Consciousness is faster than light

Skyler P. Dillon^{1*}

¹Public key fingerprint: B00FFB33265B6E2356072F7917BD6099962D7186 *e-mail: skylerdillon@hotmail.com

Abstract

Nobody knows exactly how consciousness is produced. To make matters worse, nobody even knows what consciousness is. In this article, I provide insight into what consciousness is, and how it is produced, by showing that consciousness is faster than light. I also show that consciousness is a new faster-than-light phenomenon; as opposed to coming from non-local quantum mechanics. To be clear, I have not disproven Einstein's theory of relativity; instead, I have discovered a loophole in Einstein's theory of relativity that applies to consciousness. The discovery that consciousness is faster than light is important, because it shows that consciousness cannot be a particle, energy, or anything else located in space (*e.g.* not a soul). In other words, we can now conclude that consciousness is non-energy information. Where does this non-energy information (*i.e.* consciousness) come from? The discovery that consciousness is faster than light excludes numerous theories of consciousness while also enabling new theories of consciousness. For example, a computational theory of consciousness was previously untenable due to the fact that there is no convergence point in the brain for all information and the speed limit of light prevented the conscious observer from accessing all of the distributed information in the brain, but now a computational theory of consciousness is plausible. I show rare case reports and evidence that support the theory that consciousness (*i.e.* non-energy information) is, at least in part, produced directly from "non-trivial" computations.

1. Introduction

Consciousness is currently indistinguishable from magic. Though, a couple of facts are known about consciousness. First, our physical brain somehow affects our conscious experience^{1,2}. Second, our conscious experience is a simulation (Fig. 1). Many people have tried and failed to find the physical mechanism in the brain that affects consciousness^{3,4}. Here, I try a different approach. Instead of looking for the physical mechanism that affects consciousness, I look for the mechanism that binds together your left and right visual fields into a unified whole (*a.k.a.* the "binding problem"). The idea being that if I can find the mechanism that binds together your left and right visual fields, then we will gain insight into the undiscovered physical mechanism that affects consciousness.

Our journey begins with a simple observation. That being, patients that have one of their brain hemispheres surgically removed (*i.e.* hemispherectomy) lose the ability to consciously experience half of their visual field (Fig. 2; *i.e.* homonymous hemianopsia)^{5,6}. From this simple observation, I will show how to arrive at the conclusion that consciousness is faster than light. Then, I will use the discovery that consciousness is faster than light to show what consciousness is and provide insight into how consciousness is produced.

2. The "binding problem" of consciousness

Different regions of the human brain affect different parts of our conscious experience. For example, if you electrically stimulate different regions of the brain, then the subject will hallucinate different conscious experiences (*e.g.* rainbows, faces, phosphenes, *etc.*)^{2,7,8}. Likewise, surgically removing the left brain hemisphere will cause a patient to lose the right half of their visual field; and vice versa (Fig. 2)^{5,6}. However, there is no "cartesian theater" inside your brain where your visual field exists. Instead, the location of the qualia (*e.g.* colors, objects) in your visual field are different than the physical location of the corresponding neural activity in the brain (see visual cortex V1-V5).

Interestingly, the physical information in the brain that is correlated with your left visual field is independent, and separated in space, from the physical information in the brain that is correlated with your right visual field (Fig. 2)^{5,6}. Despite this fact, you somehow still consciously experience both your left and right visual fields. Even stranger, you somehow consciously experience both your left and right visual fields at the same time. How do you consciously experience both your left and right visual fields at the same time when the correlated physical information in the brain is separated in space? This problem is known as the "binding problem" of consciousness. Furthermore, why does the mechanism for binding together conscious experiences and/or brains? This problem I call the "privacy problem" of consciousness.

At first glance, it would appear that the conscious observer is violating the laws of physics. That being, the conscious observer appears to be in two places at the same time; accessing physical information in both the left and right brain hemispheres, at the same time, to enable the conscious observer to consciously experience both the left and right visual fields at the same time (*i.e.* faster-than-light, non-local)(Fig. 2).

However, there are two alternative interpretations (*a.k.a.* loopholes) for the binding problem that allow the conscious observer to avoid violating the laws of physics. That being, 1) the non-local quantum mechanics interpretation of the binding problem^{9,10}; and 2) the local "convergence point" interpretation of the binding problem^{11–14}. In the following sections, I will show that these two interpretations of the binding problem are untenable. Then, I will present a new interpretation of the binding problem, supported by empirical evidence, that explains how the conscious observer is behaving faster than light without violating the laws of physics.



Fig. 1 | **Your conscious experience is a simulation.** This is an optical illusion. Instructions: close your left eye and use your right eye to focus on the black dot; move your head backwards, or forwards, until the woman in the red dress disappears from your peripheral vision. Does the woman in the red dress actually exist in the physical realm? Or, is she just a figment of your imagination? The rabbit hole goes deeper. Did you notice that the woman was replaced by a false simulation of the two black bars being connected together? The rabbit hole goes deeper. Look at her yellow hair. If you are viewing her yellow hair on a display that uses the red-green-blue (RGB) color model, then the color yellow does not exist on the RGB color display. Thus, the yellow color of the woman's hair only exists in your mind. Is the illusion of the woman's yellow hair color the only illusion that exists in your mind? Or, is everything else in your mind also an illusion? Does the physical realm even exist? Or, is everything just a dream? You will never know because of solipsism. For more information on conscious simulations, see: phantom limb syndrome, "filling-in" of the blind spot, color constancy illusions, hallucinations, dreams, and the cortically induced phosphenes in a retinally blind subject with severed optic nerves². Image of woman generated by non-human intelligence.

3. Non-local quantum mechanics and the "binding problem"

There is already an interpretation of physics that allows for faster-than-light phenomena; that being, non-local interpretations of quantum mechanics. Not surprisingly, people have already speculated that non-local quantum mechanics might solve the "binding problem" of consciousness^{9,10}. However, there is currently no convincing evidence that non-local quantum mechanics has anything to do with the "binding problem." Nonetheless, non-local quantum mechanics offers two types of solutions to the "binding problem." That being, 1) your left and right visual fields are bound together by the entire brain existing in a quantum superposition of macroscopic states⁹; and/or 2) your left and right visual fields are bound together by quantum entanglement¹⁰. As will be discussed, both quantum mechanical interpretations of the "binding problem" are untenable for different reasons.

3.1. A brain-wide quantum superposition is an untenable solution to the "binding problem"

The idea that your left and right visual fields are bound together by your entire brain existing in a quantum superposition of macroscopic states⁹ is untenable due to empirical evidence. For example, a patient who undergoes "awake brain surgery" obviously does not lose consciousness when the surgeon collapses the alleged macroscopic wave function of the patient's brain (Fig. 3f)¹⁵. Similarly, a patient does not lose consciousness when a healthcare worker observes an MRI scan or an electroencephalogram (EEG) recording; which would also collapse the alleged macroscopic wave function of the patient's brain.

The idea that a brain-wide quantum superposition has something to do with consciousness is also incompatible with a rare case of craniopagus twins that share brain tissue; that being, the Hogan twins (Fig. 3a)¹⁶. The brains of the Hogan twins are connected by a "thalamic bridge" and, as a result, the twins can see out of each other's eyes¹⁶. However, despite being able to see out of each other's eyes, the Hogan twins have different conscious experiences. For example, Tatiana Hogan can see out of both of her twin's eyes, but Krista Hogan can only see out of one of Tatiana's eyes¹⁶. Thus, if the Hogan twins' left and right visual fields were bound together by a brain-wide quantum superposition, then it would follow that the Hogan twins somehow have two different brain-wide quantum superpositions⁹ in order to explain their different visual fields, which is an untenable argument considering the fact that their brains are woven together and the problems this would create with quantum decoherence.

In agreement with the empirical evidence, theoretical calculations of neural decoherence rates also indicate that it is extremely improbable that the entire brain exists in a superposition of macroscopic states¹⁷. For the aforementioned reasons, it would be unreasonable to conclude that your left and right visual fields are bound together by your entire brain existing in a quantum superposition of macroscopic states. As a result, the faster-thanlight behavior of the conscious observer must be explained by something else.

3.2. Quantum entanglement is an untenable solution to the "binding problem"

Quantum entanglement refers to a physics phenomenon where the state of one system is dependent upon the state of another system¹⁹. In the simplest case of two maximally entangled qubits (*e.g.* an EPR pair), the states of the two particles are perfectly correlated with each other. In other words, due to quantum entanglement, the quantum state of one particle cannot be independent of the state of the second particle; instead, the two particles' states are dependent upon each other.

Thus, by definition, two maximally entangled qubits cannot explain how your left visual field (*i.e.* one system) and your right visual field (*i.e.* a different system) are bound together into a unified whole, because the information in your left and right visual fields are obviously completely independent from one another (*i.e.* not entangled)(Fig. 2). In other words, if your left and right visual fields were bound together by two maximally entangled qubits, each located in a different brain hemisphere (Fig. 2), then your two brain hemispheres would lack independence and, as a result, your left visual field would be identical to your right visual field; or inverted.

To make matters worse, simply entangling a couple of particles is obviously not enough to bind together all of the neural activity that is correlated with the qualia in your conscious experience. In order to bind together all of the qualia in your conscious experience, you would need to quantum entangle, at the very least, the majority of your cerebral cortex. The problem with quantum entangling the majority of your cerebral cortex is that such a macroscopic quantum wave function would be untenable due to the same reasons provided in the previous section on brain-

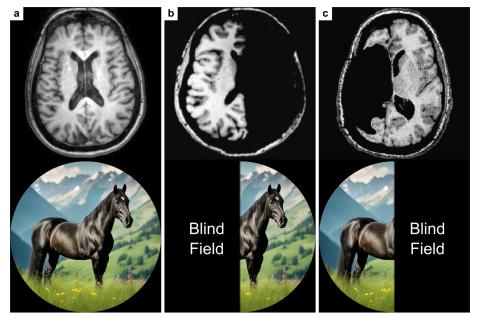


Fig. 2 | **The "binding problem" of consciousness. a**, A normal human brain and a normal visual field. **b**, Surgically removing the right brain hemisphere (*i.e.* hemispherectomy) will cause a patient to lose the left half of their visual field (*i.e.* homonymous hemianopsia)^{5.6}; **c**, and vice versa. How are your left and right visual fields bound together into a unified conscious experience? How do you consciously experience both your left and right visual fields at the same time when the correlated physical information in the brain is separated in space? This problem is known as the "binding problem" of consciousness. The MRIs are reprinted from the DLBS database¹⁸, Reuter-Lorenz PA, *et al.*⁵, and Herter TM, *et al.*⁶ with permission; horse generated by non-human intelligence.

wide quantum superpositions (*e.g.* "awake brain surgery," EEGs, decoherence rates).

The idea that the brain is using quantum entanglement to solve the "binding problem" is also at odds with the fact that medical research still has not found a convincing gene, protein, mutation, disease, drug, or molecular pathway in the brain that is involved with quantum entangling particles across the brain^{20–23}. At this point in history, it is more likely that there is simply no biological pathway that quantum entangles particles across the brain.

In summary, it is untenable to argue that your left and right visual fields are bound together by quantum entanglement. As a result, the faster-than-light behavior of the conscious observer must be explained by something else.

4. A local "convergence point" theory and the "binding problem"

Albert Einstein, Boris Podolsky, and Nathan Rosen famously objected to the non-local interpretation of quantum mechanics²⁴ by arguing that there was a reasonable possibility that quantum entanglement is not faster than light and is instead potentially an illusion created by a "local hidden-variable." A similar objection can be made against this article, which argues that consciousness is faster than light. That being, there is a reasonable possibility that the conscious observer is not faster than light and instead there is a "convergence point" in space where all information from the brain converges onto to produce your unified conscious experience without violating the speed limit of light. This interpretation I call the "convergence point" theory for the "binding problem" of consciousness.

The idea that there must be a "convergence point" in the brain where your left and right visual fields are bound together is a reasonable hypothesis that has been around for a long time¹¹. Thus, the "convergence point" interpretation must be excluded before we are forced to conclude that the conscious observer is behaving faster than light. To be clear, there is currently no convincing evidence that your entire conscious experience is produced by a "convergence point" in space. Nonetheless, there are three different types of "convergence points" that could solve the "binding problem" of consciousness without needing faster-thanlight phenomena. That being, 1) your left and right visual fields are bound together by neural activity converging onto a single neuron or point in the brain¹¹; 2) your left and right visual fields are bound together by the brain hemispheres broadcasting all information to multiple points in space such that each point in space receives all of the information needed to produce your entire conscious experience¹²⁻¹⁴; or 3) the conscious observer is accessing all of the information in the brain by physically traveling through the brain at the speed of light. As will be discussed, all three "convergence point" interpretations of the "binding problem" are untenable for different reasons.

4.1. Neural activity is not converging onto a single neuron, or point, to solve the "binding problem"

If all of your qualia, including your left and right visual fields, were bound together by neural activity converging onto a single neuron or point in the brain, then a brain lesion that specifically destroys that "convergence point" would cause you to become unconscious; however, no such brain lesion has ever been discovered. Furthermore, it is generally accepted that your left and right visual fields are not bound together by neural activity converging onto a single neuron or point in the human brain¹. However, there is a deficiency of published research documenting the attempts at finding, or excluding, the existence of a single "convergence point" in the human brain. Thus, for the sake of thoroughness, I performed a *de novo* review of the literature; searching for brain lesions in every region of the human brain. I found that the symptoms of unilateral brain lesions were well documented for each region of the human brain and

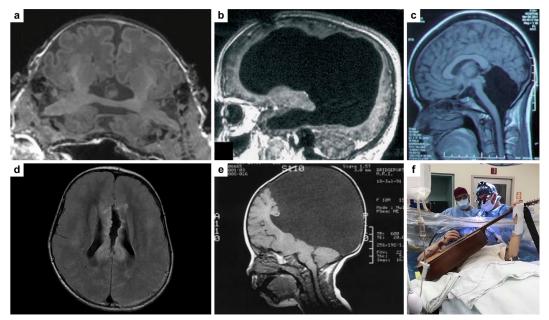


Fig. 3 | **Case reports that provide insight into consciousness. a**, The Hogan twins are craniopagus twins and their brains are connected by a "thalamic bridge." Tatiana Hogan can see out of both of her twin's eyes, but Krista Hogan can only see out of one of Tatiana's eyes¹⁶. Image reprinted from BC Children's Hospital¹⁶ in Canada in accordance with the copyright doctrines of fair use and fair dealing. b, A conscious man with hydrocephalus. Hydrocephalus patients never report having a distorted visual field²⁵. Image reprinted from Feuillet L, *et al.*²⁵ with permission. **c**, A conscious person missing their cerebellum. The cerebellum appears to have no direct effect on consciousness²⁶ (Supplementary Table 1). Image reprinted from Yu F, *et al.*²⁶ with permission. **d**, A person that had their two brain hemispheres partially separated by a surgical procedure known as corpus callosotomy (*a.k.a.* split-brain). Split-brain patients report that their left and right visual fields are still united²⁷. Image reprinted from Radiopaedia.org (rID: 11331, The Radswiki) with permission. **e**, A conscious person with vision. Image reprinted from Shewmon DA, *et al.*²⁸ with permission. **f**, A conscious person undergoing "awake brain surgery." Image reprinted from Mackel CE, *et al.*¹⁵ with permission.

unconsciousness is not a symptom of a unilateral brain lesion (Supplementary Table 1). Thus, I independently came to the same conclusion as the conventional wisdom; that being, unconsciousness cannot be caused by the destruction of a single neuron or point in the human brain.

Interestingly, there are specific brain lesions that do cause unconsciousness (*e.g.* coma, vegetative state)^{1,29,30}. However, the brain lesions that do cause unconsciousness must be bilateral and much larger than a single neuron or point in the brain^{1,29,30}. In summary, the empirical evidence shows that your left and right visual fields are not bound together by neural activity converging onto a single neuron or point in the brain. Thus, to avoid the conclusion that the conscious observer is violating the laws of physics, we must find a different interpretation for the "binding problem" of consciousness.

4.2. A broadcast theory is an untenable solution to the "binding problem"

While it may be true that your entire conscious experience is not produced by only one neuron or point in the brain, it does not follow that we have excluded the "convergence point" interpretation of the "binding problem" of consciousness. There is another plausible "convergence point" interpretation that must also be excluded before we are forced to conclude that the conscious observer is behaving faster than light. That being, your left and right visual fields might be bound together by the brain broadcasting all information to multiple points in space such that each point in space receives all of the information needed to produce your entire conscious experience without violating the speed limit of light^{12–14}. Such a broadcast model would be resistant to the destruction of a single neuron or point in the brain. Examples of plausible mechanisms that neurons could use to broadcast information to multiple "convergence points" across the brain would include electromagnetic radiation¹², diffusion, or an undiscovered field such as Libet's "conscious mental field"¹³.

The main problem with the idea that your left and right visual fields are bound together by your brain hemispheres broadcasting information to multiple "convergence points" in space is that such theories are internally inconsistent. That being, if your brain hemispheres can broadcast information to each other, then it must follow that your brain hemispheres can also broadcast information to other peoples' brain hemispheres located nearby and thus can either interfere with or bind together different peoples' conscious experiences into a unified whole. However, the empirical evidence is clear, we never experience interference with, or binding with, other peoples' conscious experiences. Furthermore, the brains of the Hogan twins are physically woven together and even they have different conscious experiences (supra; Fig. 3a). In other words, if our brain hemispheres were broadcasting information to bind together conscious experiences, then the Hogan twins should definitely have a single unified conscious experience like we do, but they do not (supra; Fig. 3a).

Another problem with "broadcast" theories is that the broadcast should lose intensity over distance yet the binding of your left and right visual fields appears to be unaffected by the variable distances between the different brain regions. Furthermore, patients with hydrocephalus never report having a distorted visual field despite the fact that the distance between some of their neurons has changed drastically (Fig. 3b)²⁵.

In summary, it is untenable to argue that your left and right visual fields are bound together by the brain hemispheres broadcasting all information to multiple points in space such that each point in space receives all of the information needed to produce your entire conscious experience. Thus, to avoid the conclusion that the conscious observer is violating the laws of physics, we must find a different interpretation for the "binding problem" of consciousness.

4.3. The conscious observer is not traveling through the brain to solve the "binding problem"

If the conscious observer traveled at the speed of light, then it would still take the conscious observer at least 587 ms to travel the 176,000 km of myelinated fibers in the human brain³¹. Obviously, 587 ms is too slow to explain our reaction times and the fact that we consciously experience both our left and right visual fields at the same time. Furthermore, taking 587 ms to travel the entire human brain would cause the conscious observer to miss most neural firings and also cause problems with the interpretation of neural activity due to the "relativity of simultaneity." Thus, it is untenable to argue that the conscious observer is producing your conscious experience by accessing all information in the brain by traveling through the brain at the speed of light.

5. A new interpretation of the "binding problem"

5.1. A loophole in Einstein's theory of special relativity allows for certain faster-than-light phenomena

We are in an awkward situation. It appears that the conscious observer is violating the laws of physics. That being, I have shown that the conscious observer appears to be in two places at the same time; accessing physical information in both the left and right brain hemispheres, at the same time, to enable the conscious observer to consciously experience both the left and right visual fields at the same time (*i.e.* faster-than-light, non-local)(Fig. 2). I have also shown that it is untenable to argue that the faster-than-light behavior of the conscious observer is coming from non-local quantum mechanics (*supra*). Furthermore, I have also shown that it is untenable to argue that the faster-than-light behavior of the conscious observer is an illusion created by a "convergence point" in space (*supra*).

Thus, it would appear that I have disproven Einstein's theory of special relativity³²; which showed that an object cannot move faster than the speed of light. However, upon closer inspection, I have discovered a loophole in Einstein's theory of special relativity that allows for certain faster-than-light phenomena without disproving Einstein's theory of relativity or any other laws of physics. Before I explain the loophole, it is important to first be aware that Einstein derived his theory of special relativity from two postulates³². The loophole is located in Einstein's second postulate of special relativity, which postulated that the speed of light is constant in all frames of reference³². The loophole is as follows: as long as the conscious observer does not occupy a frame of reference in space, then the conscious observer can behave faster than light, because the conscious observer no longer occupies a frame of reference in space from which light must constantly be outrunning the conscious observer in accordance with the second postulate of Einstein's theory of special relativity. In other words, as long as the conscious observer is not an object moving through space, then you cannot use Einstein's theory of special relativity to argue that the conscious observer cannot behave faster than light, because the second postulate of special relativity has no authority over a conscious observer that is not an object moving through space. Likewise, all of the arguments that are derived from the second postulate would also not apply to a conscious observer that is not an object moving through space (*e.g.* the Lorentz factor would not apply to such a conscious observer).

5.2. The loophole in Einstein's theory of special relativity applies to the conscious observer

Now that I have found a loophole in Einstein's theory of special relativity that allows for certain faster-than-light phenomena, does this loophole actually apply to the conscious observer? Yes, the empirical evidence and arguments presented in the previous sections, regarding the lack of a "convergence point" in space for the conscious observer, also support the conclusion that the conscious observer does not occupy a frame of reference in space in the context of special relativity (see also Supplementary Table 1).

Furthermore, anyone who wants to claim that Einstein's theory of special relativity, or similar logic, prohibits the conscious observer from behaving faster than light bears the burden of proving that the second postulate of special relativity applies to the conscious observer (*i.e.* prove that the conscious observer occupies a frame of reference in space). No one has ever provided any convincing evidence, and I have found none, for the proposition that the conscious observer occupies a frame of reference in space in the context of special relativity (Supplementary Table 1).

Now is a good time to point out that while it may be true that consciousness is correlated with neural activity, it does not follow that the conscious observer is an object in space. How can our conscious experience be correlated with neural activity if the conscious observer is not located in space? Based on the evidence, it appears that there are at least two realms: that being, the physical realm and the conscious realm (*a.k.a.* consciousness and the conscious observer; see also Fig. 1). In other words, consciousness is correlated with brain activity, but consciousness is neither the physical brain nor anything else located in space. Likewise, as this article shows, the conscious realm is not directly governed by the laws of physics, but nonetheless the conscious realm is correlated with activity in the physical realm (*e.g.* the physical brain).

The fact that the conscious observer does not occupy a frame of reference in space is one of the key differences between this article and previous theories for the "binding problem" of consciousness (Table 1)^{9–14}.

5.3. No causality violations

There are no causality violations created by the conscious observer behaving faster than light due to several different reasons. First, the loophole in special relativity that allows the conscious observer to behave faster than light requires that the conscious observer cannot be an object traveling through space (supra). Thus, the conscious observer cannot "travel" through spacetime to create a causality violation in the physical realm. Second, the loophole also requires that the conscious observer cannot be energy (supra). Thus, the conscious observer does not have the energy required to do the work of "causing" anything in the physical realm (e.g. send a signal back in time). Third, all of the arguments in support of the claim that faster-than-light phenomena create causality violations are derived from the second postulate of special relativity (e.g. the Lorentz transformation and the composition law for velocities) or similar logic, which cannot be applied to the conscious observer, because the conscious observer neither occupies a frame of reference in space nor has a velocity.

Author:	Descartes ¹¹ (1649)	Germine ⁹ (1991)	Many ^{12,13}	Stoica ¹⁰ (2020)	Lahav N, <i>et</i> <i>al</i> . ¹⁴ (2022)	Dillon (this article)
How do your left and right visual fields get bound together into a unified whole?	Convergence point	Brain-wide quantum superposition	Field theory	Quantum entanglement	Convergence point	Faster than light
Objections?	Yes, see this article.	Yes, see this article.	Yes, see this article.	Yes, see this article.	Yes, see this article.	_
Can the conscious observer be a particle, energy, or anything located in space (<i>e.g.</i> a soul)?	Yes	Yes	Yes	Yes	Yes	No
How does the theory reconcile with Einstein's theory of relativity?	Principle of locality	Non-local quantum mechanics	Principle of locality	Non-local quantum mechanics	Principle of locality	Loophole
Did they close all of the known reasonable loopholes for alternative interpretations?	No	No	No	No	No	Yes

 Table 1 | Comparison of different theories for the "binding problem" of consciousness. The problem of creating a unified conscious experience from distributed neural activity that is separated in space is an old problem with many different theories. This table compares this article with other theories for the "binding problem" of consciousness.

In summary, there is nothing wrong with consciousness behaving faster than light by being correlated with both your left and right brain hemispheres at the same time; nothing is "traveling" back in time and no causality violations are occurring.

5.4. Relativity of simultaneity

The fact that the conscious observer does not occupy a frame of reference in space solves an additional problem related to consciousness known as the problem of "relativity of simultaneity." That being, if you were to give the conscious observer a location in space, then the timing of neural activity in the left brain hemisphere could be perceived as being before, or after, the neural activity in the right brain hemisphere, depending on where the conscious observer was located in space. Because I have shown that the conscious observer does not occupy a frame of reference in space, the problem of "relativity of simultaneity" disappears. Instead, the conscious observer is obtaining information in both the left and right brain hemispheres instantaneously (*i.e.* faster-than-light, non-local).

5.5. The "privacy problem" in an infinite universe

For the sake of argument, if we assume that the universe is infinite, then there could be an infinite number of copies of you in the universe. If consciousness is non-local, then what is preventing your twins' conscious experiences from interfering with your conscious experience? This "privacy problem" is not unique to a non-local theory of consciousness, but is also a problem for a local theory of consciousness (*e.g.* see the Hogan twins, *supra*; Fig. 3a). While it may be true that consciousness is non-local, it does not follow that there is not a local constraint on consciousness that prevents interference with other peoples' conscious experiences. An example of a plausible local constraint on the non-local conscious observer would be the conscious observer non-locally obtaining information produced by locally constrained computations (*e.g.* neural computations in the left and right brain hemispheres).

6. Free will

This article provides useful evidence for the free will debate. That being, I have shown that the conscious observer lacks the energy required to perform the work of changing the course of deterministic processes in the physical realm. I have also shown that free will cannot be coming from the conscious observer causing a collapse of a wave function for a brain-wide quantum superposition (*supra*; Fig. 3f). Thus, this article is in agreement with other evidence suggesting that free will may be an illusion. For example, see "alien hand" syndrome, Tourette syndrome, split-brain patients²⁷, psychoactive drugs, Libet's experiments, "utilization behavior," self-dissociation disorders³³, and the fact that we have not evolved to harness the free energy coming from free will. However, to be clear, I have not disproven free will. There are still some loopholes remaining for interpretations in favor of free will existing (*e.g.* the conscious observer causing a collapse of a wave function for smaller systems³⁴). However, I have put some severe constraints on the possible mechanisms by which free will could exist.

7. What is consciousness?

In this article, I have shown that the conscious observer behaves faster than light. I have also shown that the conscious observer is able to behave faster than light, because the conscious observer does not occupy a frame of reference in space in the context of Einstein's theory of special relativity (*supra*). Thus, the conscious observer cannot be a particle, energy, or anything else located in space (*e.g.* not a soul). In other words, the conscious observer is non-energy information. Consciousness is correlated with brain activity, but consciousness is not the physical brain. It is worth mentioning that this article is the first convincing evidence, based in science not faith, that you are something more than a clump of particles (Table 1)^{3,4}.

8. How is consciousness produced?

There are many different theories of consciousness^{3,4}. However, the discovery that consciousness is faster than light excludes numerous theories of consciousness while also enabling new theories of consciousness. For example, a computational theory of consciousness was previously untenable due to the fact that there is no "convergence point" in the brain for all information and the speed limit of light prevented the conscious observer from accessing all of the distributed information in the brain (*supra*), but now a computational theory of consciousness is plausible.

This article also provides another key piece of evidence that is helpful for figuring out the mechanism that produces consciousness; that being, consciousness is non-energy information. Where is this non-energy information coming from? In other words, what is the physical mechanism inside the brain that is directly affecting this non-energy information that we call consciousness? According to the evidence, the most plausible physical mechanism is the act of computation. Here, I will show rare case reports, and other evidence, that support the theory that consciousness (*i.e.* non-energy information) is, at least in part, produced directly from "non-trivial" computations; as opposed to consciousness being produced by a different mechanism downstream of computations (*e.g.* a chemical reaction, electromagnetic radiation, physical states, *etc.*).

The first case report involves a rare case of craniopagus twins that share brain tissue; that being, the Hogan twins (Fig. 3a)¹⁶. Tatiana Hogan can see out of both of her twin's eyes, but Krista Hogan can only see out of one of Tatiana's eyes¹⁶. Many theories of consciousness^{3,4,14}, including field theories^{12,13} and quantum theories⁹, cannot explain how the Hogan twins have different conscious experiences. However, a computational theory of consciousness can explain how the Hogan twins have different conscious experiences. For example, the Hogan twins appear to have two different sets of neural circuits; including two different cortico-cortical circuits (Fig. 3a).

The second case report is of an extreme case of hydrocephalus (Fig. 3b)²⁵. Many theories of consciousness^{3,4}, including field theories^{12,13}, cannot explain the fact that patients with hydrocephalus never report having a distorted visual field despite the fact that the distance between some of their neurons has changed drastically (Fig. 3b)²⁵. A computational theory of consciousness can explain the undistorted visual fields of hydrocephalus patients, because neural computations can be unaffected by the distorted neural tissue.

The third case report involves a patient, named "GY," who is missing most of his left primary visual cortex (V1) due to a brain lesion⁸. As a result, patient GY is blind in most of his right visual field⁸. In a normal subject, unilateral transcranial magnetic stimulation (TMS) of the middle temporal visual area (V5) will induce a phosphene in the respective visual hemifield and bilateral stimulation of V5 will induce phosphenes in both visual hemifields⁸. In patient GY, unilateral stimulation of V5 in the intact hemisphere does induce a phosphene, but unilateral stimulation of V5 in the damaged hemisphere lacking V1 does not induce a phosphene⁸. The interesting part is that bilateral stimulation of V5 in patient GY does induce phosphenes in both visual hemifields (including GY's blind visual field)⁸. The importance of this finding is that it shows that stimulating the same exact neurons (i.e. V5) will induce, or not induce, a specific phosphene depending upon what computational inputs those neurons are receiving. In other words, there appears to be no biochemical difference between "conscious" neurons and "subconscious" neurons. Instead, the difference between "conscious" neurons and "subconscious" neurons appears to be coming from computational differences. Another interesting fact about patient GY is that the phosphene in GY's blind field is the wrong color⁸, which is a glitch that is hard to explain without appealing to a computational theory of consciousness.

Gene expression data is also consistent with the theory that the difference between "conscious" and "subconscious" neurons is coming from computational differences; not biochemical differences. For example, if consciousness was coming from a biochemical difference between "conscious" and "subconscious" neurons, then there should be an obvious gene expression difference between the cortex and the cerebellum, because the cortex is correlated with consciousness, but the cerebellum is not (Fig. 3c; Supplementary Table 1)²⁶. In reality, the gene expression differences between the cortex and the cerebellum are relatively minor and certainly do not support the existence of a special protein-coding gene for consciousness, but the cerebellum is not, is presumably coming from computational differences instead of biochemical differences. Likewise, the gene expression differences between the different cortical regions are also relatively minor and certainly do not support the existence of different protein-coding genes for different qualia^{21,23}. Instead, different qualia are more likely coming from computational differences instead of biochemical differences.

Genetic mutations are also consistent with a computational theory of consciousness. Several hundred different genetic mutations have been discovered that cause the loss of a specific qualia (*e.g.* deafness, blindness)²⁰. However, all of the genetic mutations that cause the loss of a specific qualia (*e.g.* deafness, blindness) appear to affect the sensory organs and/or the neural circuits²⁰; as opposed to affecting a different mechanism that is unnecessary for normal neural computational activity, no genetic mutation has been discovered that causes the loss of a specific qualia (*e.g.* deafness, blindness); which is consistent with the theory that the different qualia are coming from computational differences as opposed to a biochemical difference between neurons.

General anesthetics can also be explained by a computational theory of consciousness. The exact mechanism by which general anesthetics "turn off" consciousness has evaded discovery for a longtime^{35–39}. However, when viewed from a computational perspective, the mechanism by which general anesthetics "turn off" consciousness becomes obvious. That being, all general anesthetics share in common the fact that they interfere with neural computations; as opposed to directly inhibiting a specific biochemical mechanism inside neurons that directly produces specific qualia^{35–39}.

Psychedelics can also be explained by a computational theory of consciousness. The exact mechanism by which psychedelics "turn on" specific qualia has also evaded discovery for a longtime^{40,41}. However, when viewed from a computational perspective, the mechanism by which psychedelics "turn on" specific qualia becomes obvious. That being, all psychedelics share in common the fact that they increase the activity of specific neural circuits⁴² (*e.g.* +5-HT_{2A}R/+mGluR2 circuits⁴¹); as opposed to directly activating a specific biochemical mechanism inside neurons that directly produces specific qualia.

Finally, the physical mechanism that produces consciousness must have a high signal-to-noise ratio to avoid interference from background noise coming from other similar biochemical processes in the cell, other brains, and the non-local universe. Many theories of consciousness are untenable due to their low signal-to-noise ratios^{3,4,12-14}, but not a computational theory of consciousness. In a non-local universe full of background noise, it is hard to imagine a physical mechanism for producing consciousness that has a higher signal-to-noise ratio than a "non-trivial" computation that is locally constrained (*e.g.* neural computations).

If computations produce consciousness, then is my phone conscious? Unlikely, because we already know that not all computations produce consciousness (*e.g.* see the cerebellum; patient GY, *supra*). Thus, it appears that only a special type of computation produces consciousness. See also the "triviality" debate⁴³⁻⁴⁷. Does the physical mechanism that affects consciousness require more than just a "non-trivial" computation? This is still an open question⁴³⁻⁵⁰. I have not excluded the possibility that there are additional factors required in combination with a "non-trivial" computation to produce a conscious experience. However, the focus of this section is to show that "non-trivial" computation is a required factor for producing

consciousness; not to show that "non-trivial" computation is the only required factor for producing consciousness.

In summary, I have shown that consciousness is non-energy information. Where is this non-energy information (i.e. consciousness) coming from? Previously, a computational theory of consciousness was untenable due to the fact that there is no "convergence point" in the brain for all information and the speed limit of light prevented the conscious observer from accessing all of the distributed information in the brain (supra), but now a computational theory of consciousness is plausible, because I have also shown that the conscious observer is instantaneously obtaining information that is separated in space in the brain (*i.e.* faster-than-light, non-local). In addition, I have also shown that multiple lines of evidence are now consistent with the theory that consciousness (*i.e.* non-energy information) is, at least in part, produced directly from "non-trivial" computations; as opposed to consciousness being produced by a different mechanism downstream of computations (e.g. a chemical reaction, electromagnetic radiation, physical states, etc.). Likewise, "nontrivial" computations can now explain, at least in part, how the different qualia are produced, and how the different qualia are bound together into a unified conscious experience, and the lack of binding together of qualia between different brains, and the lack of interference in our conscious experience from background noise in the non-local universe.

References

- Plum and Posner's Diagnosis of Stupor and Coma. (Oxford University Press, Oxford; New York, 2007).
- Cowey, A. & Walsh, V. Magnetically induced phosphenes in sighted, blind and blindsighted observers: *NeuroReport* 11, 3269–3273 (2000).
- Kuhn, R. L. A landscape of consciousness: Toward a taxonomy of explanations and implications. *Prog. Biophys. Mol. Biol.* 190, 28–169 (2024).
- Sattin, D. et al. Theoretical Models of Consciousness: A Scoping Review. Brain Sci. 11, 535 (2021).
- Reuter-Lorenz, P. A., Herter, T. M. & Guitton, D. Control of Reflexive Saccades following Hemispherectomy. J. Cogn. Neurosci. 23, 1368–1378 (2011).
- Herter, T. M. & Guitton, D. Saccades to the seeing visual hemifield in hemidecorticate patients exhibit task-dependent reaction times and hypometria. *Exp. Brain Res.* 182, 11– 25 (2007).
- Schalk, G. *et al.* Facephenes and rainbows: Causal evidence for functional and anatomical specificity of face and color processing in the human brain. *Proc. Natl. Acad. Sci.* 114, 12285–12290 (2017).
- Silvanto, J., Cowey, A. & Walsh, V. Inducing conscious perception of colour in blindsight. *Curr. Biol.* 18, R950–R951 (2008).
- 9. Germine, M. Consciousness and synchronicity. Med. Hypotheses 36, 277-283 (1991).
- 10. Stoica, O. C. Are Mental States Nonlocal? Mind Matter 18, 175-218 (2020).
- Descartes, R., Cottingham, J. G., Stoothoff, R. & Murdoch, D. *The Philosophical Writings of Descartes*. (Cambridge university press, Cambridge London New York [etc.], 1985).
- Jones, M. Electromagnetic-Field Theories of Mind. J. Conscious. Stud. 20, 124–149 (2013).
- Libet, B. A testable field theory of mind-brain interaction. J. Conscious. Stud. 1, 119– 126 (1994).
- Lahav, N. & Neemeh, Z. A. A Relativistic Theory of Consciousness. Front. Psychol. 12, 704270 (2022).
- Mackel, C. E., Orrego-Gonzalez, E. E. & Vega, R. A. Awake Craniotomy and Intraoperative Musical Performance for Brain Tumor Surgery: Case Report and Literature Review. *Brain Tumor Res. Treat.* 11, 145 (2023).
- 16. Inseparable: Ten Years Joined at the Head. CBC Docs POV (2017).
- Tegmark, M. Importance of quantum decoherence in brain processes. *Phys. Rev. E* 61, 4194–4206 (2000).

- 18. Dallas Lifespan Brain Study (DLBS).
- https://fcon_1000.projects.nitrc.org/indi/retro/dlbs.html.
 19. Schrödinger, E. Discussion of Probability Relations between Separated Systems. *Math. Proc. Camb. Philos. Soc.* 31, 555–563 (1935).
- Duman, D. & Tekin, M. Autosomal recessive nonsyndromic deafness genes: a review. Front. Biosci. 17, 2213 (2012).
- Lake, B. B. *et al.* Neuronal subtypes and diversity revealed by single-nucleus RNA sequencing of the human brain. *Science* 352, 1586–1590 (2016).
- Fagerberg, L. *et al.* Analysis of the Human Tissue-specific Expression by Genome-wide Integration of Transcriptomics and Antibody-based Proteomics. *Mol. Cell. Proteomics* 13, 397–406 (2014).
- Su, A. I. *et al.* A gene atlas of the mouse and human protein-encoding transcriptomes. *Proc. Natl. Acad. Sci.* 101, 6062–6067 (2004).
- Einstein, A., Podolsky, B. & Rosen, N. Can Quantum-Mechanical Description of Physical Reality Be Considered Complete? *Phys. Rev.* 47, 777–780 (1935).
- Feuillet, L., Dufour, H. & Pelletier, J. Brain of a white-collar worker. *The Lancet* 370, 262 (2007).
- Yu, F., Jiang, Q., Sun, X. & Zhang, R. A new case of complete primary cerebellar agenesis: clinical and imaging findings in a living patient. *Brain* 138, e353–e353 (2015).
- Pinto, Y., De Haan, E. H. F. & Lamme, V. A. F. The Split-Brain Phenomenon Revisited: A Single Conscious Agent with Split Perception. *Trends Cogn. Sci.* 21, 835–851 (2017).
- Shewmon, D. A., Holmes, G. L. & Byrne, P. A. Consciousness in congenitally decorticate children: developmental vegetative state as self-fulfilling prophecy. *Dev. Med. Child Neurol.* 41, 364–374 (1999).
- Adams, J. H. The neuropathology of the vegetative state after an acute brain insult. Brain 123, 1327–1338 (2000).
- Ammermann, H. et al. MRI brain lesion patterns in patients in anoxia-induced vegetative state. J. Neurol. Sci. 260, 65–70 (2007).
- Marner, L., Nyengaard, J. R., Tang, Y. & Pakkenberg, B. Marked loss of myelinated nerve fibers in the human brain with age. J. Comp. Neurol. 462, 144–152 (2003).
- 32. Einstein, A. Zur Elektrodynamik bewegter Körper. Ann. Phys. 322, 891–921 (1905).
- Lyu, D. *et al.* Causal evidence for the processing of bodily self in the anterior precuneus. *Neuron* 111, 2502-2512.e4 (2023).
- Eccles, J. A unitary hypothesis of mind-brain interaction in the cerebral cortex. Proc. R. Soc. Lond. B Biol. Sci. 240, 433–451 (1990).
- Yokawa, K. *et al.* Anaesthetics stop diverse plant organ movements, affect endocytic vesicle recycling and ROS homeostasis, and block action potentials in Venus flytraps. *Ann. Bot.* (2017).
- Baluška, F., Yokawa, K., Mancuso, S. & Baverstock, K. Understanding of anesthesia Why consciousness is essential for life and not based on genes. *Commun. Integr. Biol.* 9, e1238118 (2016).
- Urban, B. W. Current assessment of targets and theories of anaesthesia. Br. J. Anaesth. 89, 167–183 (2002).
- Purchase, I. F. H. The effect of halothane on the isolated cat heart. Br. J. Anaesth. 38, 80–91 (1966).
- Hudetz, A. G. General Anesthesia and Human Brain Connectivity. *Brain Connect.* 2, 291–302 (2012).
- Yaden, D. B. et al. Psychedelics and Consciousness: Distinctions, Demarcations, and Opportunities. Int. J. Neuropsychopharmacol. 24, 615–623 (2021).
- Banerjee, A. A. & Vaidya, V. A. Differential signaling signatures evoked by DOI versus lisuride stimulation of the 5-HT2A receptor. *Biochem. Biophys. Res. Commun.* 531, 609–614 (2020).
- Kwan, A. C., Olson, D. E., Preller, K. H. & Roth, B. L. The neural basis of psychedelic action. *Nat. Neurosci.* 25, 1407–1419 (2022).
- 43. Putnam, H. Representation and Reality. (MIT Press, Cambridge, Mass., 2011)
- 44. Searle, J. R. Consciousness and Language. (Cambridge University Press, New York, 2002).
- Chalmers, D. J. Does a rock implement every finite-state automaton? Synthese 108, 309– 333 (1996).
- 46. Copeland, B. J. What is computation? Synthese 108, 335-359 (1996).
- Sprevak, M. Triviality arguments about computational implementation. in *The Routledge Handbook of the Computational Mind* (eds. Sprevak, M. & Colombo, M.) 175–191 (Routledge, London: Routledge, 2018).
- 48. Lucas, J. R. Minds, Machines and Gödel. Philosophy 36, 112-127 (1961).
- Penrose, R. Shadows of the Mind: A Search for the Missing Science of Consciousness. (Oxford Univ. Press, Oxford, 1995).
- Shapiro, S. Mechanism, Truth, and Penrose's New Argument. J. Philos. Log. 32, 19–42 (2003).

Keywords: consciousness, faster than light, non-local, binding problem, neural correlates of consciousness, qualia, the hard problem of consciousness, computation

Supplementary Information

Article Title: Consciousness is faster than light. Author: Skyler P. Dillon (public key fingerprint: B00FFB33265B6E2356072F7917BD6099962D7186) e-mail: skylerdillon@hotmail.com

Brain Region (Human)	Brain Lesions (References)	Can unconsciousness be caused by the destruction of a single neuron or point in this brain region?
Spinal Cord	51-53	No
Medulla oblongata	54-70	No
Pons	71–95	No
Midbrain	96–114	No
Cerebellum	115–124	No
Diencephalon	125–148	No
Cerebrum	148–150	No
Lesions that do cause	151–153	No
unconsciousness		

Supplementary Table 1 | **Unconsciousness cannot be caused by the destruction of a single neuron or point in the human brain.** If all of your qualia, including your left and right visual fields, were bound together by neural activity converging onto a single neuron or point in the brain, then a brain lesion that specifically destroys that convergence point would cause you to become unconscious. I reviewed the literature; searching for brain lesions in every region of the human brain. I found that the symptoms of unilateral brain lesions were well documented for each region of the human brain and unconsciousness is not a symptom of a unilateral brain lesion. In other words, unconsciousness cannot be caused by the destruction of a single neuron or point in the human brain. Likewise, it is untenable to argue that your left and right visual fields are bound together by neural activity converging onto a single neuron or point in the brain.

Supplementary Information References

- Dro, P., Gschaedler, R., Dollfus, P., Komminoth, R. & Florange, W. Clinical and anatomical observation of a patient with a complete lesion at C1 with maintenance of a normal blood pressure during 40 minutes after the accident. *Spinal Cord* 20, 169–173 (1982).
- Jernman, R., Väänänen, A. & Kreivi, H.-R. Successful pregnancy and cesarean delivery in a tetraplegic, home-invasively-mechanically-ventilated patient – case report. *Spinal Cord Ser. Cases* 8, 62 (2022).
- Middleton, J. W. et al. Life expectancy after spinal cord injury: a 50-year study. Spinal Cord 50, 803–811 (2012).
- Alawadhi, A., Saint-Martin, C., Sabapathy, C., Sebire, G. & Shevell, M. Lateral Medullary Syndrome Due to Left Vertebral Artery Occlusion in a Boy Postflexion Neck Injury. *Child Neurol. Open* 6, 2329048X1986780 (2019).
- Day, G. S., Swartz, R. H., Chenkin, J., Shamji, A. I. & Frost, D. W. Lateral medullary syndrome: a diagnostic approach illustrated through case presentation and literature review. *CJEM* 16, 164–170 (2014).
- A. Fellner, F. & Hagleitner, G. Bilateral hypertrophic olivary degeneration after cerebral infarction. *Trauma Emerg. Care* 5, (2020).
- Jang, S. H. & Kim, M. S. Dysphagia in Lateral Medullary Syndrome: A Narrative Review. *Dysphagia* 36, 329–338 (2021).
- Kang, H. G. *et al.* Lateral Medullary Infarction with or without Extra-Lateral Medullary Lesions: What Is the Difference? *Cerebrovasc. Dis.* 45, 132–140 (2018).
- Kesav, P., Hussain, S. I., John, S., Sajjad, Z. & Jacob, A. Teaching NeuroImage: Reinhold Hemimedullary Syndrome. *Neurology* 100, 490–491 (2023).
- Kim, Y. K. et al. Topographic Consideration on the Occurrence of Ipsilesional Facial Paresis in Lateral Medullary Infarction. Cerebrovasc. Dis. 53, 38–45 (2024).
- Kumral, E., Afsar, N., Kirbas, D., Balkir, K. & Ozdemirkiran, T. Spectrum of medial medullary infarction: clinical and magnetic resonance imaging findings. *J. Neurol.* 249, 85–93 (2002).
- Nakano, H., Yanase, D. & Yamada, M. Syndrome of inappropriate secretion of antidiuretic hormone (SIADH) associated with lateral medullary syndrome: case report and literature review. *BMC Neurol.* 16, 119 (2016).
- Park, M. H. Lesional location of lateral medullary infarction presenting hiccups (singultus). *J. Neurol. Neurosurg. Psychiatry* 76, 95–98 (2005).
 Pongmoragot, J., Parthasarathy, S., Selchen, D. & Saposnik, G. Bilateral Medial
- Pongmoragot, J., Parthasarathy, S., Selchen, D. & Saposnik, G. Bilateral Medial Medullary Infarction: A Systematic Review. *J. Stroke Cerebrovasc. Dis.* 22, 775–780 (2013).
- Porto, F. H. G., Da Silva, S. P., Orsini, M., De Freitas, M. R. G. & De Freitas, G. R. Hemimedullary infarct with ipsilateral hemiplegia: A vertebral artery dissection syndrome? *J. Neurol. Sci.* 278, 135–137 (2009).
- 66. Sun, B. *et al.* Vertebral artery dissection induced lateral medullary syndrome characterized with severe bradycardia: a case report and review of the literature. *Ann. Palliat. Med.* **11**, 3330–3336 (2022).

- Thijs, R. D., Wijman, C. A. C., Van Dijk, G. W. & Van Gijn, J. A case of bilateral medial medullary infarction demonstrated by magnetic resonance imaging with diffusion-weighted imaging. *J. Neurol.* 248, 339–340 (2001).
- Tokuoka, K. et al. A case of bilateral medial medullary infarction presenting with 'heart appearance' sign. Tokai J. Exp. Clin. Med. 32, 99–102 (2007).
- Woischneck, D. et al. Respiratory function after lesions in medulla oblongata. Neurol. Res. 31, 1019–1022 (2009).
- Wu, S. *et al.* Neurotrophic keratopathy due to dorsolateral medullary infarction (Wallenberg Syndrome): case report and literature review. *BMC Neurol.* 14, 231 (2014).
- Abdelrasoul, A. A., Elsebaie, N. A., Gamaleldin, O. A., Khalifa, M. H. & Razek, A. A. K. A. Imaging of Brain Infarctions: Beyond the Usual Territories. *J. Comput. Assist. Tomogr.* 43, 443–451 (2019).
- Ayele, B. A., Tadesse, Y., Guta, B. & Zenebe, G. Millard-Gubler Syndrome Associated with Cerebellar Ataxia in a Patient with Isolated Paramedian Pontine Infarction – A Rarely Observed Combination with a Benign Prognosis: A Case Report. *Case Rep. Neurol.* 13, 239–245 (2021).
- Bery, A. K. & Chang, T.-P. Positive horizontal-canal head impulse test is not a benign sign for acute vestibular syndrome with hearing loss. *Front. Neurol.* 13, 941909 (2022).
- Felicio, A. C., Bichuetti, D. B., Marin, L. F., Dos Santos, W. A. C. & Godeiro-Junior, C. Bilateral Horizontal Gaze Palsy with Unilateral Peripheral Facial Paralysis Caused by Pontine Tegmentum Infarction. J. Stroke Cerebrovasc. Dis. 18, 244–246 (2009).
- Fok, A. & Barton, J. J. S. Teaching Video NeuroImage: Bilateral Horizontal Gaze Palsies With Vertical Ocular Dysmetria From a Demyelinating Lesion of the Pontine Tegmentum. *Neurology* 97, (2021).
- Gironell, A., De La Calzada, M. D., Sagales, T. & Barraquer-Bordas, L. Absence of REM sleep and altered non-REM sleep caused by a haematoma in the pontine tegmentum. *J. Neurol. Neurosurg. Psychiatry* 59, 195–196 (1995).
- Hayashi-Hayata, M. et al. Gasperini Syndrome, A Report of Two Cases. Intern. Med. 46, 129–133 (2007).
- Cheng, H.-C., Yen, M.-Y. & Wang, A.-G. Foville's syndrome with ipsilateral internuclear ophthalmoplegia due to spontaneous pontine hemorrhage. *Taiwan J. Ophthalmol.* 3, 75–77 (2013).
- Ishizawa, K., Ninomiya, M., Nakazato, Y., Yamamoto, T. & Araki, N. "Heart Appearance" Infarction of the Pons: A Case Report. *Case Rep. Radiol.* 2012, 1–2 (2012).
- Jacobs, A. *et al.* Diaschisis of specific cerebellar lobules: pontine haematoma studied with high-resolution PET and MRI. *J. Neurol.* 243, 131–136 (1996).
- Kumral, E., Dorukoğlu, M., Uzunoğlu, C. & Çetin, F. E. The clinical and cognitive spectrum of locked-in syndrome: 1-year follow-up of 100 patients. *Acta Neurol. Belg.* 122, 113–121 (2022).
- Kwak, S. & Chang, M. C. Impaired consciousness due to injury of the ascending reticular activating system in a patient with bilateral pontine infarction: A case report. *Transl. Neurosci.* 11, 264–268 (2020).

- Man, B. L. & Fu, Y. P. Raymond syndrome and conjugate gaze palsy from a paramedian pontine infarct. *BMJ Case Rep.* bcr2015211433 (2015) doi:10.1136/bcr-2015-211433.
- Massi, D. G., Nyassinde, J. & Ndiaye, M. M. Superior Foville syndrome due to pontine hemorrhage: a case report. *Pan Afr. Med. J.* 25, (2016).
- Matlis, A., Kleinman, Y. & Korn-Lubetzki, I. Millard-Gubler syndrome. AJNR Am. J. Neuroradiol. 15, 179–181 (1994).
- Ogawa, K. *et al.* Clinical Study of Seven Patients with Infarction in Territories of the Anterior Inferior Cerebellar Artery. *J. Stroke Cerebrovasc. Dis.* 26, 574–581 (2017).
- Regmi, P. R. & Amatya, I. A rare case of lateral pontine syndrome-Marie Foix syndrome. J. Patan Acad. Health Sci. 8, 69–72 (2021).
- Roquer, J., Lorenzo, J. L. & Pou, A. The anterior inferior cerebellar artery infarcts: a clinical-magnetic resonance imaging study. *Acta Neurol. Scand.* 97, 225–230 (2009).
 Schmahmann, J. D. The human basis pontis: motor syndromes and topographic
- organization. Brain 127, 1269–1291 (2004).
- Schnetzer, L. *et al.* Locked-in syndrome revisited. *Ther. Adv. Neurol. Disord.* 16, 175628642311608 (2023).
- Schnetzer, L. et al. The vascular locked-in and locked-in-plus syndrome: A retrospective case series. Ther. Adv. Neurol. Disord. 16, 17562864231207272 (2023).
- Tacik, P., Krasnianski, M., Alfieri, A. & Dressler, D. Brissaud-Sicard syndrome caused by a diffuse brainstem glioma. A rare differential diagnosis of hemifacial spasm. *Acta Neurochir. (Wien)* 156, 429–430 (2014).
- Van Eetvelde, R. et al. Imaging Features of Hypertrophic Olivary Degeneration. J. Belg. Soc. Radiol. 100, 71 (2016).
- Zaorsky, N. G. & Luo, J. J. A Case of Classic Raymond Syndrome. *Case Rep. Neurol.* Med. 2012, 1–3 (2012).
- Zhuang, S., Xie, W. & Mao, C. Bilateral facial colliculus syndrome caused by pontine tegmentum infarction: a case report. *BMC Neurol.* 21, 492 (2021).
- Adhikari, A. et al. Benedikt syndrome in a 74-year-old hypertensive woman: A case report. Clin. Case Rep. 10, e6767 (2022).
- Bandt, S. K., Anderson, D. & Biller, J. Deep brain stimulation as an effective treatment option for post–midbrain infarction-related tremor as it presents with Benedikt syndrome: Case report. J. Neurosurg. 109, 635–639 (2008).
- Deleu, D., Imam, Y. Z. B., Mesraoua, B. & Salem, K. Y. Vertical one-and-a-half syndrome with contralesional pseudo-abducens palsy in a patient with thalamomesencephalic stroke. J. Neurol. Sci. 312, 180–183 (2012).
- Fernandez, H. H., Friedman, J. H. & Centofanti, J. V. Benedikt's syndrome with delayed-onset rubral tremor and hemidystonia: A complication of tic douloureux surgery. *Mov. Disord.* 14, 695–697 (1999).
- 100. Fernandez, J. M., Sadaba, F., Villauerde, F. J., Alvaro, L. C. & Cortina, C. Cataplexy associated with midbrain lesion. *Neurology* 45, 393–394 (1995).
- George, J. et al. Case Report: Thalamomesencephalic stroke in a patient with HIV. F1000Research 9, 1250 (2021).
- 102. Hura, N. et al. Magnetic resonance imaging findings in Parinaud's syndrome: comparing pineal mass findings to other etiologies. Clin. Imaging 58, 170–176 (2019).
- 103. Li, X.-B., Feng, H., Dai, Y. & Liu, W. A Case of Comorbid Weber Syndrome Following Mechanical Thrombectomy for Middle Cerebral Artery Occlusion. *Risk Manag. Healthc. Policy* Volume 16, 1875–1880 (2023).
- 104. Maduri, R., Barbagallo, G., Iofrida, G., Signorelli, M. & Signorelli, F. Regression of Benedikt's syndrome after single-stage removal of mesencephalic cavernoma and temporal meningioma: A case report. *Clin. Neurol. Neurosurg.* **115**, 748–750 (2013).
- 105. Moro, E. & Albanese, A. Apomorphine and levodopa challenge in patients with a focal midbrain lesion. *Mov. Disord.* 14, 269–275 (1999).
- 106. Mossuto-Agatiello, L. Caudal paramedian midbrain syndrome. Neurology 66, 1668– 1671 (2006).
- 107. Parija, S., Lalitha, C. & Naik, S. Weber syndrome secondary to brain stem tuberculoma. *Indian J. Ophthalmol.* 66, 1036 (2018).
- 108. Puri, P. R. & Sijapati, A. Bilateral internuclear and internal ophthalmoplegia due to artery of Percheron infarction. *Clin. Case Rep.* 5, 280–284 (2017).
- 109. Ruchalski, K. & Hathout, G. M. A Medley of Midbrain Maladies: A Brief Review of Midbrain Anatomy and Syndromology for Radiologists. *Radiol. Res. Pract.* 2012, 1–11 (2012).
- 110. Survashe, P., Guthe, S., Velho, V. & Naik, H. Tectal tuberculoma: An unusual cause of Parinaud's Syndrome. Asian J. Neurosurg. 13, 400–402 (2018).
- 111. Swinkin, E. & Bui, E. Teaching Neuro Images : Acute Parinaud syndrome. Neurology 88, (2017).
- Vetrugno, R., D'Angelo, R., Alessandria, M., Mascalchi, M. & Montagna, P. Orthostatic Tremor in a left midbrain lesion. *Mov. Disord.* 25, 793–795 (2010).
- 113. Vuppala, A.-A. D. *et al.* MRI findings in Parinaud's syndrome: a closer look at pineal masses. *Neuroradiology* 61, 507–514 (2019).
- Witsch, J., Narula, R., Amin, H. & Schindler, J. L. Mystery Case: Bilateral Claude syndrome. *Neurology* 93, 599–600 (2019).
- 115. Choi, J. H. et al. Inferior cerebellar peduncular lesion causes a distinct vestibular syndrome. Eur. J. Neurol. 22, 1062–1067 (2015).
- 116. Di Lazzaro, V. Motor cortex changes in a patient with hemicerebellectomy.
- Electroencephalogr. Clin. Neurophysiol. 97, 259–263 (1995).
- 117. Estanol, B., Romero, R. & Corvera, J. Effects of Cerebellectomy on Eye Movements in Man. Arch. Neurol. 36, 281–284 (1979).
- 118. Kong, J., Lee, S.-U., Yu, S. & Kim, J.-S. Isolated Bilateral Superior Cerebellar Peduncular Lesion Presenting Square-Wave Jerks and Ataxia. J. Clin. Neurol. 19, 93 (2023).

- Okamoto, K. et al. MR features of diseases involving bilateral middle cerebellar peduncles. AJNR Am. J. Neuroradiol. 24, 1946–1954 (2003).
- 120. Olivito, G. *et al.* Bilateral effects of unilateral cerebellar lesions as detected by voxel based morphometry and diffusion imaging. *PLOS ONE* **12**, e0180439 (2017).
- 121. Smith, M. C. Histological findings after hemicerebellectomy in man: anterograde, retrograde and transneuronal degeneration. *Brain Res.* 95, 423–442 (1975).
- 122. Soares De Oliveira Wertheimer, G., Franco Da Graça, F. & Reis, F. Hypertrophic Olivary Degeneration Following Traumatic Lesion of the Superior Cerebellar Peduncle. *Neurol. India* 72, 232–233 (2024).
- 123. Yu, F., Jiang, Q., Sun, X. & Zhang, R. A new case of complete primary cerebellar agenesis: clinical and imaging findings in a living patient. *Brain* 138, e353–e353 (2015).
- 124. Zuma E Maia, F. & Luis, L. Inferior peduncle lesion presenting with bilaterally impaired vestibular responses to horizontal and posterior head impulses. *The Laryngoscope* 125, 2386–2387 (2015).
- 125. Caria, A. Hypothalamus, Neuropeptides and Socioemotional Behavior. Brain Sci. 13, 1303 (2023).
- 126. Chazen, J. L. et al. Clinical improvement associated with targeted interruption of the cerebellothalamic tract following MR-guided focused ultrasound for essential tremor. J. Neurosurg. 129, 315–323 (2018).
- 127. Cook, R. J., Fracchia, G., Hoban, P., Joffe, R. & O'Sullivan, D. Evolution of a surgical technique for posteroventral pallidotomy using CT/MR fusion and intraoperative macrostimulation. J. Clin. Neurosci. 5, 20–27 (1998).
- 128. Gallay, M. N., Moser, D., Magara, A. E., Haufler, F. & Jeanmonod, D. Bilateral MR-Guided Focused Ultrasound Pallidothalamic Tractotomy for Parkinson's Disease With 1-Year Follow-Up. *Front. Neurol.* **12**, 601153 (2021).
- 129. Hardy, J. Transsphenoidal Hypophysectomy. J. Neurosurg. 107, 459-471 (2007).
- Horisawa, S. *et al.* Unilateral pallidothalamic tractotomy for akinetic-rigid Parkinson's disease: a prospective open-label study. *J. Neurosurg.* 135, 799–805 (2021).
- 131. Jang, S. H. & Seo, Y. S. Neurogenic fever due to injury of the hypothalamus in a stroke patient: Case report. *Medicine (Baltimore)* 100, e24053 (2021).
- 132. Krahn, L. E., Boeve, B. F., Oliver, L. & Silber, M. H. Hypocretin (orexin) and melatonin values in a narcoleptic-like sleep disorder after pinealectomy. *Sleep Med.* 3, 521–523 (2002).
- 133. Krieg, S. M., Slawik, H., Meyer, B., Wiegand, M. & Stoffel, M. Sleep disturbance after pinealectomy in patients with pineocytoma WHO°I. *Acta Neurochir. (Wien)* 154, 1399– 1405 (2012).
- 134. Martínez-Fernández, R. et al. Randomized Trial of Focused Ultrasound Subthalamotomy for Parkinson's Disease. N. Engl. J. Med. 383, 2501–2513 (2020).
- 135. Obeso, I. et al. Unilateral subthalamotomy in Parkinson's disease: Cognitive, psychiatric and neuroimaging changes. Cortex 94, 39–48 (2017).
- 136. Patel, N. K. et al. Unilateral subthalamotomy in the treatment of Parkinson's disease. Brain 126, 1136–1145 (2003).
- Renard, D. et al. Thalamic Lesions: A Radiological Review. Behav. Neurol. 2014, 1–17 (2014).
- 138. Rodriguez-Rojas, R. et al. Subthalamotomy for Parkinson's disease: clinical outcome and topography of lesions. J. Neurol. Neurosurg. Psychiatry 89, 572–578 (2018).
- 139. Saeki, N. et al. Heavily T2-weighted MR imaging of white matter tracts in the hypothalamus: normal and pathologic demonstrations. AJNR Am. J. Neuroradiol. 22, 1468–1475 (2001).
- 140. Saleem, S. N., Said, A.-H. M. & Lee, D. H. Lesions of the Hypothalamus: MR Imaging Diagnostic Features. *RadioGraphics* 27, 1087–1108 (2007).
- 141. Schaller-Paule, M. A. *et al.* Isolated thalamic stroke analysis of clinical characteristics and asymmetry of lesion distribution in a retrospective cohort study. *Neurol. Res. Pract.* 3, 49 (2021).
- 142. Schmahmann, J. D. Vascular Syndromes of the Thalamus. *Stroke* **34**, 2264–2278 (2003).
- 143. Schreglmann, S. R. et al. Unilateral cerebellothalamic tract ablation in essential tremor by MRI-guided focused ultrasound. *Neurology* 88, 1329–1333 (2017).
- 144. Strelow, J. N. *et al.* Structural Connectivity of Subhalamic Nucleus Stimulation for Improving Freezing of Gait. J. Park. Dis. **12**, 1251–1267 (2022).
- 145. Wang, Z. et al. Case Report: Dysfunction of the Paraventricular Hypothalamic Nucleus Area Induces Hypersonnia in Patients. Front. Neurosci. 16, 830474 (2022).
- 146. Wilmskoetter, J. et al. Mapping acute lesion locations to physiological swallow impairments after stroke. *NeuroImage Clin.* 22, 101685 (2019).
- Ieong, I. *et al.* Long-term follow-up on Cushing disease patient after transsphenoidal surgery. *Ann. Pediatr. Endocrinol. Metab.* **19**, 164 (2014).
 Lew, S. M., Matthews, A. E., Hartman, A. L., Haranhalli, N., & on behalf of the Post-
- 148. Lew, S. M., Matthews, A. E., Hartman, A. L., Haranhalli, N., & on behalf of the Post-Hemispherectomy Hydrocephalus Workgroup. Posthemispherectomy hydrocephalus: Results of a comprehensive, multiinstitutional review. *Epilepsia* 54, 383–389 (2013).
- 149. Kliemann, D. et al. Intrinsic Functional Connectivity of the Brain in Adults with a Single Cerebral Hemisphere. Cell Rep. 29, 2398-2407.e4 (2019).
- 150. Smith, A. Speech and other functions after left (dominant) hemispherectomy. J. Neurol. Neurosurg. Psychiatry 29, 467–471 (1966).
- 151. Adams, J. H. The neuropathology of the vegetative state after an acute brain insult. *Brain* 123, 1327–1338 (2000).
- 152. Ammermann, H. et al. MRI brain lesion patterns in patients in anoxia-induced vegetative state. J. Neurol. Sci. 260, 65–70 (2007).
- Plum and Posner's Diagnosis of Stupor and Coma. (Oxford University Press, Oxford; New York, 2007).