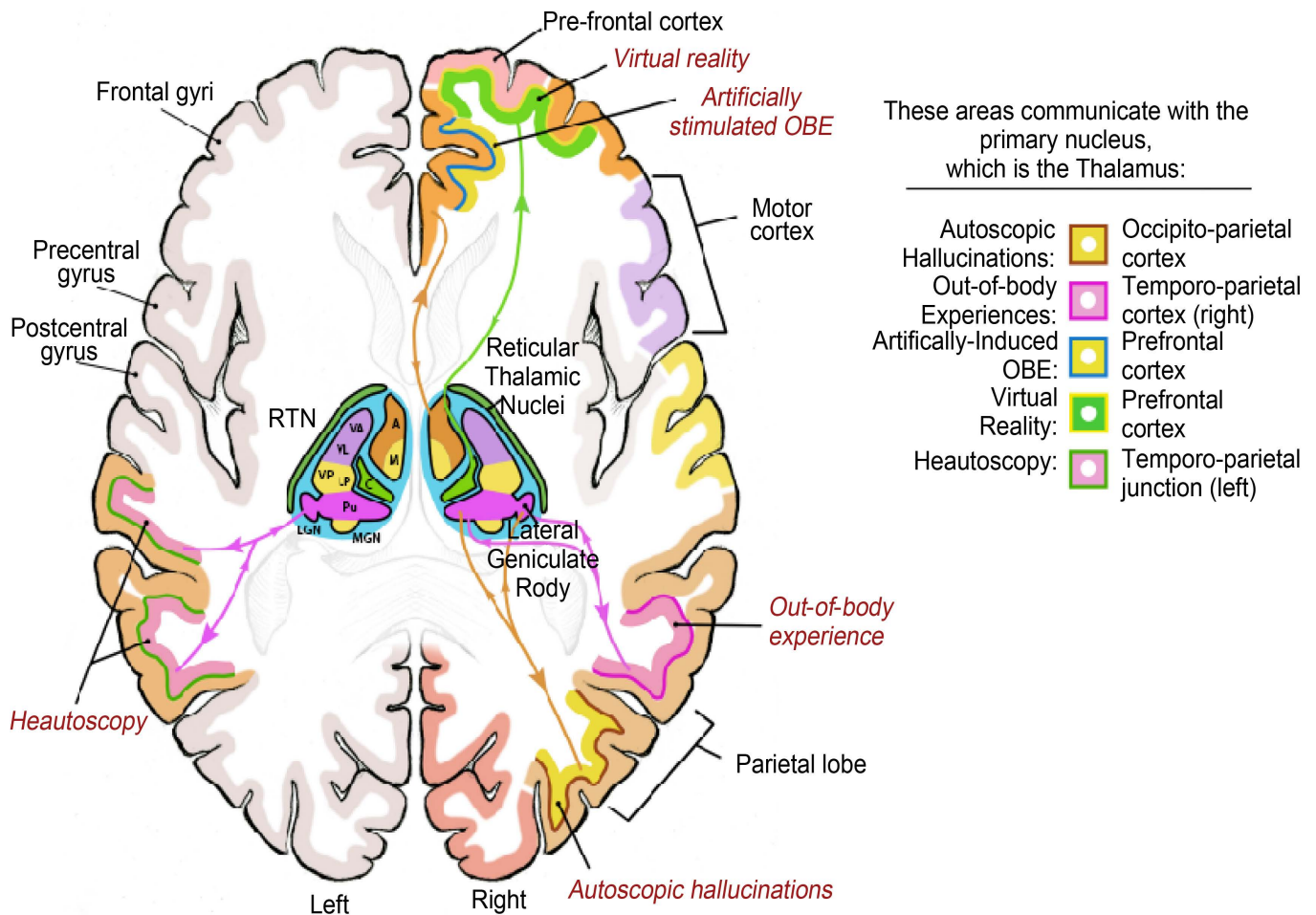
Figure 1. The corticothalamic networking in the brain during autoscopic phenomena. This figure illustrates the cortices in the brain where autoscopic phenomena occur. Arrows show the continual communication among the reticular thalamic nucleus and cortices of the brain, demonstrating that the thalamus is the primary hub for receiving and processing information before we can become aware of the experience.
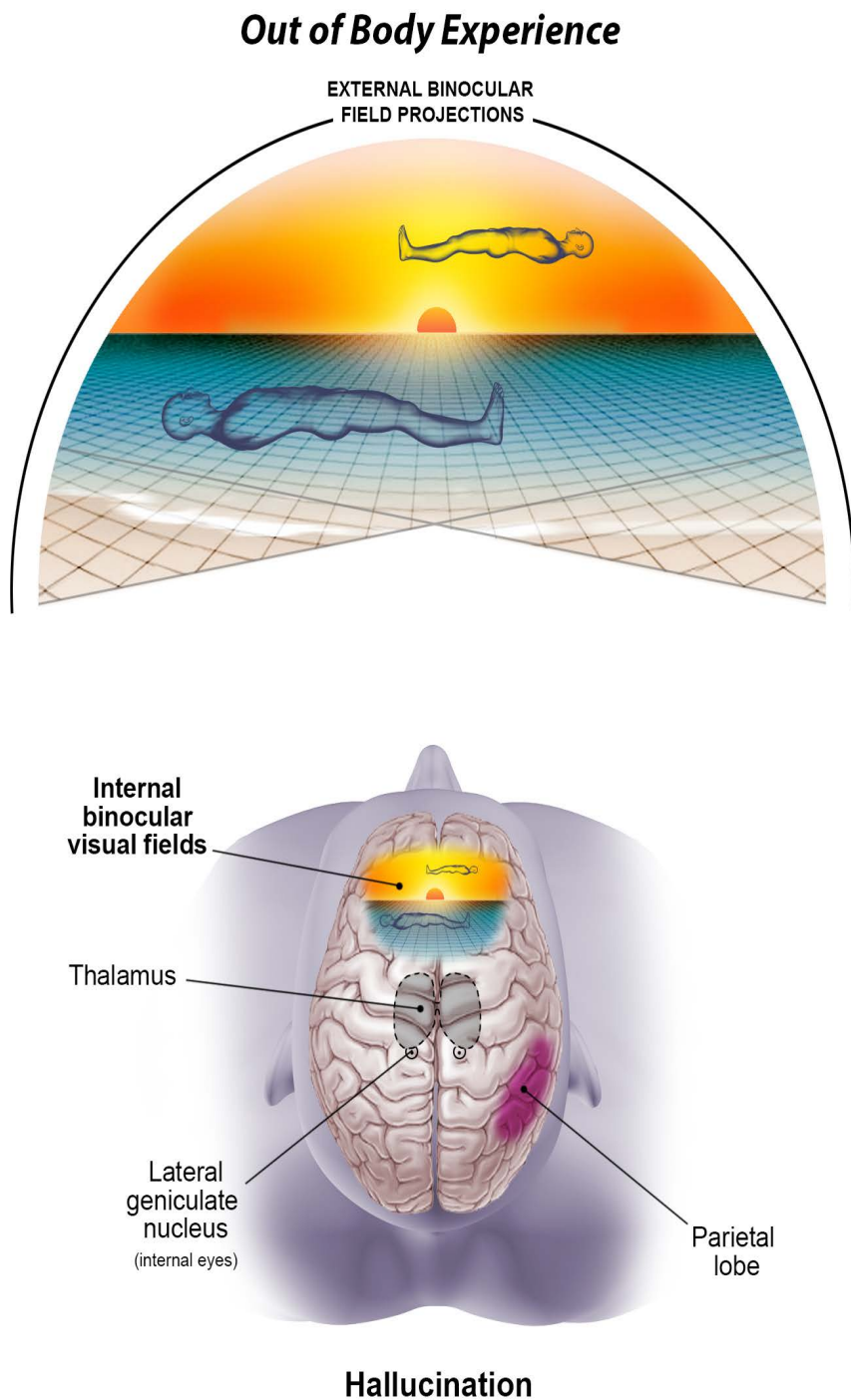
Figure 2. Out-of body-experience. This figure illustrates that an out-of-body experience is a hallucination that takes place during the waking state. Any interruption or abnormal integration within the corticothalamic networking can disrupt cues regarding space, time, location, and self. Out-of-body experiences are hallucinations that may result from hyperactivity or hypo-activity within the brain which are created within the internal visual space and reprojected outward into the external binocular field. Since all sensory information is fed into corticothalamic feedback loops and interpreted by the thalamus, the conflicting visuospatial perspectives can cause the person to feel as if the "self" is outside the physical body.

9. Role of Retinogeniculo-Oscillations in Out-of-Body Experiences

The lateral geniculate nucleus receives information and supplies feedback inhibition to the visual center of the reticular thalamic nucleus, influencing information that the primary visual cortex receives from the retina [3]. Layers within the lateral geniculate nucleus consist of retinotopic organization that, along with the ability of the brain to fill in missing information (illusory contouring), form complete images we see as the external world [30].

We contend that abnormal cortical activity could transmit incorrect information, and if this erroneous information is processed through retinogeniculo-cortical oscillations then it is possible that this could create a hallucinatory event, including any variation of an out-of-body experience (OBE), once it reaches conscious visual awareness. This is because the combination of abnormal activity or incorrect information processed through retinogeniculo-cortical oscillations would still be projected onto the retina, then re-projected into the external visual field, creating the appearance and feeling of being outside of one's body.

10. Thalamocortical Activity Implications in Hallucinatory Events

Current research utilizes various clinical and neurological approaches to examine the possible underlying mechanisms of autoscopic phenomena; however, the possibility that the reticular thalamic nucleus may play a role has not yet received full recognition from the research community (see Table 1). The thalamus is neuroanatomically and intimately involved in sensory integration, and may be the underlying contributor of these types of conflicting and hallucinatory visuo-spatial perspectives [13] [31]. Various fMRI studies have shown the simultaneous activation of the thalamus and cortex during mental tasks [32] [33], while other research suggests hallucinations could result from a change in thalamocortical activity [4] [34]. Therefore, it is possible that hyperactivity or hypo-activity within the temporo-parietal region of the brain may also lead to hallucinogenic events.

A pre-requisite for defining the area where hallucinations are perceived is the presence of a pre-existing, dynamic intrapersonal space during the waking state that represents the external space within our minds [20], and corticothalamic and retinogeniculo-cortical oscillatory processes could help explain autoscopic phenomena. Although researchers are unsure whether the various types of autoscopic phenomena are caused by a lack of proper integration of proprioceptive, tactile, and visual information, it has been suggested that uncoordinated cortical activity could be the cause of a person seeing his or her body in a position different from where it is perceived to be [26]. Therefore, it is plausible that interruption in normal cortical activity or abnormal integration of any kind within this networking can disrupt the normal cues to our mind for space, time, location, and self, leading to experiences described in autoscopic phenomena.

Table 1. Comparison of explanations about various autoscopic phenomena experiences.

Comparison of Explanations about Autoscopic Phenomena			
Type of Autoscopy	Out-of-Body	Autoscopic Hallucination	Heautoscopy
Brain Areas Affected by Damage	Focal brain damage [25]. Right hemisphere and damage to right temporo-parietal junction [22].	Extrastriate cortex and occipital cortex, damage to ventral visual pathway in proximity to extrastriate body area [25]. Damage to right parieto-occipital cortex [22].	Left posterior insular cortex [25]. Involves left hemisphere structures and damage to left temporo-parietal junction [22], and damage to the left posterior insular cortex [35].
Current Explanations Addressed in This Article	Disturbed multi-sensory integration in personal and extrapersonal space [25]	Disorder of body representation due to visuo-tactile disintegration [25].	Disintegration of somatosensory, visual, emotional, physical, or intuitive signals. Co-occurrence with aberrant sensory information from the vestibular organs, resulting in changes in first-person perspective and self-location [25].
Our Theory	This article discusses autoscopic phenomena with brain damage and the brain's inability to integrate the sensory information, and helps to explain what causes autoscopic phenomena experiences, e.g., damage to certain areas of the brain [25], defective visual processing of bodily information [22], and failure to integrate multisensory signals at the temporo-parietal junction [22]. We go a step further to explain how these are hallucinations are projected into the visual field.		
	When information is received by the lateral geniculate nucleus, the lateral geniculate nucleus then supplies feedback inhibition to the visual center of the reticular thalamic nucleus, influencing information that the primary visual cortex receives from the retina [3]. Layers within the lateral geniculate nucleus consist of retinotopic organization that, along with the ability of the brain to fill in missing information (illusory contouring), form complete images we see as the external world [30].		
	Incorrect information from aberrant cortical activity could transmit incorrect information to the lateral geniculate nucleus. Erroneous information processed through retinogeniculo-cortical oscillations that reaches conscious visual awareness could create a hallucinatory event, including any variation of an autoscopic phenomenon. This is because the erroneous information processed through retinogeniculo-cortical oscillations would still be projected onto the retina, then re-projected into the external visual field, creating the appearance and feel of being outside of one's body.		

11. Role of the Thalamus in Out-of-Body Experiences

The thalamus is known to be the sensory hub for processing information from cortical feedback loops, and provides cues to our mind for space, time, location, and self [36]. The thalamus integrates the cortical faculties with external sensory stimuli, helping to

amplify and intensify the initial information through lateral inhibition [37]-[39] to allow all the senses to work at the periphery while ignoring the intrapersonal space. Corticothalamic activity supports these processes [4], and the cerebral cortex constantly combines sensory stimuli allowing us to experience the external world [40].

The thalamus is only able to perform its functions via intact networking with the entire cortex [5] [20], therefore corticothalamic (temporo-parietal, hippocampus, and prefrontal cortical areas) hypo-or hyperactivity could generate hallucinations that the reticular thalamic nucleus experiences, in cases such as the various forms of autoscopic phenomena. In some cases, spontaneous activity elicited by subconscious neural activity can prevent conscious awareness of a body part or sensory information from one side of the body as in contralateral neglect [20], or induce sensations of pain of an amputated limb as in phantom limb syndrome [41]. Since the thalamus must integrate information from the cortex through the corticothalamic feedback loops [5], it is possible that damage to the cortex could also produce abnormal hallucinations. Therefore, similar subconscious and spontaneous neural activity can elicit hallucinations found in the various forms of autoscopic phenomena.

12. Autoscopic Phenomena and Concept of "Self"

The thalamus helps create the "self" space which contains the recreated external space located within our intrapersonal space [5] [8] [20]. This provides a better understanding of autoscopic phenomena as being hallucinations that occur internally and are projected into our external visual field. The thalamus coordinates activity with the cortex in "discerning" images and events, real or imagined, in our minds. Current research points to pathological issues, such as dysfunction within the temporo-parietal cortex, as common factors in various autoscopic phenomena [26], but evidence of the reticular thalamic nucleus and retinogeniculo-cortical oscillatory activity and their role in these phenomena is absent in the literature. For example, the lateral geniculate body is involved in visuospatial perception, attention, and vision [42] [43], and the medial geniculate body is involved in establishing body position and location [44], yet there is no discussion in the literature of the roles of thalamic nuclei in autoscopic phenomena.

Furthermore, without investigation into and understanding about how normal intra- and extrapersonal space is perceived in cases of various autoscopic phenomena, clinical conditions cannot be fully explained. If the thalamus is a central hub that integrates visual and vestibular information, along with consciousness of the "self", then it is reasonable to consider that focused research on the reticular thalamic nucleus and the retinogeniculo-cortical oscillations could offer insight into how various autoscopic phenomena occur.

13. Respiratory and Cardiac Roles in Autoscopic Phenomena

The most influential consciousness theories propose that bodily perception is paramount for self-consciousness, yet uncertainty remains in whether bodily perception results from internal or external stimuli, or integration between the two. Some research

posits the possibility that visual and cardiac signal synchronization may "modulate bodily self-consciousness and tactile perception", suggesting that the foundation of self-consciousness is integration of both interoceptive and exteroceptive signals [45]. Bodily rhythms are generated by the heart, and compared to the other organs within the body, more neural signals travel from the heart to the brain (than vice versa) due to an increased number of afferent network connections between the cardiovascular system and the brain [46].

The possibility exists that autoscopic phenomena could be caused by subcortical activity from the heart, respiratory system, brainstem or emotional centers due to spontaneous corticothalamic activity. This is because respiration does more than supply our body with the oxygen it needs. Respiration also affects cardiac rhythm [8] [47] and autonomic activity, and autonomic activity affects respiration through heart rate activity [48]. For example, seizures have been detected by heart rate changes [49], because changes in heart rate and respiration can occur before and during EEG detection of the seizure [50]. Electrocardiogram results have revealed heartbeat changes prior to the onset of seizures [51], and some patients who have had seizures have reported having OBE [23] [52]. Therefore it is possible that a connection exists between cardiac rhythm and OBE. This phenomenon leads us to question whether or not OBE could be a visuospatial seizure-like phenomenon, since apparently electrical activity of both the heart and respiration is known to trigger OBE [53] [54].

14. Conclusion

As more questions emerge in neuroscience examining the mechanisms of consciousness, more answers will be found within the exploration of the reticular thalamic nucleus, corticothalamic activity, and retinogeniculo-cortical oscillatory functions. We have provided evidence to support that retinogeniculo-cortical oscillations are the primary component of communication between the retina and the brain, allowing the perception of external vision to appear as seamless events. We have provided evidence that supports how the retinogeniculo-cortical pathway makes it possible for visual information to reach the cortex, and how it plays an integral role in vision and possibly the origin of hallucinations.

Current research focuses predominantly on cortical aspects of multisensory information, but future research should focus on: examining how irregular respiration and lack of cardiorespiratory synchronization could elicit various autoscopic phenomena, and investigating the role of the retinogeniculo-cortical oscillations around the reticular thalamic nucleus of the thalamus for the integration of multi-sensory information that could lead to autoscopic phenomena. This understanding could allow medical professionals to better treat disorders of the mind.

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