

PHOSPHENE IMAGES OF THALAMIC SLEEP RHYTHMS INDUCED BY SELF-HYPNOSIS

by Philip T. Nicholson

ABSTRACT

A medical writer describes internally-generated light sensations (phosphenes) induced by a technique of self-hypnosis that combines relaxation, convergent eye movement, and attentive fixation. The phosphene images include: (1) a threshold sequence of receding annuli, (2) amorphous phosphene mists or clouds, and (3) phosphene clouds with two levels of brightness and color saturation. These images share some similarities with visions of light reported by religious mystics. Based on an analysis of the distinctive spatiotemporal characteristics exhibited by the phosphenes, the author proposes the hypothesis that they are generated by thalamic sleep rhythms oscillating in the lateral geniculate nucleus (LGN). Since humans usually lose consciousness at the onset of non-rapid-eye-movement sleep (NREMS), the author also proposes the hypothesis that his technique of phosphene induction preserves consciousness, despite the operation of thalamic sleep rhythms, because eye movements and attentive fixation send excitatory feedback to the visual pathways. This selective facilitation of visual neurons appears to preserve their signal-processing capacity even though synchronous sleep rhythms may be installed in the non-visual thalamus. The author speculates that this selective disruption of sleep rhythm activity in the visual pathways may be the mechanism that produces the cutaneous analgesia (hypnoanalgesia) he experiences when he induces phosphenes.

KEYWORDS: Attention, brain rhythms, eye movements, hypnosis, phosphenes, sleep, thalamus, vision

INTRODUCTION

To understand the mechanisms involved in the exercise of voluntary control over mental processes, it is important to learn more about how different kinds of attention affect local brain networks.¹ This paper describes how the author's manipulations of spatial attention during a relaxed, immobile, expectant state induce light sensations in the absence of an external light source ('phosphenes'). The endogenous mechanisms that produce this effect can be identified ad hoc by analyzing the spatiotemporal characteristics of these phosphenes.

A recent study suggests that an unexpectedly large number of people will self-induce phosphenes during relaxed, immobile expectancy.² After telling 28 subjects they would be exposed to an extremely-low-frequency electromagnetic field, the researchers only simulated their usual protocol without actually activating any electromagnetism. Despite the absence of an external stimulus, 90% of their subjects reported seeing phosphenes, mostly colored, moving phosphenes (73%). The researchers concluded that phosphene reports associated with exposures to electrical or electromagnetic fields did not warrant further investigation on the grounds that "the psychological component of the perception of electric and magnetic phosphenes must not be underestimated." However, their finding raises another question, one the researchers themselves do not ask, but which will be the focus of our investigation: Why do so many subjects report seeing phosphenes while waiting in a state of immobile expectancy? If these subjects did in fact see phosphenes, what endogenous mechanisms might account for this phenomenon?

In a recent positron emission tomography (PET) study of mental visualization, subjects were asked to rest in a state of relaxed, immobile expectancy while the researchers took measurements of brain metabolism for a "baseline resting condition."³ They assumed this state would generate only minimal levels of neuronal activity in the visual cortex and thus would serve as a good base for comparison with brain metabolism during the performance of assigned tasks. The instructions (adopted from an earlier PET study⁴) were as follows: subjects were asked to remain (1) "supine," (2) "motionless," (3) "blindfolded with their eyes closed," (4) "not . . . to say anything, . . . to tense their muscles, or to change respiratory rhythms after inhalation of the tracer," and (4) "to 'have it

black in front of their mind's eye,' (*i.e.*, not to have any visual imagery)."³⁻⁴ A cortical EEG confirmed that all subjects had predominant alpha-rhythm, as is expected when subjects are relaxed with eyes closed. When they analyzed the results, the Kosslyn team was surprised to find that the baseline resting condition generated a significant increase in neuronal activity in the primary visual cortex, an increase that antedated the performance of the assigned visual tasks. The similarities between the behaviors used as the baseline resting condition in the PET studies and the behaviors used by the author to induce phosphenes suggests that similar mechanisms may be involved. In this paper, we hope to identify those mechanisms.

METHODS

The author, a fifty-year-old medical writer with no history of drug or alcohol abuse, uses autohypnosis to disperse pain during dental or medical procedures, to relieve muscle tension headaches, to warm his extremities, and to induce sleep. His method of autohypnotic induction resembles the generic "relaxation response" popularized by Benson but involves a more active manipulation of eye position and attention.⁵ To begin inducing phosphenes, the author lies on his back, closes his eyes, takes slow, deep, rhythmic breaths, and gives himself silent instructions used in autogenic training, such as "let yourself relax" or "concentrate on the breathing."⁶ If body or mind resists relaxing, he tries "progressive relaxation" techniques which involve tensing and relaxing the large muscles of the body in systematic sequence.⁷

Once noticeable relaxation is present, the author begins to manipulate eye position to accelerate dissociation. He converges his eyes with enough force to elicit a continuing sensation of "fullness" or "pressure" in the eyeball. At the same time, he tests which of two different eye positions produces the strongest feeling of having the attention "hone in" or "lock in" on the fixation point at the center of vision: (1) with eyes converged and slightly depressed, as if focusing on the tip of the nose, or (2) with eyes converged and elevated, as if focusing on the forehead or crown. If the eyes were opened in either of these positions, they would produce a non-fused image of the external world. (With long practice the response is conditioned so that a less forceful convergence is needed to produce the same effect.)

In addition to manipulating eye position, the author focuses all his attention on the fixation point at the center of the visual field, even though at this early stage in the phosphene induction process the visual field remains 'empty' of everything except random metabolic discharge referred from the retina—the *eigengrau*, or “self-generated gray light.” When the attention is fixated strongly enough, the author experiences auditory feedback—a distinctive *tinnitus cerebri* that seems part sound but also part vibration. This sensation originates inside the lower rim of the skull at about the level of the ears and feels as if it radiates upward on both sides. It sounds like “bird wings fluttering,” “the sputtering of an open wood fire,” or a “dull buzz.” The reader can experiment with eliciting a version of this same *tinnitus* by staring intently at some external object located just beyond the reach of an extended arm. With practice, this *tinnitus* can be sustained for long intervals and used as a feedback signal to mark the efficiency of dissociation. Long practice also conditions this response, so that a slight convergence of the eyes, a straight-forward stare, and fixed attention are sufficient to evoke the *tinnitus* and then the phosphenes. The author continues the stereotyped eye movements and attentive fixation until phosphenes appear and for the duration of the phosphene displays.

No less important than sustained convergence and attentive fixation is the ability to keep the level of arousal low and the mental field free of distractions. The author remains passive and ready to acquiesce in whatever happens. To minimize the distraction of unwanted thoughts, images, emotions, itches, or pains, he uses a traditional meditation strategy—not attempting to resist distracting content but rather treating its arrival with indifference, letting it drift freely in and out of consciousness. This disengagement from potentially disruptive thoughts, combined with steady breathing and the intense focusing of attention, preserves equanimity and maintains a low level of arousal.

Pain control requires adding another strategy. In response to a painful stimulus, the large muscles of the body usually tense involuntarily and fear of further pain breaks into awareness. The author counters these instinctive and involuntary reactions by deliberately posing the thought that the pain is not actually attached to the body, that the pain can be “given away” by allowing it to flow out of the body and to disperse in all directions, in which case the author need not take action in order to escape or avoid the pain. This mental strategy—in effect, a deliberate abandonment of the normal vigilance which is oriented

toward behaviorally significant external stimuli—results in a sudden, precipitous drop in tension and restoration of the former relaxed state. This relaxation occurs immediately after the muscles tense involuntarily in response to the painful stimuli. Keeping relaxed helps diffuse the pain sensations and the anxiety about pain. The anticipation that relaxation (and the relief it brings) can be quickly restored after the next painful stimulation creates confidence that events are not out of control, and that, therefore, it is not necessary to divert one's attention from the focus on the visual field that generates the phosphenes.

The author's phosphene induction method closely resembles the behaviors used as the baseline resting condition in the PET studies, except that in those studies subjects were not explicitly instructed to converge their eyes or to fixate their attention. However, the author's attempt to carry out their instructions—lying motionless with eyes closed and “having it black in front of the mind's eye”—results in his eyes focusing automatically on the near visual field, an operation which requires some convergence (although not as much as the author would normally use). His attention also focuses automatically on the center of the empty visual field. If the author's experience is typical, then the two sets of behaviors may be virtually the same with differences being in degree, not in kind.

OBSERVATIONS

PHOSPHENE “RECEDING ANNULI”

The threshold sequence of phosphene images is illustrated in Figure 1. Just before the appearance of phosphene, there is a fleeting sensation of a movement inward from 360° of the peripheral visual field (VF), as if a dark annular wave were moving through the dark background with so little contrast that it is almost imperceptible. This annular wave suddenly illuminates while still in the far periphery of the VF (at about 80 - 60° of isoeccentricity), presenting a bright, thin phosphene annulus, yellow-green in color, that decreases in diameter at a constant rate, preserving its annular symmetry until it disappears into the centerpoint after 4 seconds of elapsed time (estimated by counting, “1001 . . . 1002 . . . etc.”). There is an interval

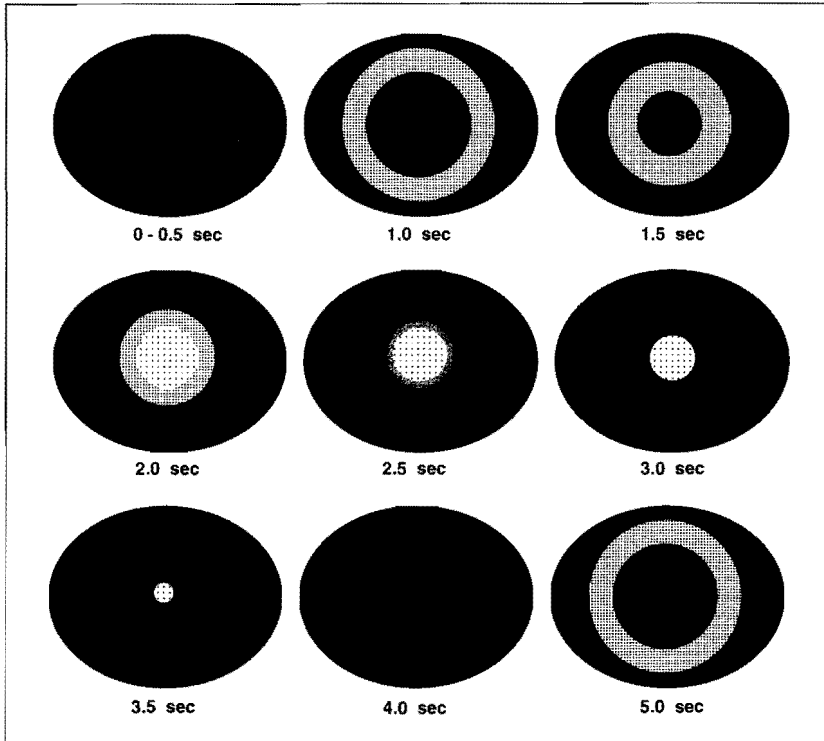


Figure 1. Schematic drawings of receding annuli phosphenes. At first there is a sensation of movement inward from 360° in the periphery of vision, then a bright, thin, yellow-green phosphene annulus suddenly illuminates at about 80° - 60° of isoeccentricity. The edges of the annulus are well-defined but more diffuse than this computer drawing program can depict—like a well-formed smoke ring. The annulus preserves its symmetry as it shrinks steadily in diameter at a constant rate of flow, creating the illusion that it is 'receding' toward the center of vision. At 2 seconds, the empty space inside the annulus fills abruptly with a disk of brighter phosphene. At 4 seconds, the image disappears into the centerpoint. At 5 seconds, another annulus sweeps into the periphery. The author sees a total of 4 'receding annuli.'

of 1 second, then the next wave appears at the periphery of the VF. Midway in the trajectory—at 2 seconds—the empty interior of the annulus fills abruptly with a disk of slightly brighter phosphene. A sequence of annuli usually terminates after 4 images, although the number of annuli can be increased by diverting the attention for a moment and then refocusing.

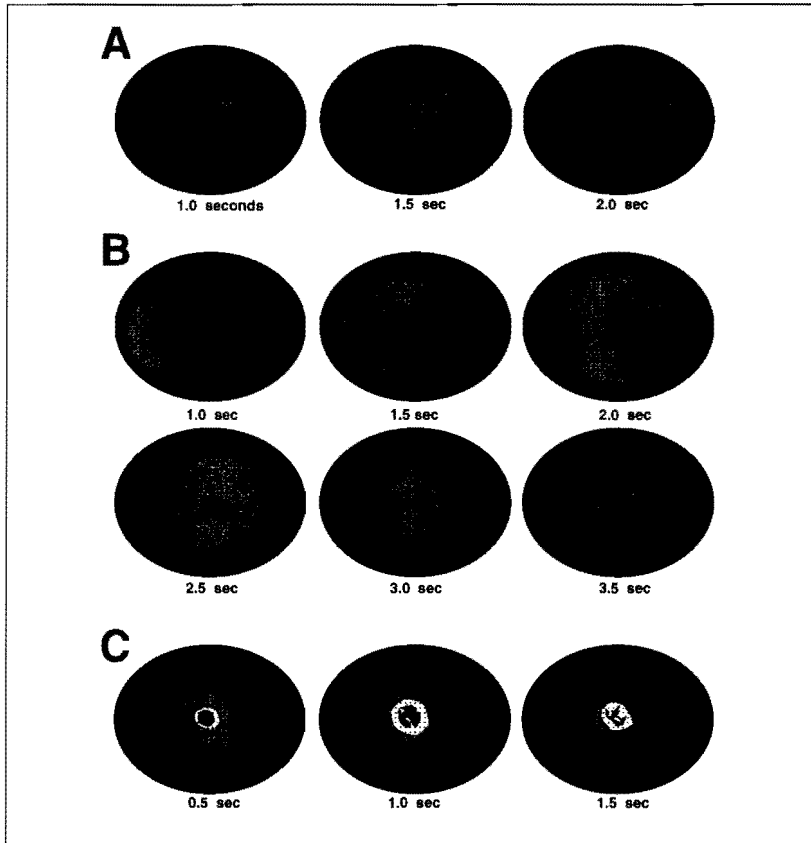
While the color of the annulus has remained the same over time, the color of the filling-in disk changed after about two years of intermittent phosphene induction from yellow-green to blue. The change began with tiny flecks of blue flashing amid the green just as the disk disappeared. Over a period of several weeks, the amount of blue expanded and appeared earlier in the fill-sequence. Within a month, the entire fill-disk was blue.

The shrinking annuli create a psychological illusion of a ring of light ‘receding’ toward a distant point in 3-dimensional space and ‘pulling’ the attention into a vortex, or, paradoxically, the same progression can create a sensation of falling backwards, an illusory motion that resembles an effect produced by optic flow: “Motion in the visual periphery elicits in the stationary observer an illusion of self-motion (vection) indistinguishable from real motion. . . . The illusion of vection is compelling, for it dominates contradictory proprioceptive signals. For example, subjects presented with optic flow consistent with backward self-motion perceive backward motion even if they are actually walking forward. . . .”⁸

The same receding annuli appear ‘spontaneously’ as hypnagogic images while the author is reclining and waiting for sleep. The receding annuli appear whenever the author’s mind and body are relaxed, his eyes closed, and his attention focused straight ahead—essentially the same behaviors used for self-hypnosis—but in the hypnagogic context, virtually no effort is required: the receding annuli feel as if they were ‘released spontaneously,’ not ‘induced.’ If the author is not relaxed, or if he is distracted by a headache or importuning thoughts, then the luminous receding annuli do not appear. Instead, the author loses consciousness abruptly at the onset of NREMS.

AMORPHOUS EXPANDING PHOSPHENES

- (A) **LOCAL, RANDOMLY-DISPERSED, EXPANDING WAVES.** Figure 2(A) illustrates one kind of amorphous phosphene ‘mist’ that appears after the receding annuli sequence terminates. These phosphenes are small, amorphous swirls of yellow-green that coalesce suddenly at unpredictable locations in the VF and expand for a second or two, then fade into the background. There are often several images drifting in the visual field at the same time, each in a different stage of evolution, and the density of the phosphene seems to vary, with some areas appearing thin and translucent, others more opaque—like puffs of smoke that



expand and curl aimlessly, then dissipate. Because these waves are so unpredictable and formless, the author is unable to predict the mean number of signals per episode, the period between signals, the site of entry or exit, or to define a typical trajectory other than in the abstract, generic manner used above. The signal-to-noise ratio in these phosphenes is very low: since phosphene stands out against the background, there clearly is a signal, but this signal presents so little pattern that it is essentially a form of phosphene noise. These localized pulsations may continue for several minutes, but usually within a minute or two they are superseded by the type of amorphous wave described next.

Figure 2 [facing page]. Schematic drawings of of amorphous, expanding phosphenes. (A) Brief, randomly-dispersed, localized pulses. These phosphenes are the first to appear after the receding annuli. They coalesce at unpredictable locations in the VF, expand asymmetrically for a second or two, then dissipate into eigengrau before there is time to extend very far beyond their locus of origin. (B) Periodic unidirectional amorphous expanding waves. These phosphene waves enter the periphery of vision from one hemifield, presenting as a loosely-defined crescent (hemi-annular) cloud that mirrors the curvature of the peripheral rim. The leading edge of the wave expands and flows across the vertical meridian into the opposite hemifield, swirling around the periphery, curling inward, and merging, while, at the same time, the trailing edge of phosphene dissipates rapidly in those regions over which the wave has already passed, like mist or smoke evaporating in air. The author is unable to time these waves, but their duration “feels roughly the same” as for receding annuli, i.e., about 4 seconds. Waves appear one after another in a cluster, with a refractory period between clusters during which no waves appear. After a prolonged session of self-hypnosis, the phosphene may not completely dissipate between successive waves, in which case the residual phosphene accumulates in a vaguely-circular cloud shape with a small area its center that undergoes continual perturbation: the phosphene pulls back, leaving a small dark circle at the center of the cloud, then fills back in, then pulls away again, ebbing and flowing in like manner until the cloud dissipates. At this stage, the image resembles an ‘eye’ with a phosphene ‘iris’ and a dark inner ‘pupil.’ (C) Two-tone phosphene. During a prolonged session, the phosphene at the very center of the amorphous cloud becomes much brighter, more fine-grained, almost opaque with highly saturated color. When a hole (‘pupil’) appears in the center of the cloud, forming an ‘eye-image,’ the brighter phosphene becomes a thin, bright rim lining the dark hole; from this bright inner rim, tiny promontories of bright phosphene flare out into the dark hole, twist and coil, and collide with other flares, forming webs and momentarily filling-in the dark hole, or receding back into the rim. The two-toned phosphene does not only appear in conjunction with the ‘eye-shape’ clouds; it also manifests at the center of a variety of elusive, ever-changing shapes. It is always restricted to the area of foveal vision.

- (B) **PERIODIC UNIDIRECTIONAL AMORPHOUS WAVES.** These phosphene waves sweep in from one hemifield, as shown in Figure 2(B). Often the wave has a vaguely crescent shape at the outset that conforms to the concave curvature of the peripheral VF, then the crescent shape gives way as the wave expands rapidly in divergent directions, flowing smoothly across the vertical meridian to envelop those regions of the VF that have not yet been illuminated. As the leading edge of the wave expands, the rear edges are already beginning to dissipate. The swirling movement and the contraction of outer boundaries leaves a cloud of phosphene near the center of the VF. This cloud may dissipate rapidly, or it may persist for several seconds, during which a pertur-

bation takes place at the center of the cloud: the phosphene ebbs away from this region, leaving a tiny dark hole, then the phosphene fills back in, then it ebbs and fills again—a fluctuation that continues as long as the phosphene cloud is visible. During the perturbations, the phosphene vaguely resembles the ‘iris’ of a human ‘eye,’ with an interior dark space as its ‘pupil.’ This ‘eye-image’ is seldom symmetrical, and its boundaries are always changing.

- (C) **TWO-TONE PHOSPHENE.** When the phosphenes are induced over a prolonged period of time, the phosphene in the central region representing foveal vision presents a more intensely bright, more opaque (finely-grained) character than the rest of the cloud, as shown in Figure 2(C). When this brilliant, opaque phosphene appears as part of an ‘eye-image,’ it forms a thin rim surrounding the ‘pupil.’ From this rim, flares jut out into the dark ‘pupil’ space, coil about, merge, or recede back into the rim.

The periodic, unidirectional, amorphous waves do not terminate after a stereotyped sequence; they can be evoked over prolonged time periods if the autohypnotic behaviors are maintained. The author is unable to retain the vision of these amorphous waves while simultaneously counting the number of seconds elapsed; it is his impression, however, that the duration of amorphous expanding waves “feels about the same” as for receding annuli, that is, a duration of about 4 seconds per wave. The amorphous waves usually appear in clusters, followed by a refractory interval during which no phosphenes appear, even if induction behaviors are sustained.

PHOSPHENES SUPERIMPOSED ON EXTERNAL OBJECTS

The author can observe amorphous phosphene images with his eyes open if he is in a room dark enough that colors cannot be distinguished, or if he is in a lighted room but one where he can keep his eyes diffusely focused on a relatively undifferentiated surface, such as a wall or ceiling with a light and uniform color and no distracting decorative adornments. In open-eye viewings, phosphenes appear as faint, translucent waves of yellow-green ‘mist’ superimposed over the surface or in the dark space that fills the darkened room. If the author attempts to focus on the features of external objects, the phosphenes disappear.

RECEDING ANNULI EVOKED BY MAGNETIC RESONANCE IMAGING (MRI)

As a patient undergoing an MRI scan, the author observed an unusual sequence of receding annuli. He was not relaxed, as in a normal induction, but rather anxious about his medical condition and about being confined in the narrow MRI tube. To keep calm, he concentrated on breathing slowly and stared straight ahead. While the sound of MRI machine was audible, the author observed a stream of receding annuli that differed from the usual sequence (self-induced and hypnagogic) in three ways: (1) the MRI annuli did not terminate at 4 cycles but continued for as long as the author kept his attention focused; (2) the MRI annuli were only yellow-green in color even though, in other inductions, the fill-in disk had already changed from green to blue; and, (3) MRI annuli completed their trajectories in half the normal time, disappearing in 2 seconds instead of 4. An interval of 2 seconds between radio pulses is often selected as optimal interval for capture of the signals emitted by hyperexcited atoms in the subject's body during the precessional relaxation.⁹⁻¹⁰ These observations suggest that energy released in the wake of the radio bombardment during MRI moves through the LGN as a standing wave in the same way that spindle bursts move, but that the 2-second duration of the MRI wave is not long enough to activate the neurons that generate a blue sensation.

THE ANESTHETIC EFFECT ASSOCIATED WITH SELF-INDUCED PHOSPHENES

On several occasions the author has preferred to undergo minor surgery without local anesthetic and to rely instead on autohypnotic analgesia—for example, during extraction of an infected abscess from the back, during laser surgery to remove keloids, and during the filling of dental caries. His experience on a recent occasion, when a dentist had to abrade the surface of six teeth with damaged enamel in order to create a strong bonding surface, are worth describing here, because the experience implies that spindle waves can be induced even during a state of high physiological arousal. For this appointment, the author arrived in the office too late to hypnotize himself in advance,

so he had to begin the normal induction procedures after the dental work was already underway.

As the dentist worked on the first tooth, the author experienced apprehension, sharp sensations of pain, and an involuntary tensing of the large muscles of the body with each application of the drill. As the author kept his eyes converged and his attention focused on the center of the visual field, the involuntary tensing in response to pain alternated with a precipitous relaxing of those same muscles immediately after the stimulus. With the repeated relaxation came a sense of relief and of “letting go” that ameliorated the pain. The pain-driven relaxation was much stronger than the relaxation that normally accompanies the author’s self-induction of phosphenes, and the alternation of a pain-driven tensing, on one hand, and the distancing effect of focusing attention on the visual field, on the other hand, resulted in a rapid dissociation from all external and internal stimuli, including a significant amelioration of painful sensations.

Most striking is the fact that, by the time the dentist reached the third tooth—after only a few minutes of this pain-driven self-hypnosis—the author saw a normal sequence of 4 receding annuli, followed by swirling clouds of amorphous, expanding phosphene, even though the dentist was still using the drill contemporaneously. This experience suggests that thalamic sleep rhythms can be voluntarily induced even when the subject’s level of physiological arousal is high.

SIMILARITIES WITH ATTENTION-INDUCED PHOSPHENES REPORTED BY OTHER OBSERVERS

Taxonomies of phosphene images always include examples of simple geometric phosphenes with circular or semi-circular shapes (disks, annuli, or crescents) as well as formless phosphene mists.¹¹⁻¹⁴ There are also a number of anecdotal accounts that are relevant, for example, Friedman, an ophthalmologist who studied “entoptic images” by introspection, describes a similar sequence of annuli induced by sustained inward concentration: “Circular waves of blue color may be seen in the dark by the dark adapted retina. They start at the

periphery and move inward toward the macula as a concentrically contracting solid circle, disappear at the macula and begin again at the periphery.”¹⁵

A valuable but often-neglected resource of information about phosphene imagery is presented in the literature of religious mysticism. Many mystics reported having seen translucent, colored lights while absorbed in prayer or meditation. A list of descriptions excerpted from self-reports is presented in Table I. (These excerpts are representative of phosphene images that appear during states of calm and inward-oriented concentration; they do not include images associated with paroxysmal raptures.) These phosphene images exhibit a relatively limited set of shapes and colors: the shapes are either circular (e.g., “rings,” “disks,” “wheels,” “suns”) or formless (e.g., “mists,” “snow,” “smoke”), and the colors are usually a variant of either green or blue. The phosphene visions of mystics display shapes and colors that are comparable to the images described by the author; this, and the many similarities in the behaviors used to induce phosphenes, suggest that both sets of phosphenes have a common etiology.

DISCUSSION

Whether the author uses self-hypnosis to induce phosphenes or experiences them as hypnagogic images, the threshold sequence of receding annuli has the same characteristics. In both these instances, the antecedent behaviors involve a state of low physiological arousal combined with mental withdrawal from external stimuli. These parallels suggest the hypothesis that phosphene displays are related to sleep-wake transitions.

PART I: RECEDING ANNULI AND THALAMIC SPINDLES

Mechanisms controlling the transition from waking to NREMS. Based on the work of several research teams,¹⁶⁻²⁰ a consensus scenario for the generation of NREMS has emerged. In this scenario, NREMS is the product of three brain rhythms that interact with each other in thalamocorticothalamic circuits. During the drowsy transition to sleep, which in humans is called ‘stage 1’ NREMS, the low voltage activity in the cortical electroencephalograph (EEG) still shows a pattern of intermittent desynchronization. The polarization of

Table I
Excerpts from self-reports of light visions by religious mystics.*

HEBREW MYSTICS

- "... the appearance of the *wheels* . . . was like the gleaming of *beryl*; and the *four* had the same form, their construction being something like a *wheel within a wheel*" Ezekiel
- "... like a *dome*, shining like *crystal*, spread out about their heads . . . And above the dome, something like a throne, in appearance like *sapphire*. . ." Ezekiel
- "... a *round ladder* . . . like a full *sphere*, rolling back and forth before him . . . bright *blue*." R. Abulafia
- "... A glowing light . . . *clear* brilliance . . . A *purple* light that absorbs all lights. . . ." R. Moses de Leon

HINDU AND BUDDHIST MYSTICS

- "... a perfectly *round*, beautiful *deep-blue* shape, of a size appropriate to the center of a mandala, as if exquisitely painted, of *extreme clarity*..." Tsong Khapa
- "... a *luminous revolving disc*, studded with lights . . . a *lotus flower* in full bloom . . ." Gopi Krishna
- "... both my eyes became centered . . . When this happened, a *blue light* arose in my eyes . . . like a *candle flame without a wick*, and stood motionless in the ajna chakra." S. Muktananda

MUSLIM MYSTICS

- "... [like] *chandeliers*...sublime *lights*, [like] stars, *moon*, or the *sun*..." Sharafuddin al-Maneri
- "... its color is *deep blue*; it seems to be an *upsurge*, like . . . *water from a spring*." Najmoddin Kobra
- "... visualize yourself as lying at the bottom of a *well* [looking up at the light of the opening] and the well . . . in lively downward movement." Najmoddin Kobra
- "... the *color green* is the...suprasensory uniting all the suprasensories." Alaoddawleh Semnani
- "... the light rises in the Sky of the heart taking the form of . . . *light-giving moons* . . ." Najm Razi

CHRISTIAN MYSTICS

- "... his own state at...prayer resembles . . . a *sapphire*; it is as *clear* and bright as the sky." Evagrius
- "... descending like a *bright cloud of mist* . . . like a *sun*, *round as a circle*." Simeon Neotheologos
- "... saw something in the air near him. He did not understand the type of thing, but in some ways it appeared to have the form of a serpent, with *many things that shone like eyes*, although they were *not eyes*." Ignatius of Loyola
- "... the soul puts on . . . a *green almilla* [cape worn on shoulders, beneath armor] . . ." John of the Cross
- "... [the almilla is like a helmet which] covers all the senses of the head of the soul . . . It has *one hole* through which the eyes may look upwards . . ." John of the Cross
- "... a *round* thing, about the size of a *rixdaler*, all bright and *clear with light like a crystal*." H. Hayen
- "... I saw a *bright light*, and in this light the figure of a man the color of *sapphire*...blazing with a gentle glowing fire . . . the three were in *one light*. . ." Hildegard von Bingen
- "... saw a *blazing fire*, incomprehensible, inextinguishable . . . with a *flame* in it the *color of the sky* . . ." Hildegard von Bingen
- "... saw placed beneath her feet the whole machine of the world as if it were a *wheel*. She saw herself placed above it, her eyes of contemplation magnetized towards the incomprehensible . . ." Beatrice of Nazareth
- "... I saw the *eyes* . . . I do not know if I was asleep or awake. . ." Angela of Foligno
- "... kind of sunlit, winged being . . . like a *child's head* beneath two little wings." Abbess Thaisia
- "... *round patterns*: *small circles would expand and eventually dissipate, only to be followed by other small circles of light*." Philip St. Romain
- "... an extraordinary *circle of gold light* . . . pulsating against a *deep violet* background. . . . There were *always four or five*. . . . *As soon as one would fade, another would appear*. . ." Philip St. Romain

*Italics added to mark words referring to (1) rings appearing in sets of four, (2) amorphous shapes such as clouds or flames, (3) circular or eye-like shapes, (4) colors from the center of the visible spectrum (gold, yellow, yellow-green, green, bluish green), and (5) colors from the lower end of the visible spectrum (blue, purple, violet).

cell membranes (V_m) has already begun to drop ('hyperpolarize') in the thalamic reticular nucleus (RTN), a thin sheath of GABAergic inhibitory neurons that acts as a pacemaker. When the V_m of RTN cell membranes drops to approximately $V_m = -60$ millivolts (mV), RTN cells stop firing single spikes, which are associated with waking states, and instead begin firing high-amplitude, 7-14 Hertz (Hz) spike-bursts. These bursts wax and wane over 1 to 3 seconds—hence the name 'spindle'—and recur at 3 to 10 seconds. Spindle volleys are relayed across the cortical mantle by thalamocortical (TC) sensory relay cells. In human studies, the primary criterion for onset of the first fully-established episode of NREMS, or 'stage 2 sleep,' is the presence of globalized spindles in the cortical EEG.

When spindle bursts from the RTN stimulate TC cells to fire in synchrony, this synchronous bursting pattern "overwhelms" the normal capacity of TC cells to relay afferent sensory signals. The loss of consciousness at the onset of stage 2 NREMS is thought to be caused by this blocking effect.²¹

While the RTN is firing spindle bursts through the thalamus, another process is activated in the cortex: large numbers of cells across the cortical mantle begin to oscillate at a very low frequency (< 1 Hz). The cortical slow rhythm, referred back to the thalamus via corticothalamic circuits, intensifies the hyperpolarizing effect of the RTN spindle bursts on TC cell membranes. When $V_m = -75$ mV, the TC cells begin to generate their own intrinsic calcium currents, which further increases hyperpolarization. At $V_m = -90$ mV, the RTN neurons stop firing spindle bursts. The TC cells continue to fire low-threshold calcium spikes in patterns which are controlled by the cortical slow rhythm. The interaction of these two oscillations generates the delta waves (1 to 4 Hz) which are associated with stage 2 NREMS.

In a study of sleep rhythms using the cortical EEG, Uchida, Atsumi, and Kojima found that the mean number of spindles required to induce delta wave activity (that is, to induce $V_m = -90$ mV) is 4.78 ± 1.62 spindles (from 3 to 7 spindles).²² The mean number of spindles in a volley remains the same for a single individual over multiple trials.

Based on the consensus scenario for generation of NREMS, we can predict that a phosphene image generated as an epiphenomenon of a thalamic spindle

Table II

Data show the degree of correspondance between the pulse patterns of thalamic spindles based on intracellular research and the spatiotemporal patterns of receding annuli phosphenes induced by the author using a technique of self-hypnosis.

	Receding annuli phosphenes	Thalamic spindle bursts
Mean number of signals	4 annuli	4.87 (\pm 1.6) bursts
Spatial extension vs. duration of signal	Thin band (\approx 0.5 -1.5 cm)	1 - 3 seconds
Periodicity of signals	5 seconds	3 - 10 seconds
Simultaneity in LGNs	Yes	Yes
Point of entry	Peripheral VF	Ventral & lateral LGN
Trajectory	Bilateral concentric	Unknown
Rate of flow	Constant rate	Unknown
Signal-to-noise ratio	High (sharp, bright, discrete)	High (short bursts)
Termination of sequence	After \approx 4 signals	After 4.87 (\pm 1.6) signals

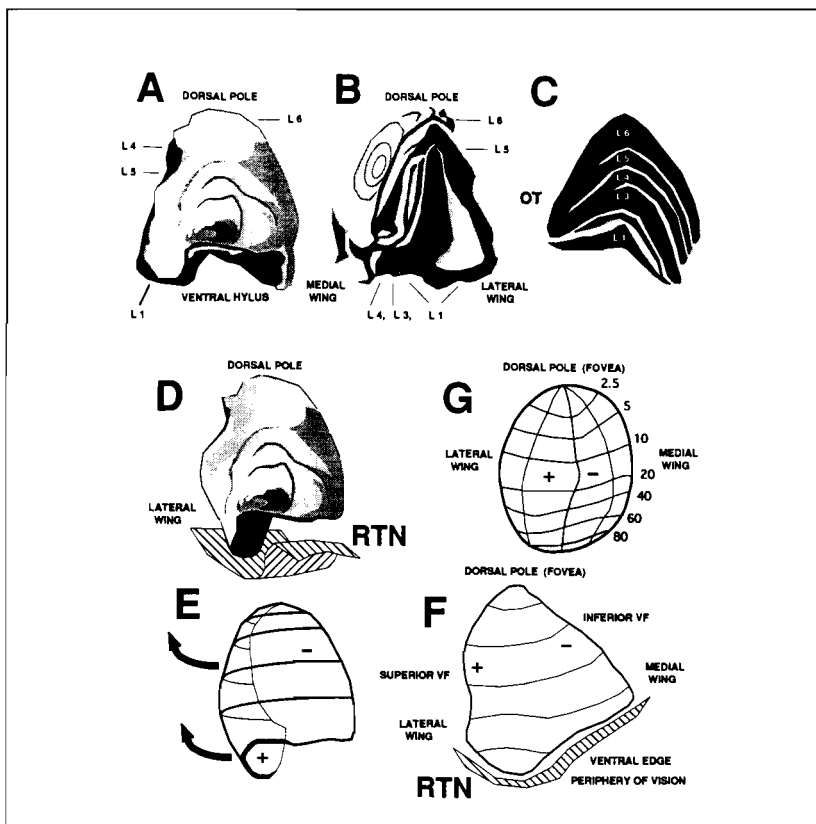
will present the following characteristics: (1) a high-definition, burst-like signal, (2) that recurs within 3 to 10 seconds, (3) that follows a trajectory compatible with having entered the LGN from the RTN, (4) that has a predictable mean for the individual subject, and a range that falls somewhere between 2 to 7 annuli, and (5) that the spindles will terminate automatically.

The temporal patterns of receding annuli and thalamic spindle bursts are similar. Table II illustrates the close match between the timing of receding annuli phosphenes and thalamic spindle bursts. The most striking similarities are the mean number of signals required to induce termination (4 annuli versus 4.87 spindles) and the interval between signals (5 seconds for annuli versus 3-to-10 seconds for spindle bursts). Neither alpha, theta, nor beta waves present this kind of stereotyped temporal pattern.

The spatial features of receding annuli are functions of geniculate anatomy. A spindle burst moving through the LGN to the cortex will acquire certain distinctive spatiotemporal characteristics because of the unique curvilinear and laminar anatomy of the LGN. A drawing of a macaque LGN based on a clay model constructed by Le Gros Clark²³ is shown in Figure 3. The primate LGN is composed of six sheets of cells, or laminae, that fold over a central hylus. In macaques, the hylus rises steeply as it extends rostrocausally, forming a high arch at the posterior pole; in humans, the hylus is much more shallow, but the functional anatomy is thought to be essentially the same in both cases.²³⁻²⁴ The geometry of the arch forces each lamina to bend so as to constitute, in effect, one-half of a conical surface, and each lamina with its hemi-conical shape contains visual relay cells representing the contralateral half of the visual field (VF). Operating as a pair, the complementary hemicones of the right and left LGN form the functional equivalent of a single conical surface which represents the entire VF. This conical geometry of the paired LGNs means a burst of otherwise formless excitation will acquire a distinctive shape as it propagates through the laminae of the LGN. Imagine a horizontal section moving along the surface of a cone from base to apex: that moving section forms an annulus that shrinks in diameter as it approaches the apex but retains its annular symmetry throughout its trajectory.

Figure 3 also includes drawings that show how Connolly and Van Essen²⁴ converted the 3-dimensional structure of lamina 6, the largest and most dorsal layer, into a 2-dimensional map. Connolly and Van Essen used data obtained by Malpelli and Baker²⁵ to superimpose a grid that specifies the areas in lamina 6 that contain TC cells representing various degrees of isoeccentricity in the VF. We will use this map at a later point to show how the movement of a spindle wave through a pair of LGNs correlates with what appears in the VF.

The location of the RTN relative to the LGN is also shown in Figure 3. The RTN spindle pacemaker is a thin sheath of GABAergic inhibitory cells that extends along the surface of the whole posterior ventral thalamus, not just the LGN. This extension brings the RTN into close proximity with the portion of the LGN ('lateral wing') that protrudes out from the main body of the thalamus. Some of RTN wraps around and beneath the ventral edges of the LGN.²⁶ The RTN comes closest to the LGN in two areas: along the ventral edges of the longest laminae (lamina 6 and 1), and along the edge of the lateral

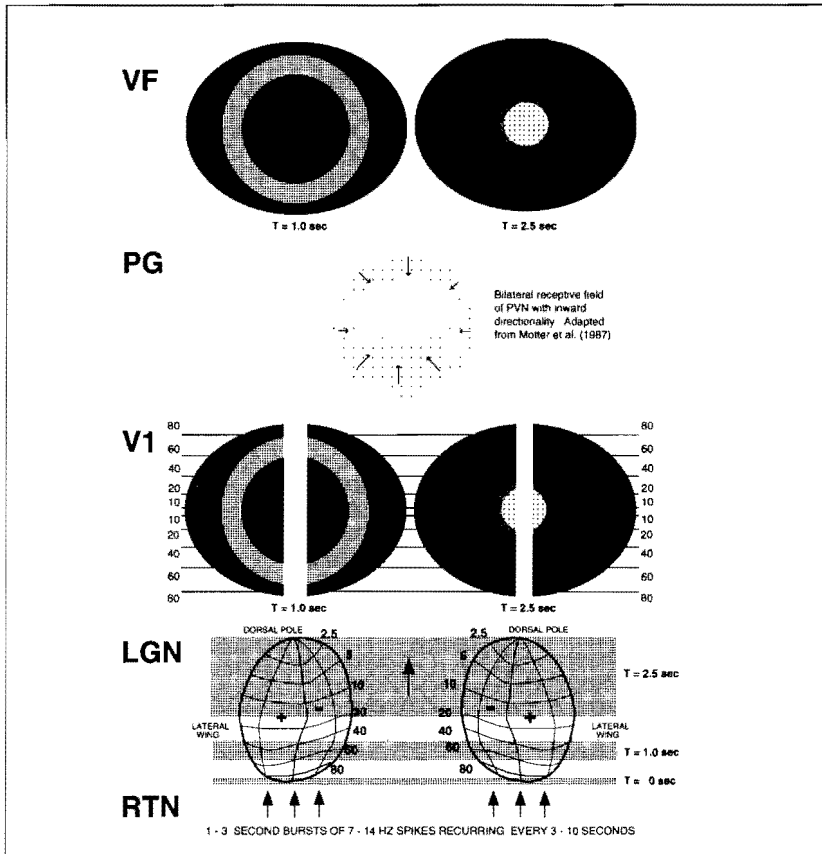


wing. These areas contain TC cells that represent the far periphery of the VF. If a spindle burst enters the LGN at locations where laminae are in closest proximity to the RTN—along the ventral edges—then the discharge of TC cells will begin in the most ventral portions of the laminae, the regions that represent the far periphery of the VF. This matches the phosphene sequence observed by the author.

Our hypothesis about how this proximity between RTN and LGN affects the trajectory of spindle waves is illustrated in Figure 4. We propose that it is not possible for the author to observe a thin annulus of phosphene sweeping in from all 360° of the peripheral VF unless spindle bursts exhibit the following

Figure 3 [facing page]. Drawings of primate geniculate anatomy and functional anatomy relevant to the generation of phosphenes. (A) Anterior view of a macaque monkey LGN based on a clay model reconstruction by Le Gros Clark.²³ (B) Posterior view of the same model showing 6 laminae folded over a central hylus to form lateral and medial wings, with the hylus arching steeply upward near the posterior pole to shape the laminae into layers of hollow half-cones nested together. Note that the lateral wing of the LGN is slightly larger than the medial wing, so that in situ the lateral wing protrudes out from the main body of the thalamus, creating a small knob visible during dissection.²⁶ (C) Schematic drawing of an oblique section through median axis (also adapted from Le Gros Clark)²³ showing how the laminae fuse together at the anterior pole. The afferent ganglion cell axons that form the optic tract (OT) penetrate through the anterior surface to reach their target relay cells in the laminae. (D) Drawing of the most dorsal lamina—layer 6—separated from the rest of the LGN. The geometric shape is a hollow half-cone, so that right and left LGNs placed back-to-back form, in effect, a single cone. Note also the position of the RTN, shown here as a striped bar, relative to lamina 6. The position of the striped bar shows that the RTN makes direct contact with lamina 6 at the edge of the lateral wing and also along the ventral edges. (E - F) Schematic drawings adapted from Connolly and Van Essen²⁴ that show how a 3-dimensional lamina—lamina 6—can be transformed into a 2-dimensional, triangular figure: lamina 6 is removed and its wings unfolded (E), creating a slightly asymmetric triangular figure (F), which is adjusted to an oblong shape (G) in order to superimpose a grid matrix with minimal distortion. The grid plots locations of TC cells that represent various isoelevations and isoazimuths in the VF, locations derived from the experimental observations published by Malpeli and Baker.²⁵ Note the relatively large amounts of laminar surface allocated to central vision (0° - 30°) as compared to peripheral vision (30° - 80°).

characteristics: (1) Spindle bursts must move simultaneously through both geniculate nuclei. This pattern of movement is consistent with anatomical studies in primates that found axonal projections linking the right and left RTNs,²⁷⁻²⁸ and with experimental studies in cats that found spindles appearing simultaneously in both right and left thalami.²⁹ (2) Spindle waves must enter the ventral edges of the LGN first and move ventrodorsally in a highly coherent pattern—moving, in effect, as a standing wave. This pattern of movement is consistent with in vitro experiments using sagittal slices from a ferret LGN where spindle waves originated spontaneously at the edge of a slice, in an area which contains cells that represent peripheral vision, then propagated toward that area of the slice containing cells that represent central vision in the ferret.³⁰



To our knowledge, the LGN is the only anatomical structure in the visual system that has the capacity of passively forcing a wave of excitation to flow across a conical surface, thereby generating the sensation of a phosphene annulus that retains its symmetry as it shrinks in diameter. The retina has a circular anatomy, but it is not likely that the mechanical deformation of the retina would produce a wave that began simultaneously in all 360° of the retinal rim, nor that the symmetry of annular wave would be preserved as it flows inward toward the foveal region.

The hypothesis presented here—that there is a structure in the visual system that imprints a conical shape on standing waves that move through it—explains

Figure 4 [facing page]. Schematic drawing of contemporaneous events in selected regions of the visual processing hierarchy. (A) The left and right RTNs fire spindle bursts simultaneously. (B) The bursts pass through the right and left geniculate laminae as standing waves of depolarization, entering from the ventral edges and moving ventrodorsally in parallel. In order for the phosphene annulus to appear to be flowing at a constant rate, the spindle wave flowing through the LGN must expand in width as it ascends ventrodorsally, so that by the time the fill-in disk appears—when the center 30° of the VF is covered by the phosphene annulus—the spindle wave must have expanded enough to activate relay cells in the most dorsal 50% of lamina 6. (C) Signals representing two complementary hemi-annuli are kept separate in the primary visual cortices (area VI). (D) Signals representing the two hemi-annuli are integrated into signals of a single annulus when they reach area PG in the posterior parietal cortex, where the receptive fields of single parietal visual neurons have the capacity to register bilateral signals that exhibit ‘opponent vector movement’ (that is, signals converging from opposite directions along the central axes—as if an object were moving away from the viewer).⁴⁶ Neurons in area PG also integrate wavelength (color) signals from the extrastriate visual pathway with signals about brightness, motion, and depth from the parietal visual pathway to compose a representation of an object in space—in this case, of an annulus that appears to recede.³²

the author’s experience during MRI when he observed a continuous succession of receding annuli every 2 seconds: in this view, the energy of the electromagnetic bombardment, when released during precessional relaxation, flows through the geniculate laminae in the same way that the RTN spindle bursts do, that is, as a standing wave, and thus the visual epiphenomenon is the same in both cases if the subject keeps the eyes focused straight ahead and the attention fixated. In a study of phosphenes evoked by electricity in conjunction with hallucinogenic drugs, Knoll, Kugler, Höfer, and Lawder found that each subject sees specific kinds of phosphene at different frequencies, and that these frequency-specific images are stable enough to be reproduced after six months.³¹ They conclude that “. . . the tunability of the electrical stimulation frequencies within the EEG range to various definable phosphenes requires the existence of a ‘resonance system.’” One mechanism contributing to that resonance system may be the functional anatomy of the LGN.

The colors of receding annuli as functions of geniculate laminar anatomy. The colors of receding annuli phosphenes are consistent with the patterns of discharge spindle waves can be expected to stimulate as they move through

geniculate laminae. As shown in Figure 5, the six laminae differ (1) in the types of wavelength signals relayed, (2) in physical size, and (3) in the area of the VF represented.²⁴⁻²⁵ Only 4 out of 6 laminae ('parvocellular' layers 3—6) contain TC cells that relay wavelength signals; the other 2 laminae ('magnocellular' layers 1 and 2) do not encode wavelength. Among parvocellular laminae, only lamina 6, the longest and most dorsal layer, contains TC cells that can represent the far periphery of the VF (from 80° to 50° of isoeccentricity); therefore, when the spindle wave enters the peripheral VF, only those wavelengths relayed by lamina 6 will participate in composing a "cortical lightness record." (See Zeki)³²

In lamina 6, ganglion cell axons arriving from the retina carry two kinds of wavelength signals: (1) center-ON signals from medium-wavelength cones (M-cone center-ON), and long-wavelength cones (L-cone center-ON).³³⁻³⁵ Using the cone spectral sensitivity curves (adapted from Marks *et al.*³⁶), we average the wavelengths that elicit maximal responses for M-cones (535 nanometers) and L-cones (520 nm), which yields a mean wavelength value of 550 nm. In humans this wavelength generates a sensation of green or yellow-green, the sensation observed by the author.

As the spindle burst moves ventrodorsally, it reaches the dorsal areas in the geniculate laminae which contain TC cells representing the central 0° to 20° of isoeccentricity in the VF. All 4 parvocellular laminae have TC cells which are potentially capable of representing this central region of vision, but the author's observation that the color of the filling-in disk changed from green to blue implies that, in the early years of phosphene induction, the spindle wave must have remained close to the surface of the LGN, stimulating only laminae 5 and 6, which generates a sensation of yellow-green. When the fill-in disk was observed to change color from green to blue, the spindle wave must have been rechanneled so that it began to penetrate below laminae 5 and 6 and to activate laminae 3 and 4, the only laminae that relay 'blue-sensitive' signals (S-cone center-ON signals).³⁷ In light of the author's MRI experience, it seems likely that a spindle wave requires a minimum of 2 seconds to reach the 'blue-sensitive' cells of laminae 4; therefore, during MRI, when the receding annuli disappeared after 2 seconds, half the normal duration, the fill-disk had a green color, even though it had long since change to blue in other induction settings with a 4-second trajectory.

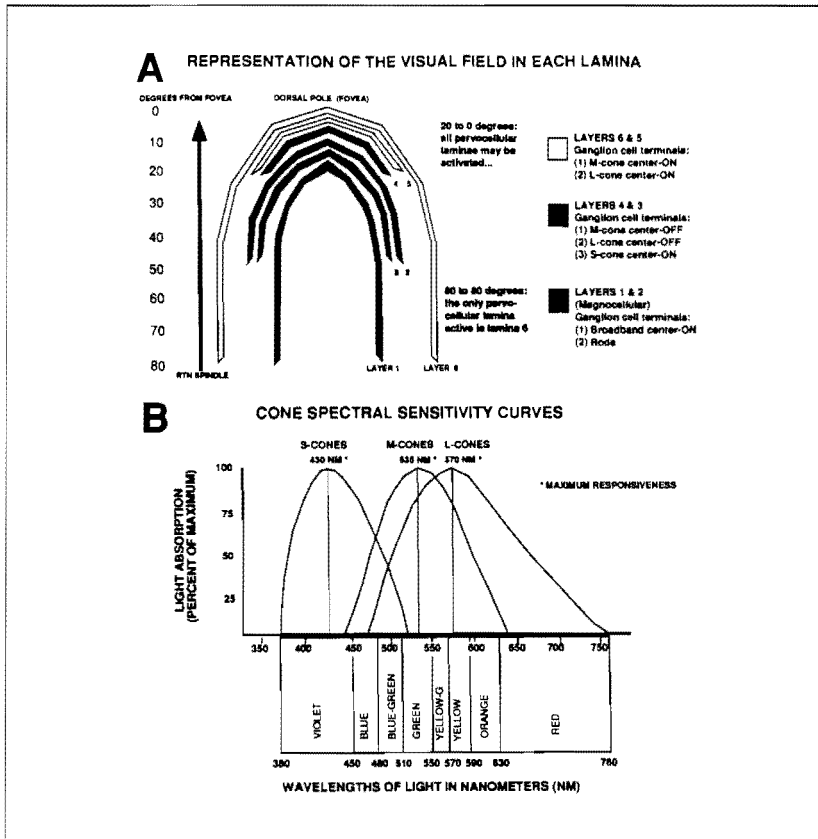


Figure 5. Phosphene color as a function of ganglion cell distribution in parvocellular laminae. (A) Schematic drawing of the proportion of the VF represented in each laminae. The proportions shown here are based on the work of Connolly and Van Essen.²⁴ The far periphery of vision (50° to 80° of isoeccentricity) is represented in only two laminae—laminae 1 and 6—and of these two, only lamina 6 is a parvocellular lamina capable of encoding wavelength signals. Therefore, only those wavelengths relayed by lamina 6 will participate in forming a cortical lightness record for the far periphery of the VF. (B) Cone spectral sensitivity curves (adapted from Marks et al.)³⁶ Using these cone sensitivity curves, we can estimate the sensation stimulated by a spindle wave moving through lamina 6 by averaging the wavelengths that elicit maximal responses for M-cones (535 nanometers) and L-cones (520 nm), which yields a mean wavelength value of 550 nm. In humans this generates a sensation of green or yellow-green, the sensation observed by the author when the receding annulus enters at the far periphery of the VF. For a calculation of the cortical lightness record when a spindle wave moves through 20° to 0° of the VF, stimulating all 4 parvocellular laminae, see the text and Figure 6.

Once the spindle wave penetrates to laminae 3 and 4, it may activate all 4 parvocellular laminae, stimulating TC cells to send contradictory wavelength signals for the same retinotopic location: the signals sent from laminae 4 and 3 will encode blue light (S-cone center-ON plus M-cone center-OFF and L-cone center-OFF), while the signals sent from laminae 5 and 6 encode yellow-green light (M-cone center-ON plus L-cone center-ON). This kind of contradiction does not occur in normal, retina-based perception where light energy striking the retina is structured by the retina's center-surround and color-opponent organization to encode only one set of wavelength signals for each spatial locus.³² In Figure 6, we estimate the cortical lightness record that results when contradictory wavelength signals compete to be represented in consciousness. Our calculations suggest that the outcome will be a mean wavelength which in humans elicits a dark blue or violet sensation.

PART II: AMORPHOUS PHOSPHENES & THALAMIC DELTA ACTIVITY

Mechanisms controlling the transition from spindle bursts to delta activity. In the consensus scenario for the generation of NREMS,¹⁶⁻²⁰ we noted that the RTN shifts to firing spindle bursts when polarization of thalamic cell membranes drops to $V_m = -60$ mV. At the same time, cortical cells begin oscillating with a synchronous slow rhythm (< 1 Hz). Both processes further decrease the V_m of TC cell membranes. When $V_m = -75$ mV, TC cells begin to generate their own intrinsic calcium currents, which also decreases V_m . At $V_m = -90$ mV, RTN neurons stop firing spindle bursts. At the same time, the TC cells begin firing low-threshold calcium spikes in alternation with after-hyperpolarizations (LTS-AHP). Because sensory relay neurons in the LGN are not linked to one another by locally projecting axons, the firing of one TC cell does not excite its neighbor. However, a lack of local connectivity does not mean that TC cells occupying the same laminae release their AHP-LTS calcium spikes in a random distribution; rather, spikes are released simultaneously by groups of cells in synchrony with the advent of the cortical slow wave. In effect, the distribution of LTS-AHP spikes firing in the LGN is determined by the spatial propagation of the cortical slow wave. Thus the delta wave (1 to 4 Hz) activity detected by cortical EEG during NREMS is a product of the interaction between the AHP-LTS calcium spikes released by TC cells and the cortical slow rhythm.

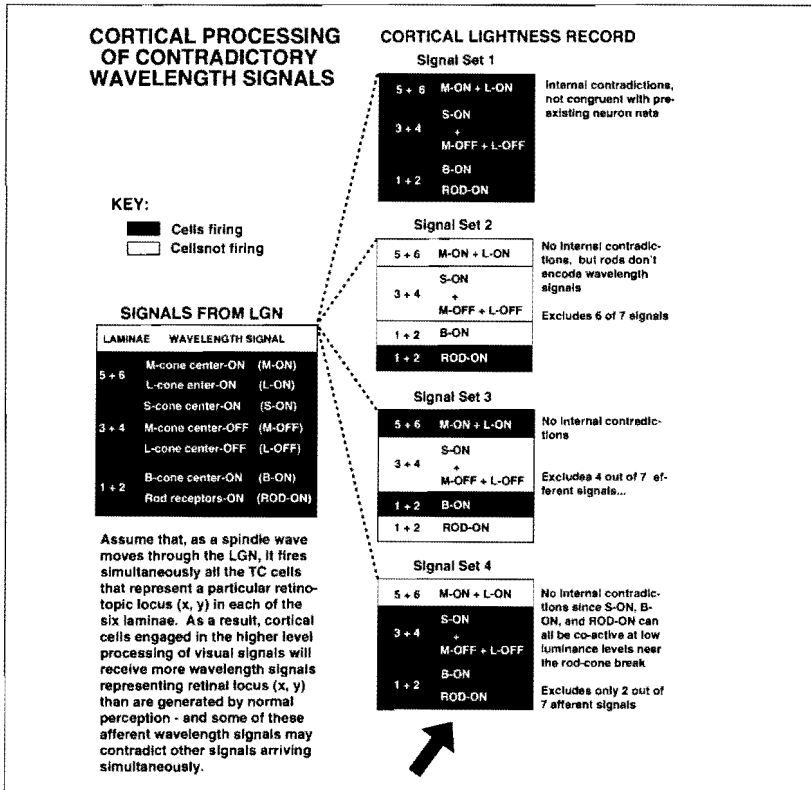


Figure 6. Selection of competing wavelength signals based on neuronal group selection. This thought experiment is based on Edelman's concept of neuronal group selection.⁷³ We assume that all possible combinations of signals will form sets and that all signal-sets will compete to make the most efficient match with pre-existing neuronal networks that have been forged over years of normal, retina-based perception. We also assume that pre-existing neural networks are most likely to expedite those sets that satisfy 2 criteria: (1) the signals in a set do not contradict each other, and (2) fewer signals are excluded than in the other sets that satisfy the first criterion. In our analysis, Set 4 wins the competition because it does not contain internal contradictions and excludes fewer signals (2 out of 7) than Set 3 excludes (4 out of 7). It is not an internal contradiction that Set 4 contains both rods and cone signals: in normal, retina-based perception, when luminance levels approach the 'rod-cone' break, S-cone center-ON receptors send signals, as do rod receptors, and there is even evidence that rod activity may actually drive S-cone activity even after luminance levels fall below the rod-cone break.⁷⁴ To calculate the cortical lightness record for Set 4, we average the wavelength that elicits maximal responses from rods (511 nm) and for S-cones (430 nm), which yields a mean wavelength of 470 nm, which in humans results in a dark-blue or violet sensation.

The cortical slow rhythm is an example of a generic pattern of neuronal excitation that occurs in large networks of neurons linked by local projections. A recent experiment by Maeda, Robinson, and Kwana³⁸ shows that networks of locally-connected neurons spontaneously generate periodic, synchronized bursts of calcium transients that propagate across the network in an expanding spatial wave, a pattern that represents “. . . a stable mode of firing towards which these neuronal networks ‘relax’ following perturbations, for example, by electronic stimulation or by drugs.” Understanding the characteristics of these waves is an important prerequisite to understanding the mechanisms that produce the amorphous phosphene images as a visual epiphenomenon.

To study the spontaneous, periodic, synchronized bursts, the researchers prepared cultures of dissociated cortical neurons and allowed them to mature for several days *in vitro* (DIV). After 3 to 4 DIV, when concentrations of extracellular magnesium were low, electrodes began to detect spontaneous, synchronized bursts of low frequency calcium transients (between 0.1 and 1 Hz) that originated in different locations on the sheet every 10 to 20 seconds and spread across the sheet of cells at a relatively slow speed, averaging 50 mm/second. The researchers could not predict where a wave would originate or where it would expand; rather, the initiation and expansion of waves was generated “by spatial and temporal summation of a continuous random background of synaptic inputs, including ‘miniature’ spontaneous synaptic events,” but the pattern was not random, since waves propagated “sequentially from electrode to electrode, as each local group of neurons ‘charges up’ its neighboring, nonrefractory areas, rather than in a random sequence of regions. . . .”³⁸

The excitability of the cells in the sheet determines the probability, first, that there will be a synchronous burst, and, second, that it will spread in a particular direction. Cells become more excitable as the cultures mature, which means the refractory intervals between successive bursts were much shorter in mature cultures (> 30 DIV) than in early cultures (< 7 DIV). Some cells became so insensitive to inhibition that they continued to fire bursts even when extracellular concentrations of magnesium were relatively high. Finally, there were refractory periods of 5 to 10 seconds during which no bursts occurred, even when an electrical stimulus was applied. This refractory interval for cultured networks is slightly longer than the refractory interval observed of the cortical slow wave *in vivo*, which 2.5 to 4 seconds.¹⁸⁻¹⁹

Based on the observations about cortical slow waves, we can predict that phosphenes generated as epiphenomena of delta wave activity will present the following signal characteristics: (1) signals that appear at unpredictable locations in the VF, (2) signals that expand and diverge asymmetrically, following unpredictable trajectories which may encompass large areas of the VF, (3) signals that move relatively slowly (50 mm/sec); (4) signals that recur with a period of 2 to 4 seconds, reflecting entrainment by the cortical slow wave, (5) signals that do not terminate automatically after a predictable number of cycles.

The spatial patterns of phosphene ‘mists’ and thalamic delta waves are similar. The spatiotemporal characteristics of amorphous phosphenes match the pattern of wave propagation associated with periodic, spontaneous synchronized bursts in large neuron networks: the amorphous phosphenes enter the visual field at unpredictable locations, expand and diverge asymmetrically with a slow, steady rate of flow, follow trajectories that are unpredictable, and exhibit a refractory interval. The amorphous mists do appear to have a periodicity, although the author is unable to sustain the vision while also counting the seconds; therefore, he is unable to determine if it compares to the period of the cortical slow wave (2.5 to 4 seconds), but his impression is that it “feels roughly the same as” as with receding annuli, which recur at 5 seconds. Also, the refractory interval feels as if it decreases as the induction session is extended. These similarities suggest that the amorphous phosphenes are generated by stage 2 NREMS delta wave activity, which is an activity governed by the reverberation of the cortical slow wave in thalamo-corticothalamic circuits. The cortical slow wave is referred to the LGN, where it modulates the firing of visual relay cells in the laminae, and since relay cells do not have local dendritic connections, the expansion of a wave of depolarizations moving from one relay cell to the next across in the laminar surface will mirror, at least in part, the expansion of the cortical wave that is spreading contemporaneously through locally-connected cortical cells.

PART III: RETENTION OF CONSCIOUSNESS

In a normal transition to NREMS, the volley of synchronous spindle bursts that marks the onset of stage 2 NREMS “overwhelms” the normal firing pattern

of thalamocortical cells, preventing their relay of afferent sensory signals, and inducing a loss of consciousness.²¹ But the loss of consciousness cannot be timed precisely using cortical EEG; the thalamus is too remote for the onset of spindling to be reliably detected by scalp electrodes.³⁹ In experiments with cats where electrodes could be implanted in the thalamus, researchers found that spindling often reached peak levels of activity in the thalamus—a level of activity consonant with stage 2 NREMS—at a time when the cortical EEG was still showing intermittent desynchronizations, a pattern characteristic of the drowsy stage 1 transition, not a fully-established stage 2 NREMS episode.⁴⁰ In other words, there is a short period of time in which the synchronous rhythms of stage 2 NREMS are installed in the thalamus but not in the cortex, where cortical neurons continue to depolarize in response to afferent sensory signals.

This finding suggests a possible explanation for the author's ability to retain consciousness while spindle bursts are sweeping through the LGN: if he is able to extend the duration of this time lag between the onset of spindling in the thalamus and spindling in the cortex, if he is able to stimulate the visual cortices in such a way that they continue to depolarize, then he may be able to keep on processing visual signals—to remain 'visually aware'—even after the non-visual thalamus and non-visual cortex become entrained by synchronous sleep rhythms. The author's induction technique includes several behaviors which are known to enhance the responsiveness of visual neurons—eye movements, attentive fixation, and an attitude of expectancy.

Effects of eye movements and attention on the LGN. Sustained convergence causes periorbital proprioceptors to send excitatory feedback directly to the LGN.⁴¹ Also, convergence further enhances the excitability of geniculate cells by stimulating the superior colliculus (SC), which sends its own excitatory signals to the LGN.⁴²⁻⁴³ These signals from the SC are known to selectively enhance the excitability of visual relay cells that represent the periphery of vision.⁴⁴ We know in the author's case that the SC is being stimulated, because he is able to keep on fixating, an ability which would normally be lost during the transition to NREMS.⁴⁵ The ability to fixate can be retained, however, if the SC is stimulated (even if stimulated during NREMS).⁴⁶

Effects of eye movement, attention, and expectancy on the primary visual cortex. The PET study of mental imagery by Kosslyn *et. al.*³ showed that even a “baseline resting state” of relaxed, immobile expectancy can generate significant increases in neuronal activity in the primary visual cortices. Further increases can be induced by sustained eye movements.

A sustained convergence will release a stream of ponto-geniculo-occipital (PGO) waves. These PGO waves, called ‘eye movement potentials,’ or EMPs, last only as long as the eye movement,^{17,47} whereas the PGO waves associated with rapid-eye-movement sleep (REMS) fire continually for the duration of the REMS episode. Both REMS-related PGO waves and EMPs have the same desynchronizing effect on the visual pathways: they move through the LGN, depolarizing the thalamocortical cells which relay the signals to cortical neurons. The responsivity of neurons in the primary visual cortices is enhanced by the stimulation of PGO waves.^{17,47-49} If the passage of EMPs causes the geniculate relay cells and their cortical targets to depolarize regularly, then a volley of 4 spindle bursts moving through the same pathways will register as just another series of depolarizations; in effect, the spindle bursts are processed by the visual system as if they were sensory signals sent from the retina rather than an event stimulated by endogenous mechanisms.

The impact of eye movements and fixation on the parietal visual cortex. Convergence and attentive fixation will also enhance the responsiveness of light-sensitive parietal visual neurons (PVNs) in area PG of the posterior parietal cortex.⁵⁰ In experiments where monkeys are trained to fix their attention on a screen—and to keep on fixating even if no targets appear—the excitability of these light-sensitive PVNs increases by as much as 350% .⁵¹ Responsiveness is also facilitated by the angle of gaze.⁵²⁻⁵³ Also, area PG is the first region in the visual pathways containing individual neurons with the capacity to respond to large, bilateral stimuli moving in opposite directions along the central meridians of vision (“opponent vector motion”).^{8,53-55} Facilitation of PVNs in area PG has the effect of sensitizing the subject to bilateral opponent vector motion, and this is especially true if the movement appears in the periphery of the VF.⁸

Intracortical amplification and feedback to the LGN. When PGO waves and signals from the SC arrive in the primary visual cortex, they may be relatively weak; however, weak signals can be amplified by reverberating in large networks

of cortical cells linked by local projection.⁵⁶ There are nine times as many excitatory links among cortical cells as there are projections from the visual cortex to the LGN, an imbalance which suggests leads Suarez, Koch, and Douglas to propose that “. . . geniculate input provides only a minor fraction of the excitatory input, the majority originating in neighboring and recurrently connected cortical cells.”⁵⁷ If the excitability of cortical neurons is “slightly higher” than normal, then this process of amplification may continue even after the LGN signals that initiated the reverberation are no longer being transmitted by the LGN (“ . . . a ‘hysteretic’ mode of operation”).⁵⁷

Feedback from hyperexcited cortical neurons will stream back to the LGN via corticogeniculate projections, which outnumber geniculocortical projections ten-to-one. One effect of this feedback is that the LGN is able to absorb large amounts of information flowing back from the cortex.⁵⁸ As signals reverberate back and forth in thalamocorticothalamic circuits, a “matching process” takes place: those geniculate relay cells that fire in patterns consistent with the firing patterns of active cortical cells will have their responsiveness enhanced, while those geniculate cells that fire in patterns not consistent with cortical cells will be suppressed.⁵⁹

Preservation of consciousness by selective facilitation of the visual pathways. By combining all of the facilitatory effects produced by the author’s eye movements and attentive fixation—(1) stimulation of the LGN by feedback from periorbital proprioception, both directly and indirectly via the SC, (2) the release of PGO waves (EMPs) that facilitate neurons in the LGN and primary visual cortex as they pass, (3) the facilitation of the primary visual cortex by a state of relaxed, immobile expectancy, (4) attentive fixation and angle of gaze that facilitate PVNs in area PG, (5) intracortical amplification of signals received from the LGN, and (6) corticogeniculate feedback that stimulates the LGN—we delineate a circuit of enhanced excitability that extends from the LGN through the post-geniculate visual hierarchy to area PG where signals are integrated into representations of objects.

The hyperexcitability of neurons all along the post-geniculate visual pathways creates conditions in the visual cortices which are similar to those present during EEG-desynchronized states of waking and REMS. Therefore, neurons in the visual pathways are too excitable to be entrained by synchronous bursting

patterns when spindle waves move through them; instead, the spindle bursts stimulate visual neurons to depolarize and then recover, as happens in response to afferent sensory signals during waking states. It is important to note that, for this to occur, none of the constituent neuron assemblies is required to perform an abnormal operation; the abnormality is a function of the author's intent to activate contemporaneously neuron assemblies that would not normally be co-activated.

PART IV: HYPNOTIC ANALGESIA

Manipulation of spatial attention as a method for alleviating pain sensations.

In a theoretical article on the neurophysiology of hypnosis, Crawford and Gruzelier⁶⁰ propose that variations in hypnotizability are function of variability in the efficiency of the frontolimbic (anterior) attentional system in humans: high-hypnotizables are better able to sustain focused attention on a task and to ignore extraneous stimuli than are low-hypnotizables.

In a related empirical study that compares the use of attention by subjects with high versus low hypnotizability, Crawford, Brown, and Moon⁶¹ report that “highly hypnotizable persons continue to show physiological reactivity while cognitively not perceiving the painful stimuli. . . Through disattentional processes, they can dissociate themselves from the awareness of pain.” The author's phosphene induction behaviors—and the cutaneous analgesia he experiences—appear to involve the same kind of behaviors, that is, a “mobilization of frontolimbic attention” used to withdraw attention from external stimuli and to concentrate it on internally-generated phosphene images. This suggests the hypothesis that the mechanisms that generate phosphenes might also produce hypnotic analgesia. There are several ways that visual signals and pain signals might interact to produce this analgesia.

Competition between pain and vision signals for limbic registration. The fully-developed sensation of pain requires integration of two kinds of information. Signals that encode the location and intensity of a noxious stimulus (nociceptive signals) are relayed via the thalamus to the somatosensory cortices, which registers only the “sensory-discriminative” aspects of pain perception;⁶² before reaching consciousness, the nociceptive signals must be sent to the limbic

system where they are linked with signals from long-term memory that encode the affective significance the stimulus has acquired for that individual. Studies of brain metabolism show this linking of somatosensory information with limbic associations takes place in the anterior cingulate (CG) and anterior insula.⁶²⁻⁶⁴

PET studies also show that the anterior CG is activated when subjects shift their spatial attention in response to expectations as to where stimuli will next appear.⁶⁵⁻⁶⁶ This is precisely what takes place during the state of immobile expectancy that induces phosphenes. Since visual signals and nociceptive signals both require limbic enhancement, which is only possible if they get access to the anterior CG, there might be conditions in which the two types of afferent sensory signals have to compete for that access. In a theoretical article on the mechanisms of attention, Posner and Rothbart⁶⁷ propose that visual signals can interfere with nociceptive signals by blocking access to the anterior CG. As behavioral evidence, they cite studies showing that when young infants make distress calls, they can be distracted by the presentation of a new visual stimulus: “We have shown the evidence of such control at about three months. Orienting to visual events can be employed to quiet or calm negative vocalizations. However, the distress appears to be maintained and reappears when the infant’s attention to the stimulus is reduced. Caregivers also report the use of visual orienting to block overt manifestations of distress at about this age.”⁶⁷

The effects of disattention on the somatosensory cortices. If there is a competition between visual signals and nociceptive signals for access to the anterior CG, it is likely that the competition will be affected by the relative volume of signals received. A recent study suggests that focusing attention on internally-generated visual stimuli may reduce the number of nociceptive signals relayed by the somatosensory cortices. Drevets, Burton, Videen, Snyder, Simpson, and Raichle⁶⁸ have shown that the mere *anticipation* of a touch at a specific cutaneous site increases the flow of blood to those cortical somatosensory neurons that are linked with that site where touch is expected, and decreases the flow of blood to neurons linked with sites where touch is not expected. In this experiment, the researchers designated touch as the “behaviorally significant sensory stimulus,” but their findings suggest that the outcome could be reversed if the instructions were reversed: if subjects were

instructed to direct attention *away* from a cutaneous site where a noxious stimulus was being applied, if they were instructed to relax, close their eyes, and concentrate on the play of phosphenes in the visual field as the “behaviorally significant sensory stimulus,” then it would be reasonable to expect that the flow of blood would decrease to the somatosensory neurons linked with the cutaneous stimulus, and that the flow of blood would increase to the visual cortices. The decrease in blood flow to somatosensory neurons implies less neuronal activity, which implies that fewer signals are being sent forward to the anterior CG than would have been sent if instead the subject were treating the touch as the behaviorally significant sensory stimulus. If fewer pain signals arrive at the anterior CG, it is reasonable to infer that the conscious experience of pain will be muted by the diversion of attention to internal visual stimuli.

Differential impact of sleep rhythms in the visual and nonvisual thalamus. There is another mechanism that might also contribute to the analgesic effect of inducing sleep rhythms. PET studies show that the thalamus is involved in the relay of nociceptive signals to the cortex,⁶³ although some confusion remains about which kinds of signals are relayed by which subregions in the thalamus.⁶² One thalamic site clearly identified by anatomical studies as a relay for nociceptive signals is the ventroposterior lateral nucleus (VPL).⁶⁹ If synchronous sleep rhythms are active in the thalamus, we can reasonably expect that the VPL will itself be entrained by synchronous oscillations, and that it will relay the sleep rhythms to somatosensory cortices. Relay neurons in the VPL are not stimulated by feedback from eye movements and fixation, as are relay neurons in the LGN, which suggests that sleep rhythms may impede the relay of nociceptive signals via the VPL but not the relay of visual signals via the LGN.

Brain waves associated with anesthesia and NREMS. The hypothesis presented here—that consciousness can be preserved despite the presence of NREMS activity—is consistent with a recent report that the kinds of fast brain waves which are normally associated with conscious states are also present during NREMS and anesthesia, two states which were thought to involve minimal brain activity.⁷⁰ Another recent study found that there is a close resemblance between the slow brain waves that appear during NREMS and those that appear during certain types of anesthesia.⁷¹

CONCLUSION

In this paper we have presented evidence supporting the hypothesis that phosphene images can be generated as an epiphenomena of thalamic spindle bursts and delta wave activity normally associated with NREMS . The prerequisites for inducing these sleep rhythm phosphenes are relaxed, immobile expectancy, sustained convergence, and attentive fixation of an undifferentiated visual field. Our hypothesis provides a necessary and sufficient explanation for the author's induction of phosphenes and perhaps also for the generation of phosphenes reported to appear 'spontaneously' during the drowsy transition to sleep ('hypnagogic states'), during sensory deprivation, or during certain kinds of prayer or meditation, all of which involve a similar state of relaxed, immobile expectancy.⁷²

• • •

CORRESPONDENCE: Philip T. Nicholson • 52 Norfolk Road • Chestnut Hill, MA 02167.

REFERENCES & NOTES

1. M. I. Posner, Modulation by Instruction, *Nature* 373 (1995), pp. 198-199.
2. J. Reissenweber, E. David, & M. Pfothenhauer, Investigations of Psychological Aspects of the Perception of Magnetophosphenes and Electrophosphenes, *Biomedizinische Technik* 37,3 (1992), pp. 42-45.
3. S. M. Kosslyn, W. L. Thompson, I. J. Kim & N. M. Alpert, Topographical Representations of Mental Images in Primary Visual Cortex, *Nature* 378 (1995), pp. 496-498.
4. P. E. Roland & B. Gulyas, Visual Memory, Visual Imagery, and Visual Recognition of Large Field Patterns by the Human Brain: Functional Anatomy by Positron Emission Tomography, *Cerebral Cortex* 5,1 (1995), pp. 79-93.
5. H. Benson, *The Relaxation Response*. (Morrow, New York, NY, 1975.)
6. J. H. Schultz & W. Luthe, *Autogenic Training: a Physiological Approach to Psychotherapy* (Grune & Stratton, New York, NY, 1969)
7. E. Jacobson, *Progressive Relaxation* (University of Chicago Press, Chicago, IL, 1938.)
8. M. A. Steinmetz, B.C. Motter, C.J. Duffy & V. B. Mountcastle Functional Properties of Parietal Visual Neurons: Radial Organization of Directionalities Within the Visual Field. *Journal of Neuroscience* 7,1 (1987) pp. 177-191.
9. R. R. Edelman, Magnetic Resonance Imaging of the Nervous System, *Discussions in Neuroscience* 7,1 Elsevier Science, Amsterdam (1990); also J. H. Newhouse & J. I. Wiener, *Understanding MRI*, 1st Ed. (Little Brown. Boston, MA, 1991).

10. J. W. Thorpe, G. J. Barker, S. J. Jones, I. Moseley, N. Losseff, D. G. MacManus, S. Webb, C. Mortimer, D. L. Plummer, P. S. Tofts, W. I. McDonald & D. H. Miller, Magnetisation Transfer Ratios and Transverse Magnetisation Decay Curves in Optic Neuritis: Correlation with Clinical Findings and Electrophysiology, *Journal of Neurology, Neurosurgery, and Psychiatry* 59 (1995), pp. 487-492.
11. M. J. Horowitz, *Image Formation and Cognition* (Appleton-Century-Crofts, New York, NY, 1978).
12. J. A. Ardis & P. McKellar, Hypnagogic Imagery and Mescaline, *Journal of Mental Science* 102 (1956), pp. 22-29.
13. M. Knoll, Anregung Geometrischer Figuren und Anderer Subjektiver Lichtmuster in Elektrischen Feldern, *Sweizerische Zeitschrift für Psychologie und ihre Anwendungen* 17 (1958), pp. 110-126.
14. M. Knoll, Subjective Light Pattern Spectroscopy in the Encephalographic Frequency Range, *Nature* 184 (1959), pp. 1823-1824
15. B. Friedman, Observations of Entoptic Phenomena, *Archives of Ophthalmology* 28 (1911), pp. 285-307.
16. D. A. McCormick & H. C. Pape, Properties of a Hyperpolarization-Activated Cation Current and Its Role in Rhythmic Oscillation in Thalamic Relay Neurons, *Journal of Physiology* (London) 431 (1990), pp. 291-318.
17. M. Steriade & R. W. McCarley, *Brainstem Control of Wakefulness and Sleep* (Plenum Publishing, New York, NY, 1990.)
18. M. Steriade, A. Nuñez & F. Amzica Intracellular Analysis of Relations Between the Slow (< 1 Hz) Neocortical Oscillation and Other Sleep Rhythms of the Electroencephalogram. *Journal of Neuroscience* 13,8 (1993), pp. 3266-3283.
19. M. Steriade, D. Contreras, R. Curró Dossi, & A. Nuñez, The Slow (< 1 Hz) Oscillation in Reticular Thalamic and Thalamic Cortical Neurons: Scenario of Sleep Rhythm Generation in Interacting Thalamic and Neocortical Networks, *Journal of Neuroscience* 13,8 (1993), pp. 3284-3299; also see M. Steriade, D. A. McCormick, & T. Sejnowski, Thalamic Oscillations in the Sleeping and Aroused Brain, *Science* 262 (1993), pp. 679-685.
20. M. von Krosigk, T. Bal, & D. A. McCormick, Cellular mechanisms of a synchronized Oscillation in the Thalamus, *Science* 261 (1993), pp. 361-364.
21. D. Contreras & M. Steriade Cellular Basis of EEG Slow Rhythms: A Study of Dynamic Corticothalamic Relationships, *Journal of Neuroscience* 15,1 (1995), pp. 604-622.
22. S. Uchida, Y. Atsumi, & T. Kojima, Dynamic Relationships Between Sleep Spindles and Delta Waves During a NREM Period, *Brain Research Bulletin* 33 (1994), pp. 351-355.
23. W. E. Le Gros Clark, The Laminar Organization and Cell Content of the Lateral Geniculate Body in the Monkey, *Journal of Anatomy* 75 (1940-41), pp. 419-433.
24. M. Connolly & D. Van Essen, The Representation of the Visual Field in Parvocellular and Magnocellular Layers of the Lateral Geniculate Nucleus in the Macaque Monkey, *Journal of Comparative Neurology* 226 (1984), pp. 544-564.
25. J. G. Malpelli & R. H. Baker, The Representation of the Visual Field in the Lateral Geniculate Nucleus of Macaca Mulatta, *Journal of Comparative Neurology* 161 (1975), pp. 569-594.

26. W. Kahle, *Color Atlas and Textbook of Human Anatomy, Vol. 3: Nervous System and Sensory Organs*, 4th ed. (Georg Thieme Verlag, Stuttgart, Germany, 1993) ; see also P. L. Williams & R. Warwick, *Functional Neuroanatomy of Man* (W. B. Saunders, Philadelphia, PA., 1975) .
27. D. Paré & M. Steriade, The Reticular Thalamic Nucleus Projects to the Contralateral Dorsal Thalamus in Macaque Monkeys, *Neuroscience Letters* 154 (1993), pp. 96-100
28. V. Raos & M. Bentivoglio, Crosstalk Between the Two Sides of the Thalamus Through the Reticular Nucleus: A Retrograde and Anterograde Tracing Study in the Rat, *Journal of Comparative Neurology* 332 (1993), pp. 145-154.
29. D. Contreras, A. Destexhe, T. J. Sejnowski & M. Steriade, Spatiotemporal Patterns of Spindle Oscillations in the Cortex and Thalamus, *Journal of Neuroscience* 17,3 (1997), pp. 1179- 1196.
30. U. Kim, T. Bal, D. McCormick, Spindle Waves are Propagating Synchronized Oscillations in the Ferret LGN in vitro, *Journal of Neurophysiology* 74,3 (1995), pp. 1301-1323.
31. M. Knoll, J. Kugler, O. Höfer, & S. D. Lawder , Effects of Chemical Stimulation of Electrically-Induced Phosphenes on Their Bandwidth, Shape, Number and Intensity, *Confinia Neurologia* 23 (1963), pp. 201-226.
32. S. Zeki, *A Vision of the Brain* (Blackwell Scientific Publications, London, 1993)
33. J. G. Malpelli & P. H. Schiller, Lack of Blue Off-Center Cells in the Visual System of the Monkey, *Brain Research* 141 (1978), pp. 385-389.
34. P. H. Schiller & J. G. Malpelli Functional Specificity of Lateral Geniculate Nucleus Laminae of the Rhesus Monkey, *Journal of Neurophysiology* 41 (1978), pp. 788-797.
35. D. M. Dacey, Physiology, Morphology, and Spatial Densities of Identified Ganglion Cell Types in Primate Retina, In *Higher-Order Processing in the Visual System* (Ciba Foundation Symposia 184, John Wiley and Sons, West Sussex, England, 1994), pp. 12-27.
36. W. Marks, W. Dobbelle & E. MacNichol, Visual Pigments of Simple Primate Cones, *Science* 143 (1961), p. 1181.
37. B. M. Dow, Nested Maps in Macaque Monkey Visual Cortex, In *Science of Vision* (K. N. Leibovic, Ed., Springer-Verlag, New York, NY, 1990), pp. 84-124.
38. E. Maeda, H. P. C. Robinson & A. Kwana, The Mechanisms of Generation and Propagation of Synchronous Bursting in Developing Networks of Cortical Neurons, *Journal of Neuroscience* 15,10 (1995), pp. 6834-6845.
39. M. Steriade, Cellular Substrates of Brain Rhythms, In *Electroencephalography: Basic Principles, Clinical Applications, and Related Fields*, 3rd Ed. (E. Niedermeyer & F. Lopez da Silva, Eds, Williams & Wilkins, Baltimore, MD, 1993), pp. 27-62.
40. M. Lancel, H. van Reizen & A. Glatt The Time Course of Sigma Activity and Slow-Wave Activity During NREMS in Cortical and Thalamic EEG of the Cat During Baseline and after 12 Hours of Wakefulness, *Brain Research* 596 (1992), pp. 285-295.
41. I. M. L. Donaldson & R. A. Dixon, Excitation of Units in the Lateral Geniculate and Contiguous Nuclei of the Cat by Stretch of the Extrinsic Ocular Muscles, *Experimental Brain Research* 38 (1980), pp. 245-255.
42. M. E. Goldberg & R. H. Wurtz, Activity of Superior Colliculus in Behaving Monkey: I. Visual Receptive Fields of Single Neurons, *Journal of Neurophysiology* 35 (1972), pp. 553-586.
43. J. S. Nelson, M. A. Meredith & B. E. Stein, Does an Extraocular Proprioceptive Signal Reach the Superior Colliculus? *Journal of Neuroscience* 62,6 (1989), pp. 1360-1374.

44. J. T. Xue, C. B. Y. Kim, R. J. Moore & P. D. Spear, Influence of the Superior Colliculus on Responses of Lateral Geniculate Neurons in the Cat, *Visual Neuroscience* 11 (1994), pp. 1059-1076.
45. V. Henn, R. W. Baloh & K. Hepp, The Sleep-Wake Transition in the Oculomotor System, *Experimental Brain Research* 54 (1984), pp. 166-176.
46. M. S. Raybourn & E. L. Keller Colliculoreticular Organization in Primate Oculomotor System, *Journal of Neurophysiology* 40,4 (1977), pp. 861-878.
47. S. Datta, Neuronal Activity in the Peribrachial Area: Relationship to Behavioral State Control, *Neuroscience and Biobehavioral Reviews* 19,1 (1995), pp. 67-84.
48. M. Steriade, Alertness, Quiet Sleep, and Dreaming. In *Cerebral Cortex*, Vol. 9 (A. Peters & E. G. Jones, Eds., Plenum Press, New York, NY, 1991), pp. 279-357.
49. R. H. Wurtz & C. W. Mohler, Enhancement of Visual Responses in Monkey Striate Cortex and Frontal Eye Fields, *Journal of Neurophysiology* 39 (1976), pp. 766-772.
50. J. S. Bazier, L. G. Ungerleider & R. Desimone, Organization of Inputs to the Inferior Temporal and Posterior Parietal Cortex in Macaque, *Journal of Neuroscience* 11,1 (1991), pp. 168-190.
51. V. B. Mountcastle, R. A. Andersen & B. C. Motter, The Influence of Attentive Fixation Upon the Excitability of the Light-Sensitive Neurons of the Posterior Parietal Cortex, *Journal of Neuroscience* 1,11 (1981), pp. 1218-1235.
52. R. A. Andersen, G. K. Essick & R. M. Siegel, Neurons of Area 7 Activated by Both Visual Stimuli and Oculomotor Behavior, *Experimental Brain Research* 67 (1987), pp. 316-322; see also R. A. Andersen & V. B. Mountcastle, The Influence of the Angle of Gaze Upon the Excitability of the Light-Sensitive Neurons of the Posterior Parietal Cortex, *Journal of Neuroscience* 3,3 (1983), pp. 532-548.
53. R. A. Andersen, R. M. Bracewell, S. Barash, J. W. Gnadt & Fogassi L, Eye Position Effects on Visual, Memory, and Saccade-Related Activity in Areas LIP and 7a of the Macaque, *Journal of Neuroscience* 10,4 (1990), pp. 1176-1196.
54. B. C. Motter, M. A. Steinmetz, C. J. Duffy & Mountcastle VB, Functional Properties of Partial Visual Neurons: Mechanisms of Directionality Along a Single Axis, *Journal of Neuroscience* 7,1 (1987), pp. 154-176.
55. R. A. Andersen, Visual and Eye Movement Functions of the Posterior Parietal Cortex, *Annual Reviews of Neuroscience* 12 (1989), pp. 377-403.
56. P. S. Churchland, V. S. Ramachandran & T. J. Sejnowski, A Critique of Pure Vision, In *Large-Scale Neuronal Theories of the Brain* (C. Koch & J. L. Davis, Eds., MIT Press Cambridge, MA, 1994), pp. 23-61.
57. H. Suarez, C. Koch & R. Douglas, Modeling Direction Selectivity of Simple Cells in Striate Visual Cortex Within the Framework of the Canonical Microcircuit, *Journal of Neuroscience* 15,10 (1995), pp. 6700-6719.
58. J. W. McClurkin, L. M. Optican & B. J. Richmond, Cortical Feedback Increases Visual Information Transmitted by Monkey Parvocellular Lateral Geniculate Nucleus Neurons, *Visual Neuroscience* 11 (1994), pp. 601-617.
59. A. Gove, S. Grossberg & E. Mingolla, Brightness Perception, Illusory Contours, and Corticogeniculate Feedback, *Visual Neuroscience* 12 (1995) 1027-1052.
60. H. J. Crawford & J. H. Gruzelier A Midstream View of the Neuropsychophysiology of Hypnosis: Recent Research and Future Directions, In *Contemporary Perspectives in Hypnosis Research* (E. Fromm & M. Nash, Eds., Guilford Press, New York, NY, 1992), pp. 227-266.

61. H. J. Crawford, A. M. Brown & C. E. Moon, Sustained Attentional and Disattentional Abilities: Differences Between Low and Highly Hypnotizable Persons, *Journal of Abnormal Psychology* 102,4 (1993), pp. 534-543.
62. R. C. Coghill, J. D. Talbot, A. C. Evans, E. Meyer, A. Gjedde, M. C. Bushnell & G. H. Duncan, Distributed Processing of Pain and Vibration by the Human Brain, *Journal of Neuroscience* 14,7 (1994), pp. 4095-4108.
63. A. K. P. Jones, W. D. Brown, K. J. Friston, L. Y. Qi & R. S. J. Frackowiack, Cortical and Subcortical Location of Response to Pain in Man Using Positron Emission Tomography, *Proceedings of the Royal Society of London, Series B: Biological Sciences* 244 (1991), pp. 39-44.
64. J. D. Talbot, S. Marrett, A. C. Evans, E. Meyer, M. C. Bushnell & G. H. Duncan, Multiple Representations of Pain in the Human Cerebral Cortex, *Science* 251 (1991), pp. 1355-1358.
65. M. Corbetta, F. M. Miezin, S. Dobmeyer, G. L. Shulman & S. E. Petersen, Selective and Divided Attention During Visual Discriminations of Shape, Color, and Speed: Functional Anatomy by Positron Emission Tomography, *Journal of Neuroscience* 11,8 (1991), pp. 2383-2402.
66. M. Corbetta, F. M. Miezin, G. L. Shulman, & S. E. Petersen, A PET Study of Visuospatial Attention, *Journal of Neuroscience* 13,3 (1993), pp. 1202-1226.
67. M. I. Posner & M. K. Rothbart, Constructing Neuronal Theories of Mind, In *Large-Scale Neuronal Theories of the Brain* (C. Koch & J. L. Davis, Eds., MIT Press, Cambridge, MA, 1994), pp. 183-200.
68. W. C. Drevets, H. Burton, T. O. Videen, A. Z. Snyder, J. R. Simpson & M. E. Raichle, Blood Flow Changes in Human Somatosensory Cortex During Anticipated Stimulation, *Nature* 373 (1995), pp. 249-252.
69. F. A. Lenz, M. Seike, Y. C. Lin, F. H. Baker, L. H. Rowland, R. H. Gracely & R. T. Richardson, Neurons in the Area of the Human Thalamic Nucleus Ventralis Caudalis Respond to Painful Heat Stimuli, *Brain Research* 623 (1993), pp. 235-240.
70. D. Contreras & M. Steriade, State-Dependent Fluctuations of Low-Frequency Rhythms in Cortico-Thalamic Networks, *Neuroscience* 76,1 (1997), pp. 25- 38.
71. M. Steriade, D. Contreras, F. Amzica & I. Timofeev, Synchronization of Fast (30-40 Hz) Spontaneous Oscillations in Intrathalamic and Thalamocortical Networks, *Journal of Neuroscience* 16,8 (1996), pp. 2788-2808; also see D. Contreras & M. Steriade, State-Dependent Fluctuations of Low-Frequency Rhythms in Corticothalamic Networks, *Neuroscience* 76,1 (1997), pp. 25-38.
72. A. Mavromatis, *Hypnagogia* (Routledge & Kegan Paul, London, 1987)
73. G. M. Edelman, *Neural Darwinism: The Theory of Neuronal Group Selection* (Basic Books, New York, NY, 1987).
74. U. Stabell & B. Stabell, Mechanisms of Chromatic Rod Vision in Scotopic Illumination, *Vision Research* 34,8 (1993), pp. 1019-1027.

∞ ∞ ∞