

MEDITATION, SLOW WAVE SLEEP AND ECSTATIC SEIZURES: *The Etiology of Kundalini Visions*

Philip Nicholson

ABSTRACT

This paper describes phosphene images observed by a medical writer during the onset and evolution of a partial seizure with an ecstatic emotional accompaniment. This seizure was inadvertently induced by the author's attempt to practice meditation during the early morning hours while in a sleep-deprived condition. A neurological workup did not find evidence of epileptic lesions or interictal activity. The phosphene sequence matches descriptions of light visions in the ancient Vedic scriptures and in yoga meditation texts of the Hindu and Tibetan Buddhist traditions, suggesting the possibility of a common etiology. Analysis of the phosphene spatiotemporal characteristics in light of recent research in the neuroscience of sleep, vision, and epilepsy suggests that the images were generated by the following sequence of neural events: (1) activation of slow wave sleep rhythm oscillators in corticohalamocortical circuits (CTC); (2) destabilization of sleep rhythm oscillators, triggering emergence of hypersynchronous spike-waves and fast runs in CTC circuits; (3) a build-up of rhythmical activity in the right hippocampus (H) due to the synergistic interaction of synchronous sharp waves, high-frequency ripples, and afferent visual stimuli; (4) an outbreak of paroxysmal discharges in the contralateral left H; and, (6) precipitation of a bilateral mesotemporal seizure.

KEYWORDS: Hippocampus, hippocampal commissures, lateral geniculate nucleus, phosphenes, religion and medicine, temporal lobe epilepsy, sleep, vision

INTRODUCTION

The concept of a subtle energy called kundalini, a relatively late development in the evolution of religious mysticism on the Indian subcontinent, is based primarily on metaphysical interpretations of visual experiences. In this introduction we summarize the ideas that gave rise to the concept of kundalini and its contemporary manifestations based on scholarly analyses of the ancient Vedic scriptures,¹⁻³ yoga meditation texts in the Hindu mainstream⁴⁻⁶ and esoteric Tantric traditions,⁷⁻⁹ histories of Buddhism,¹⁰ yoga meditation texts in the Tibetan Buddhist tradition,¹¹⁻¹⁴ and comparative analyses of meditation strategies in Hindu and Buddhist traditions.¹⁵

The ancient Vedic scriptures refer to a fourth state of consciousness different from waking, sleeping, or dreaming, a state called *turiya*.³ During the practice of meditation, humans enter *turiya* where they see the ceaseless transformations of a radiant energy that creates and sustains the cosmos, the Ultimate Reality, or *Brahman*. In the *Rg Veda*,^{1,4} a collection of hymns chanted to the Vedic gods compiled in written form between 1300 and 1000 B.C., the vision of Truth (*Rta*) is described as flowing into the world in the wake of the *dhitayah*, the “flame-arrows” and “lightnings” of Agni, God of Fire and Light. These flame-arrows “assemble like streams of water into holes (RV: 10, 25,4),” swirling around a central locus, then disappearing into it. In the *Upanishads*, or ‘Commentaries on the Vedas,’ written between 800 and 500 B.C., more elaborate metaphors are used to describe the cosmic lights: “Fog, smoke, sun, wind, fire, fireflies, lightning, crystal moon, these are the preliminary forms which produce the manifestation of Brahman in Yoga (*Svetasvatara* U. II: 11, p. 721).”³ Or this: “. . . a small lotus flower; within it is a small space. What is within—that should be sought, for that, assuredly, is what one should desire to understand (*Chandogya* U. VIII: 1: 1, p. 491).”³ Also, an important distinction is made more clear: “Verily, indeed, of the Brahma light there are these two forms, one, the tranquil and the other the bounding (*Maitri* U. VI: 36, p. 849).”

In Vedic metaphysics, each human is endowed with a small portion of the cosmic energy, the *atman* or *purusha* (‘person’ or ‘self’). Cultivation of the inner light is considered to be the highest goal in life. By practicing self-discipline and accumulating spiritual merit, a yogin nurtures and strengthens the *atman* to the point that it penetrates the barriers that confine it within the

physical body and merges in blissful union with the bright primordial radiance of Brahman. This merger of atman and Brahman is called by various names in Hindu traditions—‘aloneness (*kaivalpa*),’ ‘release’ (*moksha*), ‘union’ (*yoga*), and ‘Opening of the Divine Eye,’ called the *caksus* to differentiate this inner, spiritual eye from the physical eye.

New Hindu sects began to appear in the Indian subcontinent between 500 and 1,000 A.D. These sects taught esoteric ideas and practices, collectively called the *Tantras*. The yoga meditation texts of these Tantric sects added more detail to the descriptions of the cosmic lights in *turiya*.^{4,7-9} The lights are described as having shapes like wheels (*chakras*) and colors that change in predictable ways as a yogin makes progress on the path to enlightenment.

The progression of colored chakras was envisioned as a hierarchy of ‘subtle energy wheels’ (also called chakras) aligned in parallel with the physical body, as shown in Figure 1. The chakras are connected by three ‘subtle channels’ (*ida*, *pinghala*, and *sushumna*) through which the cosmic energy can flow. The cosmic energy rests in potential form in a reservoir at the base of the trunk (*kunda*, or ‘bowl’). Once ignited by the practice of yogic meditation, the newly-awakened kundalini energy rises up through the hierarchy of subtle energy centers, activating each chakra in succession—and the yogin in *turiya* sees a progression of light visions that glow with the colors associated with a particular chakra. The kundalini hierarchy is sometimes



Figure 1. Conventional representation of a hierarchy of subtle energy centers (chakras) aligned in parallel with the physical body.⁸

analogized to a cobra rearing its head: “When you strike a snake with a rod, it draws itself up, as still as a rod. This is how you must perceive [kundalini] when she is aroused by the Guru.”^{8(p.57)} While Tantric texts can differ about the colors of the chakras that appear during the earliest stages of kundalini rising, there is general agreement that the final common pathway is as follows: (1) a green chakra, (2) a dark blue or purple chakra, (3) a *brachmarandhra* (the ‘Egg’ or ‘Aperture’ of *Brahman*), and (4) Enlightenment, that is, merger with the bright flood of white light that is the primordial radiance of Brahman.

In Buddhist traditions, an opening of the Divine Eye is described as ‘*nirvana*’ (‘extinction’) rather than as a merger or union. This usage probably reflects the experience of the original Buddha, Gotama Sakyamuni. He was a Hindu yogi who had withdrawn from society and spent seven years in the forest practicing meditation and severe austerities. When these efforts were unavailing, he fell into despair, left the forest, collapsed beneath a tree and vowed to remain there in a meditative state until he either succeeded in finding release or died in the attempt. After meditating during the entire first night, Gotama saw the Divine Eye open just before dawn. His description of this event, as preserved in a Buddhist canon (*Samyuda Nikaya*), is: “Coming to be! Coming to be! At this thought, brethren, there arose in me, brethren, vision of things not taught unless the Divine Eye opens: there arose in me knowledge, insight, wisdom, light.”¹ The fact that Gotama later proclaimed a doctrine of no-atman (*anatman*) suggests that either he did not see a vision of something resembling the *atman* as he’d been led to expect, or, alternatively, that he did see such a vision but that he considered it to be just as apparitional, transient, and unreliable as the rest of the phenomenal world. As Buddhist traditions evolved, the cultivation of light visions as a spiritual path survived only in Tibetan Buddhism, where it received primary emphasis.

In this paper we compare the descriptions of light visions in the ancient Vedic scriptures and in yoga meditation texts in the Hindu, Tantric, and Tibetan-Buddhist religious traditions with detailed, systematic, and empirically-oriented descriptions of phosphene images observed by the author. We will also analyze the phosphene spatiotemporal characteristics in light of recent research on the neuroscience of vision, sleep, and epilepsy, in order to identify *ad hoc* the kinds of neurophysiological events that would have to take place contemporaneously in the visual pathways in order for these images to appear in the visual field.

CASE HISTORY

The author is a medical writer with no history of drug or alcohol abuse, no family or personal history of epileptic symptoms, and no sectarian affiliation. In graduate school he learned how to hypnotize himself using a combination of progressive muscle relaxation,¹⁶ silent instructions like “let yourself relax” or “concentrate on the breathing” adapted from autogenic training,¹⁷ and mental images of floating or drifting—a technique that resembles the generic “relaxation response” popularized by Benson.¹⁸ He used self-hypnosis for various purposes—to relax to sleep, to generate new ideas by free association, to dissipate muscle tension headaches, and to divert attention during minor surgical or dental procedures. This image-based approach did not generate phosphenes. After 15 years practicing this technique on sporadic occasions, the author attended an evening course in Buddhist meditation and learned how to meditate without mental imagery. The phosphene images began to appear during this class.

The details of the author’s phosphene induction technique, which he was using at the time of the paroxysmal events, is as follows: he lies on his back, closes his eyes, takes slow, deep, rhythmic breaths, keeps his eyes converged and slightly depressed, and keeps his attention fixated on the center of the visual field. The eye convergence is sustained with enough forcefulness to elicit a sensation of “fullness” or “pressure” in the eyeball, and the fixation of attention is forceful enough to evoke a sensation of ‘locking in,’ often accompanied by a characteristic *tinnitus cerebri*—a “buzzing” that is part sound, part vibration, and that feels as if it were radiating upward from a site located inside the skull and behind both ears. (This buzzing sound/vibration can also be generated by staring intently at the tips of the fingers of the fully extended arm, which suggests that it may signal activation of brain circuits involved in grasping for something just out of reach.) To keep his level of arousal low and his mental field free of distraction, he maintains a passive, indifferent attitude, allowing stimuli that are potentially distracting to drift in and out of consciousness without any attempt at suppression. During this behavioral state of deep calm and inwardly-focused attention, the author begins to see waves of brightly-colored phosphene that follow a predictable sequence. The same phosphene sequence appears spontaneously at sleep onset if the author fixes his attention on the center of the visual field.

At the time of the paroxysmal episode, the author was 45 years old and receiving psychotherapy for atypical dysthymic depression secondary to chronic posttraumatic stress disorder (PTSD). The presence of depression is important because depression is associated with cortical instability and bilateral activation of the mesotemporal cortices and the thalamus.¹⁹⁻²² Also, a diagnosis of chronic PTSD is associated with an increased risk of sclerosis in the right H, a condition which, if present, would predispose to hippocampal seizure.²³⁻²⁵ In this case a neurological exam performed several months after the seizure did not find evidence of organic lesions or epileptiform events, but, as we note in the discussion, the presence of pre-existing damage in the right H would explain why hypersynchronous activity emerged in that structure.

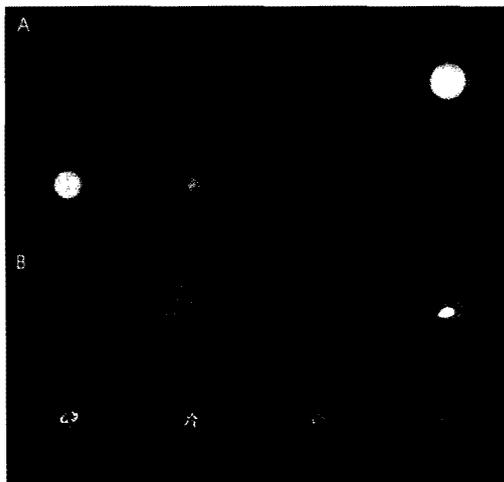
Circumstantial factors other than depression may have contributed to a lowering of the author's seizure threshold on the morning of the seizure: first, having just flown east across three time zones on the previous day, he was suffering from 'jet lag.' Second, as a result of these circadian disturbances he had slept only four of the 44 hours preceding the seizure, an amount of sleep loss that is associated with a lowered seizure threshold.²⁷⁻³⁴ This amount of sleep loss is also associated with a decrease in the latency of stage 2 slow wave sleep, a rebound effect that is present in this case.³⁵ Finally, the effects of sleep loss are enhanced by emotional stress, including depression.¹⁹⁻²²

On the morning of the seizure, feeling fatigued from loss of sleep but otherwise alert at 4 a.m., the author forced himself to go to bed and tried to use his familiar technique of phosphene induction to put himself to sleep. When he closed his eyes, he was surprised to see that, without his having to employ the usual calming and focusing behaviors, the familiar phosphene images began to appear almost immediately, as if they had been activated spontaneously. The display moved rapidly through the familiar sequence but then began to evolve into images he'd never seen before, as described below.

OBSERVATIONS

The full progression of phosphene images is illustrated in schematic drawings in Figures 2 through 4. (Detailed descriptions of the images are presented in the figure legends so they are readily available for comparison with the graphic

Figure 2. Sequence of phosphene images induced by meditation or activated spontaneously at sleep onset. A. One cycle of a 'receding annuli' sequence. Initially the author sees a dark, barely-perceptible wave—a sensation of movement—that flows inward from the 360° perimeter of vision, then sees a bright yellowish-green phosphene annulus illuminate in the visual field at about 80° of isoeccentricity. The annulus continues to shrink in diameter at a constant rate, preserving its symmetry throughout, and disappears into the center of vision after 4 seconds (as estimated by the author's count of "1001. . . 1002.").



The shrinking generates an illusion that the annulus is 'receding' in 3D space. A new annulus appears every 5 seconds (0.2 Hz) until the sequence terminates automatically after a total of 4 to 5 cycles. About halfway through the trajectory, the annulus fills in with a phosphene disk. During the early years of phosphene induction, the color of the fill-disk was a brighter, more opaque green than the annulus itself, but after several years of phosphene induction, the color of the fill-disk changed to dark blue. B. Examples of amorphous waves of expanding and contracting phosphene with a 'mist-like' texture. The first row shows an amorphous wave of yellowish-green phosphene—dark blue after the change noted above—which sometimes has a vaguely-defined crescent shape, as shown here. The amorphous wave illuminates upon reaching 80° of isoeccentricity, like the annuli. The waves enter from either the right or the left perimeter and sweep across the visual field with an expanding and enveloping motion. Meanwhile, behind the leading edge, the phosphene begins to dissipate, so that the rear of the wave is shrinking inward at the same time that the forward edge continues to expand into as yet untouched regions. Within a few seconds, all of the remaining phosphene shrinks into the center of vision, like the receding annuli. After a prolonged session of phosphene induction, the amorphous expanding clouds often last longer and develop a brighter, more finely-grained, opaque, and iridescent phosphene at the core. This bright central core keeps ebbing back from the fixation point and then filling back in, producing an image resembles a disembodied 'eye' with a bright 'iris' and a dark inner 'pupil'. On the morning of the seizure, the central, 'eye-like' phosphene gradually condensed into a tiny, 'star-like' cluster of thin, flashing filaments of white and blue phosphene.

illustration.) Note that these drawings are unable to reproduce the elusive, amorphous, ever-changing "smoke ring" quality of the phosphenes that makes it so difficult to render them in static, two-dimensional artwork; they do, however, provide an approximation of the actual phenomena that highlights the relevant spatiotemporal characteristics.

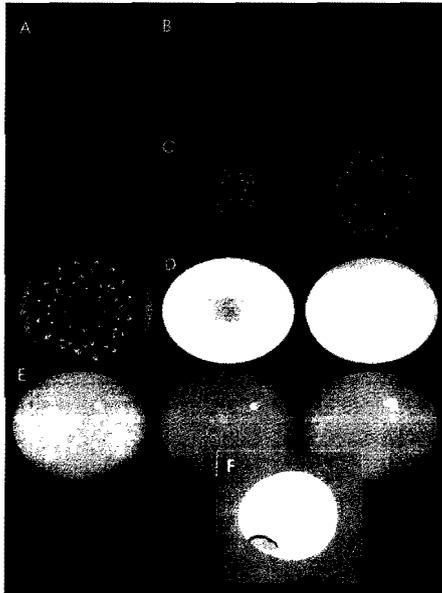
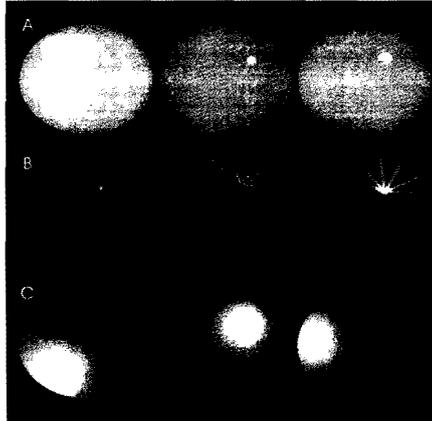


Figure 3. Transitional phosphene images. A. The 'star-like' image of thin, flashing filaments of white and blue phosphene. B. A stream of dark, barely-perceptible 'receding annuli' that entered the visual field at a rate of more than one per second (≥ 2 Hz), never illuminating as colored phosphene. The influx of dark waves generated an illusion of movement through a dark, 'tunnel-like,' 3D space. After a few seconds, the flood of dark annuli stopped abruptly, eclipsed by onset of the next visual image. C. A radiating spray of phosphene 'mist' interspersed with many beige-colored 'flecks' replaced the 'tunnel' of dark annuli. The spray seemed to radiate toward the viewer along a conical trajectory through 3D space and to 'strike' the forehead, so that the subject felt compelled to pull his head back against the pillow and

arch his back. There were also muscle tremors in the face and extremities. D. A gradual brightening and bluing of the visual field. This effect slowly but steadily obscured more and more of the radiating spray until it was the only visual sensation present. The approximate hue of blue, based on the author's comparison of color swatches from a CMYK Process Color Chart, is 40% Cyan without any Magenta, Yellow, or Black—a color similar to the blue of the sky on a clear, dry day in the summer or autumn. E. The appearance of a bulbous, hollow, translucent white phosphene that seemed to 'protrude' through the bright blue visual field, as if there were 3D 'depth' in the visual field. At first the bulbous image appeared as a faint glow, but when the author focused his attention on it, the glow became brighter, revealing a bulbous nose. As the phosphene glowed even more brightly, it revealed more caudal extension. This change created an illusion that the figure had just 'moved' forward in 3D space, and, conversely, as attention waned, the phosphene dimmed so that its caudal extension shortened, making it seem to 'recede'. Beneath the anterior pole of the bulbous image was a thin line or shadow, shaped like an inverted crescent. After many successive 'protrusions' and 'recessions,' the bulbous image suddenly glowed brightly—so much so that it looked as if the bulb had 'lurched' toward the vertical meridian—and, in the same moment, it disappeared and its former site was occupied by a cluster of three thin, white, phosphene rays (see Figure 4). The motion and speed of this change created an illusion that the bulb had somehow 'ruptured' and 'released' the phosphene rays.

Figure 2 describes the sequence of phosphene images usually observed during self-hypnosis (or during a normal, drowsy transition to NREMS), including threshold images of "receding annuli" and the subsequent images of "amorphous expanding waves." Figures 3 and 4 describe the new phosphenes that inaugu-

Figure 4. Paroxysmal phosphenes. A. The white phosphene bulbous image. B. When the bulbous image disappeared, it was replaced instantly by three thin white phosphene rays, and, at the same time, the bright blue background disappeared, leaving the rays silhouetted against the normal, charcoal-colored visual field (*eigengrau*). In the first presentation, the three white rays extended less than halfway to the perimeter of vision, and the tallest ray had a distinctive 90° bend to the left at the tip. One second later, the rays reappeared, now in a new, realigned version in which the rays had doubled in number (from 3 to 6), had lengthened so as to extend all the way to the peripheral rim of vision, and had fanned farther apart at the tops. In the next second, the author observed a third transformation: he saw the rays fan much farther apart, a movement that resembled the petals on a flower wilting in the heat. In this third display, the bases of the rays had tiny, shard-like triangles of bright, opaque white phosphene superimposed. The third display of the rays was the final one, and it persisted in the visual field for about 10 to 12 seconds. There were no auditory or sensorimotor symptoms accompanying any of the transformations of the rays. C. Serial flashes ('explosions') of dull white phosphene, appearing in either the right or left hemifield. Single flashes never occupied more than about a third of the visual field and seemed to alternate between the right and left side in a non-rhythmical pattern. The experience felt like being inside a dark storm cloud illuminated from within by flashes of sheet lightning. The photoparoxysm was accompanied by loud, crackling sounds, sensorimotor sensations of bilateral polymyoclonus that seemed to involve an 'ascending current of energy,' an orgasmic sensation diffused throughout the body, and psychic symptoms of euphoria and of awe mixed with fear.



rate a transition to paroxysmal experiences and that eventually culminate in a simple partial seizure.

The photoparoxysm was accompanied by non-visual symptoms: (1) a loud, crackling and buzzing sound reminiscent of an electric circuit 'shorting out'; (2) psychic symptoms that included euphoria and also a blend of awe and fear; (3) an orgasmic sensation that was not localized in the genitals but rather diffused throughout the body; and (4) motor symptoms of bilateral polymyoclonus which seemed to flow through the body in one direction, creating the illusion of a 'current of energy' flowing into the lower body (toes, lower legs, and perineal area), then flowing up the trunk, activating myoclonus in the arms and fingers, and to the facial muscles and the back of the neck.

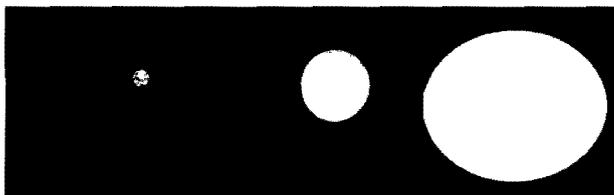


Figure 5. Postictal visual symptoms. For several weeks after the photo-paroxysm, when the subject went to bed and closed his eyes, a white glow

appeared in the same place where the bulb and rays appeared earlier. When attention was focused on this glow, it began to expand, as if billowing out toward the viewer in 3D space. The surface of the expanding phosphene presented a fractal-like pattern resembling the surface of a cauliflower, a billowing cumulus cloud, or a froth of soap bubbles. After a few days, the glow still appeared, but now it expanded with a surface that presented an undifferentiated, fog-like whiteness. Once the wave of white phosphene expanded, the entire visual field appeared white, as if the viewer were enveloped by fog or a snow "whiteout." If the author kept his attention focused, the field remained white for a relatively long amount of time, sometimes for more than several seconds. While this whiteness was present, the author experienced a weaker version of the seizure-related sensorimotor and psychic symptoms.

For many nights after the seizure, when the author closed his eyes at night, he saw a faint white glow at the spot in the upper right quadrant of the visual field where the bulbous phosphene and the rising rays had once appeared, as shown in Figure 5. If he fixed his attention on this faint glow, it became brighter and began to expand, as if it were radiating toward him in 3D space. The outer surface of the white expanding cloud had a 'cauliflowered' texture like the agitated surface of a billowing storm cloud or a foam of soap bubbles. This expansion brought a reactivation of a less intense version of the same psychic and sensorimotor symptoms that had accompanied the earlier seizure.

DISCUSSION

PART I: PHOSPHENES COMPARED WITH KUNDALINI LIGHT VISIONS

The similarities between the phosphenes and written descriptions of light visions in authoritative texts in the Hindu and Tibetan Buddhist traditions are illustrated in Tables I and II. (The source texts for excerpts are listed in the Table legends.) Despite the diversity of source documents, there is a remarkable match between the phosphene images described by the author and the descriptions from source texts, whether one reads across the horizontal rows, matching descriptions of a single image type, or one reads down the vertical

columns to see how a particular text describes the sequence of images. There are sizable gaps in some sequences which are most often attributable to our not being able to be sure that the reference was to a vision of light, or to our failure to find a clear, concise, unequivocal description that would not require an extended hermeneutic analysis to demonstrate that a light vision was involved.

We acknowledge that any comparison of excerpts drawn from works written in different historical epochs by different authors who lived in different cultures and practiced religious traditions could be challenged on the grounds that all cultural concepts and values are relative, so that it is not intellectually justifiable to compare material drawn from different cultures against some purportedly universal standard, as we do here, not to mention the complication interposed by the indeterminacy inherent in all language, which makes it problematical to say how accurately words communicate the underlying experience.^{36,37} These are important challenges to a work like ours, but, given constraints on space, we do not address those issues in this article.

PART II: PHOSPHENE IMAGES OF SLOW WAVE SLEEP RHYTHMS

Origins of the Receding Annuli

The threshold images of receding annuli have the same timing as a type of synchronous slow wave that normally occurs at the onset of the 'stage 2' transition to non-rapid-eye-movement sleep (NREMS). The transition from waking to NREMS is governed by the complex interaction of three brain rhythms in the reciprocal projections that link the cortex and thalamus in a corticothalamocortical (CTC) circuit.³⁸⁻⁴² The transition to NREMS begins with a drowsy waking state ('stage 1'). At this time, large populations of cortical cells begin to oscillate in synchrony with a very slow rhythm of less than one wave per second (< 1 Hertz [Hz]). This cortical slow rhythm, referred to the thalamus, lowers the polarization ('hyperpolarization') of thalamic cell membranes, including those of the thalamic reticular nucleus (RTN), a thin sheet of GABAergic cells that forms an outer cover over large portions of the lateral posterior thalamus. The hyperpolarization causes the RTN to stop firing the single spikes associated with the processing of sensory signals during waking,

Table 1
 Excerpts of Light Visions from Vedic and Hindu Yoga Meditation Texts*

	THE RG VEDA	SELECTED UPAMSHADS	THE YOGASUTRAS
ANNULI	When the visions that are concealed begin to glow. The	Fog, smoke, sun, . . . these are the preliminary forms (SvetasUp, 11:11)	In the light, everything is encompassed, the seer as well as the seen (4:23)
MISTS OR CLOUDS	Seers begin to glow by the stream of <i>rta</i> (8.6.8) The flame-arrows of Agni . . . assemble like streams of water into holes (10.25.4)	. . . a small lotus flower; within it, a small space (Chandogup, VIII: 1:1)	
STARS			
RADIAL SPRAY		. . . fireflies . . . are [also] preliminary forms (SvetasUp, If: 11)	One sees countless bright speckles. Keep watching: when the whirling [<i>vini-vrttib</i>] ends, the abode of <i>atman</i> appears (4:24 -25)
SPARKS			
BRIGHT BLUENESS	the divine radiance [Asurias] (IX 71)		
BULBOUS IMAGE	The Soma that the Brahmins know—that no one drinks. (X:85) [I]n a cloth like to a cloud (IX 69) Soma, storm cloud imbued with life . . . Navel of the <i>Rta</i> . . . (IX 74)	That which hangs down between the palates like a nipple, that is the birthplace of Indra (TaittrUp, I: 6: 1) The person [<i>purusa</i>] the size of a thumb . . . like a flame without smoke. (Kathaup, II: 1:12, 13)	That bending-down image [<i>viveka-nimnam</i>]—it bears the Aloneness [<i>Kaivalpa</i>] behind it (4: 26)

RAYS

The sharp seer, in heaven's navel,
is magnified in the woolen filter
(IX 12)

The Seers milk . . . the bull-
Soma (IX: 85)

He [Soma] sloughs off the divine
radiance, abandons his envelope,
and goes to rendezvous with the
Sky (IX 71)

The filter of the burning has
been spread . . . Its dazzling
mesh spread afar (IX: 83)

In jets, the pressed Soma is
clarified (IX 72)

**BRIGHT
WHITE
FLASHES**

The divine eye [*caksus*] is
altogether with the sun (Ix 10)

. . . procure for us the bright
substance which excels in worth
the outside; which procures
brilliant light . . . which shines
powerfully, O thou art born of
the *ṛta*. (2.13.15)

He is of the measure of a thumb,
of appearance like the sun, . . .
the *atman* seems to be of the size
of the point of a goad
(SvestasUp, V: 13)

The bird of golden hue . . . a
swan [*hamsa*], of surpassing
radiance (MaitrUp, VI: 34)

. . . like a wheel of fire, of the
color of the sun (MaitrUp, VI:
24)

. . . the ocean of light. In it,
worshippers become dissolved like
salt (MaitrUp, VI: 36)

. . . like lightnings from the light
within the clouds (MaitrUp VI:
36)

That tearing apart [*chidrescu*] you
see—it will release more changes
(4: 27)

. . . the vision of ultimate
discernment bursts forth like a
storm cloud of cosmic
dimensions [*dharmamegha-
samadhi*] (4: 29)

Table II Light Visions from Tantric Hindu and Tibetan-Buddhist Yoga Meditation Texts**		
	TANTRIC TEXTS	TIBETAN-BUDDHIST TEXTS
ANNULI	In the middle of the vault of the palate, like the tapering flame of a candle, the . . . fiery effulgences shine continuously (GA, p.10-11)	'Meditate on the four wheels, each like an umbrella or like the wheel of a chariot' (I: ii: 62)
MISTS OR CLOUDS		The forming of thoughts ceases, and phenomena, appearing like smoke, mirage. . . (I: ii: 98)
STARS	Above this energy [the ajna chakra midway between the eyes] dwells the dot, <i>bindu</i> (GA, p. 10- 11)	
RADIAL SPRAYS	When the <i>bindu</i> explodes and shatters, it expands immediately. . . (GA, p. 10-11)	Phenomena, appearing. . . like fireflies (I: ii: 98)
SPARKS	. . . a continuous whirling movement [<i>ghurni</i>] until there appear dazzling sparks just as the kundalini rises (AT, 5: 101, 107, 111, KS, II: 3)	The Flaring will appear as a yellow radiance (I: ii: 125)
BRIGHT BLUENESS		. . . something resembling the light of dawn, and something resembling a cloudless sky (YSD, I: ii: 98)
BULBOUS IMAGE	When the bindu explodes and shatters, it expands immediately and forms the <i>mastaka</i> [the 'Egg of Brahman'], similar to the triangular fruit of the water chestnut (GA, p. 10-11)	. . . a vision of the form of the Buddha outlined against a cloudless sky, like the moon's reflected form. . . Or one sees, as a form reflected in a mirror, the unobscured, radiant [Pure Illusory Body] (II: ii: 19-20)
	Concentrate on the image that resembles the stomach of a fish. . . [unfolding and contracting] (TA, 5: 57-61)	The Pure illusory Body. . . springs forth from the State of the Clear Light like a fish leaping forth from water, or like the form of the Buddha, which

	<p>...the supreme <i>linga</i> [phallus] of the skull. . . the womb in the middle of the forehead. . . . Having raised it to the surface of the <i>brahmadanda</i>, similar to an ivory tusk, the conch [<i>sankhini</i>] releases its flow. Inside the tusk there is but one orifice, the mouth of the conch, known as the tenth door (GA, p. 10)</p>	<p>rises [moves] as one does upon waking from sleep (YGS, IV: iii: 34)</p> <p>. . . the invisible psychic protuberance on the crown of the head. When the protuberance becomes filled with the vital force of the transmuted seminal fluid, one. . . realizes the State of the Great <i>Vajra-Dhara</i> [Wielder of the Thunderbolt] (I: ii: 144-145)</p>
<p>RAYS</p>	<p>The supreme energy blossoming into bliss. . . like a five hooded cobra as she rises (AT: 248-251)</p> <p>The swan [<i>hamsa</i>] of dazzling whiteness (AT: 136)</p>	<p>Simultaneously with this realization, the white fluid . . . flows upward to the crown of the head (I:ii: 144-145)</p>
<p>BRIGHT WHITE FLASHES</p>	<p>When the energy with five modalities draws herself up. . . and enters Brahman's seat, she flashes forth like lightning. . . such is the so-called serpent peircing. (AT: 248-251)</p>	<p>One gains . . . mastery of the Very Bright (IV: iii: 35)</p> <p>. . . the State of the Great <i>Vajra-Dhara</i> [Wielder of the Thunderbolt] (I: ii: 144-145)</p>

***Table I References.** (1) *The Rg Veda* oral verses transcribed between 1300 and 1000 B.C., into the world's first written scriptures; ^{1,4} (2) *The Svestasvatara, Chandogya & Maitri Upanishads* ('Commentaries'), written by anonymous authors between 800 and 500 B.C.;^{3,4} (3) *The Yogasutras* attributed to Patañjali and written between 0 and 200 A.D.

****Table II References:** (1) Abhinavagupta's *Tantraloka* (AT), ca. 1014 A.D.;⁸ (2) Goraksanatha's *Amaraughasana* (GA), ca. 1200 A.D.;⁸ (3) Ksemaraja's *Shivasutravimarshini* (KS), ca. 1000 AD.;⁸ (4) The Tibetan Buddhist version of Naropa's *The Epitome of the Six Abridged Doctrines* compiled by monks of the Tibetan Kargyu-tpa School about 1050 A.D. ^{1,3-14}

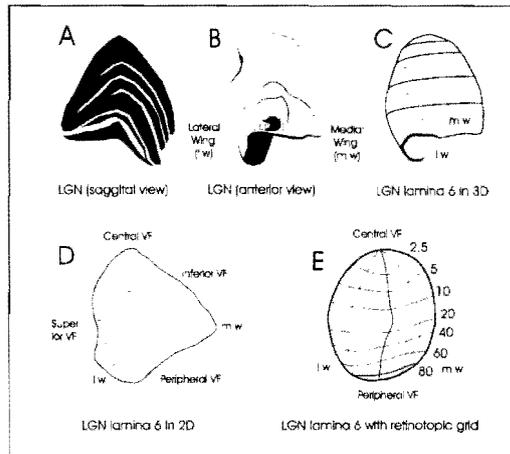
and, instead, the RTN fires short, 1 to 3 second bursts of 7-10 Hz spikes that wax and wane (like 'spindles') and recur at intervals of 3 to 10 seconds (0.3 - 0.1 Hz). By the time the RTN has fired three to seven bursts (4.78 ± 1.62), the thalamic cell membranes have been further hyperpolarized, which causes the RTN to stop firing spindles.⁴³ At this time, the next stage of NREMS ('stage 3') begins, but since a very different kind of sleep rhythm is involved there, we will delay discussing this until after we see how spindles can generate epiphenomenal phosphene images.

The phosphene images of receding annuli enter the visual field every 5 seconds (0.2 Hz). This timing falls within the range of RTN spindle burst emissions (0.1 to 0.3 Hz). Also, receding annuli terminate automatically after three to seven cycles, just like spindle bursts. There is, to our knowledge, no other brain rhythm that operates in the visual pathways that shares these same temporal characteristics. Therefore, based on timing alone, it is likely that the receding annuli are generated by RTN spindle bursts.

This hypothesis can be strengthened by showing that the symmetrical, annular shape of this phosphene image and its centripetal trajectory can be explained by reference to spindle waves moving through the RTN matrices where they interact with cells in the visual relay of the thalamus. Light receptors in the retina send projections that terminate in the lateral geniculate nucleus (LGN) of the thalamus. The retinal axons form one-to-one synapses with the thalamocortical (TC) neurons that relay visual signals to targets in the primary visual cortex. As shown in Figure 6, LGN is composed of six thin sheets (laminae) folded over a central hilus. These drawings, adapted from artwork by Le Gros Clark,⁴⁴ and from anatomical studies by Connolly and Van Essen⁴⁵ and Malpelli and Baker⁴⁶ show how the largest and most dorsal lamina—lamina 6—can be removed, flattened, converted from a 3D to a 2D structure, then adjusted to accept a grid of retinotopic grid coordinates. Since each LGN contains TC cells representing the contralateral half of the visual field, the perception of a single, unitary field results from a fusion of neural events taking place in both the right and left LGN.

The relay of afferent retinal signals by TC cells in the LGN is controlled by inhibitory neurons arrayed in a thin sheet—the RTN—that covers the outer surface of the posterior thalamus, including the LGN. When TC cell axons

Figure 6. Functional anatomy of the primate lateral geniculate nucleus (LGN). A. Sagittal silhouette of the monkey LGN showing six layers or sheets (“laminae”) folded over a central hylus. Afferent visual signals from the retina arrive via the optic track, enter the LGN through the rostradorsal surface, and form one-to-one synapses with thalamocortical (TC) vision-relay cells in specific laminae. The geniculate TC cell axons exit through the ventrocaudal surfaces of the laminae and are intercepted by



RTN neurons that impose inhibitory synapses on the TC cell axons. B. Drawing of the anterior surface of the rostradorsal lamina 6, adapted from an anatomical study by Le Gros Clark.⁴⁴ The human LGN has less of a hilar arch than the monkey LGN, but functionally the two structures are similar. C.-D. Schematic drawings showing how a lamina 6 from a monkey LGN was removed and converted from a 3D anatomical structure into a 2D topographic map by Connolly and Van Essen.⁴⁵ The triangular shape of the 2D drawing of lamina 6 adjusted to accommodate a grid that shows lines of equidistance (isoeccentricities) from the center of the retina, adapted from Connolly and Van Essen⁴⁵ who based the grid on data published by Malpelli and Baker.⁴⁶ This 2D map of lamina 6 shows that TC cells representing peripheral vision are distributed in the more ventral regions of the lamina, while TC cells representing central vision are located nearer the dorsal pole. Note that the cells in the dorsal half of lamina 6 that subservise central vision occupy a relatively large proportion of the total area of the lamina.

leave the LGN, they must pass through the RTN where they receive synapses that subject the axons to RTN regulation. During a waking state, the inhibition imposed by RTN cells is selective so that individual sensory signals are efficiently relayed to the cortices, but at sleep onset, when the RTN shifts to firing spindle bursts, this synchronous activity interferes with normal signal-processing. When a spindle burst is fired, a wave of excitation moves through the RTN matrices, imposing a wave of inhibition on all the TC cell axons it passes. Once the wave passes and the inhibition is removed, the TC cells usually fire rebound spikes.⁴⁷ In Figure 7 we use the 2D retinotopic map of lamina 6 to show how spindle wave moving simultaneously through the RTN matrices in both hemispheres would generate a pattern of TC cell rebound firing that can account for the phosphene image of a receding annulus. The

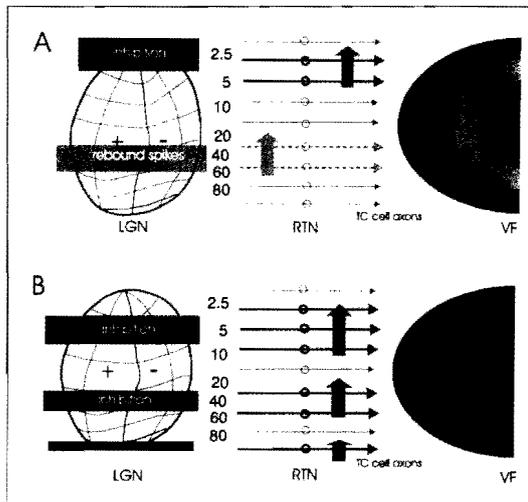


Figure 7. Origins of the receding annuli. A. The spatiotemporal characteristics of "receding annuli" can be explained in terms of spindle bursts moving ventrodorsally through the RTN network in a relatively thin, coherent spatial wave. As it passes, the spindle wave inhibits a narrow band of TC cells in the geniculate lamina 6, generating a dark annulus which blocks all other afferent visual signals, including the random metabolic discharge of the retinal receptors that generates the normal charcoal or eigengrau

background. In the wake of the spindle wave, when TC cells are released from inhibition, many fire rebound spikes. Since each LGN represents half of the visual field, a wave of TC cell rebound spikes moving along lamina 6 from the more ventral regions (representing peripheral vision) toward the dorsal pole (representing central vision) will register in the visual cortices as a hemi-annulus that shrinks in diameter. Spindle waves are fired simultaneously in both RTNs, so their passing will release simultaneous waves of TC cell rebound spikes in both LGNs; in the visual field, this pairing of TC cell rebound spikes will generate two complementary (reverse-image) phosphene hemi-annuli with tips fused to form a single annulus that shrinks in diameter. B. The same mechanism can explain the dark, fast-paced annuli. If the RTN were to fire spindle waves at a rate ≥ 2.0 Hz—ten times the normal rate of 0.2 Hz—then the rapidly repeating waves of inhibition will not allow TC cells in lamina 6 enough time to recover and fire rebound spikes. The author observed an influx of dark annuli arriving at intervals of more than one per second (≥ 2.0 Hz) which is explained in the text as cortical spike-wave (SW) complexes driving the RTN to fire spindle bursts at this accelerated rate.

annulus shrinks steadily in diameter as the wave of TC cell rebound spikes moves up from the ventral regions of the LGN (where TC cells represent the periphery of the visual field) toward the dorsal pole (where TC cells represent the center of the visual field).

The same mechanism can explain the dark, fast-paced annuli. If the RTN were driven by cortical influences to fire spindle bursts at a rate of ≥ 2 Hz—an acceleration that does in fact occur during the emergence of hypersynchronous, absence-like seizures, as we discuss below—then there is not enough time

between the successive waves of spindle burst inhibition for the TC cells to recover enough to fire rebound spikes. The result will be a stream of thin, dark, receding annuli that enter the visual field at this hypersynchronous rate of ≥ 2 Hz. This stream of dark annuli generates a sensation of 'optic flow,' that is, an illusion of motion through a dark, tunnel-like space even though the subject is not moving, as described by Steinmetz et al.: "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. . . ."^{48(p.189)}

This analysis suggests that the functional anatomy of the RTN matrix can, in some circumstances, constitute a 'resonating circuit' that, when activated, can generate an epiphenomenal visual image of a slow-moving phosphene annulus or of a 'tunnel' of dark, fast-moving annuli. This hypothesis of a resonating circuit is supported by the author's observation, described in an earlier article, that when he was given a magnetic resonance imaging (MRI) text, and thus exposed to a radio beam every 2 seconds (0.5 Hz, which is the interval that maximizes recapture of energy released by atomic precession), he saw a stream of phosphene receding annuli that appeared at the same rate (0.5 Hz) instead of the normal rate of 0.2 Hz, and, in this MRI-driven variant, the receding annuli did not stop automatically after a normal volley of four but continued for as long as the MRI test was underway.⁴⁹ Since the MRI beams energy into the body at two second intervals and then recaptures the energy given off by atoms as they return to their normal alignments, the vision of a continuous stream of receding annuli at 0.5 Hz can be explained as the flow of physiological energy released by atoms in the body through a resonating structure in the thalamus—the RTN matrix. The hypothesis of a resonating circuit has also been proposed by Max Knoll and colleagues based on studies in which they applied electrical stimuli in the EEG range to the heads of subjects, although they were unable to identify the neural mechanisms that constituted the resonator.^{50,51}

Two alternative hypotheses that might be put forward to explain the receding annuli can be easily dismissed. A mechanical discharge of retinal receptors generated by torsion-induced deformation of the converged eyeball might be

proposed as a competing hypothesis to explain the receding annuli, but if such a discharge were to occur, it is difficult to envision how it would selectively and simultaneously discharge the receptors situated along the full 360° perimeter of the retina—nor is it likely that the initial symmetry of such an annulus, were it to be initiated, would be preserved as the wave of depolarization of retinal receptors flowed inward from the periphery toward the central fovea. Also, there is no reason to suppose that RTN sleep spindles are referred to the retina, which would make it hard to explain the timing of the discharges. Another hypothesis might be that attention fixated on the center of the visual field would strongly facilitate certain visual neurons in the posterior parietal cortices, specifically, area PC, which is known to have large bilateral receptive fields and a center-surround, “macula-sparing” spatial structure.⁵² This hypothesis can also be rejected, first, because there is no evidence that visual signals are ever initiated in this region, and, second, because the centers of the receding annuli fill in with disk shapes midway through the trajectory, an observation that cannot be explained by activation of macula-sparing receptive fields in area PG. (For a discussion of the filling-in of a disk shape, see Nicholson.⁴⁹)

Origins of the Amorphous Expanding Clouds

During the transition from a waking state to NREMS, the synchronous spindle volleys of stage 2 induce a further drop in the polarization of thalamic cell membranes. Once a threshold value of hyperpolarization is reached after three to five spindle bursts, the RTN cells stop firing spindle bursts.⁴³ At the same time, TC cells begin firing low-threshold calcium spikes. The TC cell calcium spikes are not released randomly but rather as groups of spikes fired in response to the receipt in the thalamus of the synchronous pulsations of cortical cells oscillating with cortical slow (< 1 Hz) wave activity. The interaction of the synchronous cortical slow wave and the TC cell calcium spikes generates waves in the delta (1 - 4 Hz) frequency band which is characteristic of NREMS stages 3 and 4.³⁸⁻⁴²

One important clue about the mechanisms responsible for generating the amorphous phosphene waves is that the propagation patterns observed are similar to those manifested by the cortical slow (< 1 Hz) rhythm. The cortical slow wave is one example of a generic type of periodic spontaneous expanding

wave that is often observed in large networks of locally-connected neurons. Maeda and colleagues have studied these periodic spontaneous expanding waves using matured cultures of dissociated cortical neurons.⁵³ They found that periodic synchronized bursts originate every 10 to 20 seconds at random locations in the sheet of locally-connected cells and spread with unpredictable, asymmetrical patterns at a relatively slow speed, averaging 50 mm/second and propagating “sequentially from electrode to electrode, as each local group of neurons ‘charges up’ its neighboring, nonrefractory areas.”

The amorphous expanding phosphene clouds exhibit these same spatiotemporal characteristics: they begin at unpredictable locations in the visual field, usually in the periphery, and sweep across the visual field, activating new regions at the same time that passed over regions go dark again, with a duration that lasts several seconds—a relatively long time in terms of neural events.

PART II: PHOSPHENE IMAGES OF HYPERSYNCHRONY IN CTC CIRCUITS

Research on the Emergence of Hypersynchronous Seizures At Sleep Onset

Research in animal models shows that hypersynchronous seizures can emerge spontaneously in CTC circuits as a result of small, incremental changes in the operation of the same cellular mechanisms that govern a normal transition to NREMS.⁵⁴⁻⁶⁰ For this to happen, cortical cells must already be abnormally excitable at the time the transition to NREMS begins. The incremental emergence of a hypersynchronous seizure involves an alternation between two kinds of paroxysmal activity: (1) hypersynchronous spike-wave (SW) complexes at 1.5 to 2 Hz intervals fired by cortical cells, which then drive thalamic RTN cells to fire spindle bursts at the same rate, or (2) ‘fast runs’ of 10 to 15 Hz spike bursts fired by cortical cells, which then drive TC cells to discharge in an expanding wave pattern, as shown in Figure 8.⁵⁴⁻⁶⁰ This “expanding epileptic penumbra” was first observed in computer simulations by Lytton et al. then later confirmed in animal studies.^{56,57} The pattern results when cortical fast runs drive paroxysmal discharges in about 40% of TC cells distributed in a periphery around a locus of non-participating TC cells which are too inhibited by corticothalamic signals to discharge. “At the focus,” Lytton explains, “intense cortical and [RTN] neuron activity would

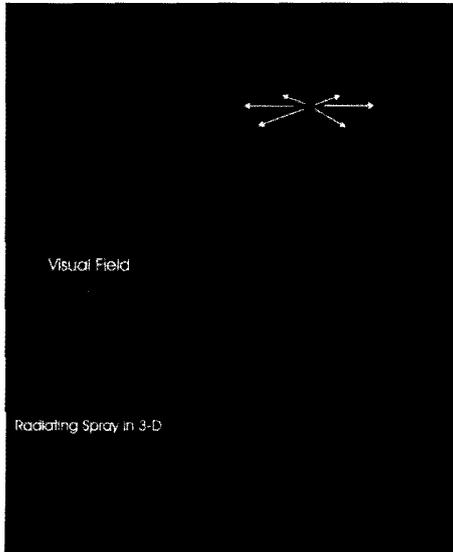


Figure 8. Origin of the radiating spray. A hypersynchronous seizure in CTC circuits can manifest as cortical fast runs. These fast runs form an expanding wave or “epileptic penumbra,” that drives the discharge of TC cells in the LGN.^{56,60} The expanding wave, when referred via corticothalamic projections to lamina 6 with its triangular or dome-like structure, will generate waves of TC cell discharges that reflect the same ‘outward’ direction of movement—in lamina 6, the movement will begin in the more dorsal regions and flow toward the more ventral regions, generating the image of a radiating spray, as shown in the inset. Note that the author observed the spray moving through

the middle and outer periphery of the visual field and does not recall anything happening at the center of vision.

produce relative TC neuron quiescence, . . . In the penumbra, [RTN] neuron divergence would produce less intense hyperpolarization in TC neurons, allowing [low-threshold spikes] and the appearance of the mutual oscillation . . . Such inhomogeneities might be transiently present on seizure initiation as one or more foci quickly coalesce and synchronize in a process of generalization. The rapidity of this process would make it hard to detect.”^{56(p.1694)} Because cortical cells do not fire in synchronous waves during fast runs, neither do the target TC cells in the LGN; therefore, the “expanding epileptic penumbra” will have a variegated texture.⁶⁰

Origins of the Tunnel Image and the Radiating Spray

Based on the new research about incremental emergence of hypersynchronous seizures at sleep onset, the phosphene image of a ‘tunnel’ formed by dark receding annuli can be explained as an epiphenomena of cortical SW complexes in CTC circuits: the sudden influx of dark, non-illuminating receding annuli

that enter the periphery of vision at a rate of ≥ 2 per second is consistent with cortical SW complexes driving RTN cells to fire spindle bursts at ≥ 2 Hz intervals, ten times faster than the normal rate of 0.2 Hz. Spindle bursts driven at this rate would impose successive waves of inhibition on TC cells in the LGN which does not allow time for them to recover and fire rebound spikes.

The shift from an influx of dark annuli to a radiating spray of phosphene flecks signals that cortical cells have shifted from firing SW complexes to firing the other kind of hypersynchronous discharges—cortical fast runs that form an expanding epileptic penumbra. The variegated ‘texture’ of the radiating spray is consistent with cortical fast run activity, because this drives large numbers of TC cells to discharge their spikes independently, generating a pointillist or mist-like spray that appears to radiate out toward the viewer for the reasons explained in Figure 8, above.⁶⁰ The tiny, bright, pellet-like ‘flecks’ amid the mist probably result from the simultaneous discharge of small clusters of contiguous TC cells.

The researchers who documented the cellular processes involved in the emergence of sleep-onset seizures were “surprised by the high incidence of seizures that occurred spontaneously,” an observation that suggested to them “the possibility that many spontaneous electrographic seizures in ‘normal’ subjects are unrecognized, and that those sleeping individuals pass in and out of seizures during their slow sleep oscillation, as we showed here for cats.”^{58(p.1476)} This suggests that meditators who are inducing sleep rhythm phosphenes might also make the transition in and out of hypersynchronous seizures with relative ease, as the author’s experience in the present article would indicate. In this regard, it is interesting to note that Persinger found SW complexes in an EEC recording of a subject who was practicing glossolalia at the time and who felt a sense of unity with the cosmos during this event.⁶¹

PART III: PHOSPHENE IMAGES ASSOCIATED WITH LIMBIC SEIZURE

Origins of the Uniform Brightening Effect

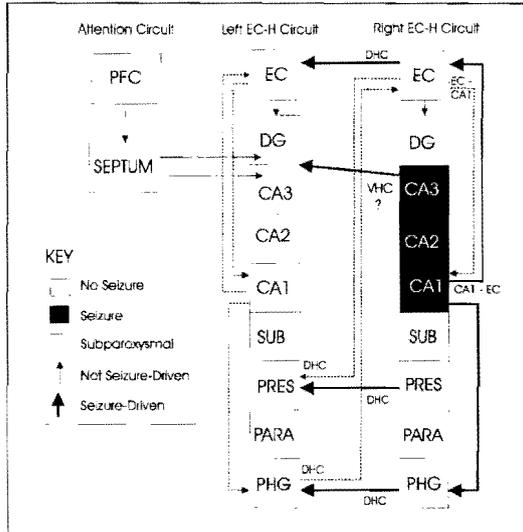
Two recent reviews surveying the basic mechanisms of epilepsy point out that, in patients with mesotemporal epilepsy, the initial event during the transition to partial seizure is usually a gradual increase in interictal-like rhythmical

discharges in the H.^{62,63} This is illustrated in a recent study by Bragin et al.,⁶⁴ where seizures were induced in rats by injecting kainic acid in one H: the researchers found that a rhythmic build-up began 10 seconds before the seizures started, first with irregular interictal spiking that increased in rate from 0.28 Hz to 0.82 Hz, then with a shift to rhythmical spike-bursts that occurred at intervals of >1 Hz. A similar pattern has been reported in earlier studies by Paré et al.⁶⁵ and Barbarosie and Avoli.⁶⁶ The build-up of rhythmical activity in field CA3 of one H usually does not trigger an outbreak of paroxysmal discharges; for this to occur, the rhythmical firing must usually be augmented by local recruiting waves.^{62,63} Once recruiting waves are active, paroxysmal discharges usually break out in the contralateral H, not the one engulfed by rhythmic activity.^{62,63}

A gradual build-up of rhythmical activity in the H can explain two phosphene images observed by the author—the gradually brightening of the visual field, and the appearance of a bulbous white image amid the bright blue. Our hypothesis about how the build-up of rhythmical activity in one H might generate a brightening effect in both halves of the visual field is shown in Figure 9. The basic idea is that output from the right H, where the build-up of rhythmical activity must occur (see below), is relayed through the dorsal hippocampal commissure to the left EC. These signals, which are intense but still subparoxysmal signals and which do not have specific spatial patterns, are relayed by the EC into the left H. Thus the output of both the right and left H will include these signals, even though there is no rhythmical activity taking place in the left H. There are a number of complicated mechanisms that contribute to this process that we discuss below.

It is generally accepted that the neural mechanisms that cause mesotemporal seizures are different from those that cause hypersynchronous seizures in CTC circuits (which include absence seizures and also generalized seizures in the penicillin epilepsy model).^{62,63} This distinction was reaffirmed in a recent study by Kandel et al.⁶⁷ who showed that stimulating the cortical cells of rats to the point of including SW seizures did not drive neuron activity in the H. Based on this information, we can infer that it is not likely that a build-up of rhythmical activity in the H would be driven by the presence of hypersynchronous activity in CTC circuits. It is, however, important to note that the study by Kandel et al. was performed while the rats were awake and physically

Figure 9. Origin of the uniform brightening and bluing. This schematic drawing, adapted from a series of drawings in an anatomical atlas by Duvenoy⁸¹ and from analyses of hippocampal function by Gloor,⁷⁵ represents the various subfields of the unfolded hippocampal formation aligned in the order that afferent sensory signals are processed. Using this schematic drawing, we can show how a build-up of rhythmical spiking in the right H (fields CA3 and CA1) is capable of generating a brightening in both halves of the visual field, not just the right half. This happens because the output of



the right H, which consists of excitatory signals that are stronger than normal but still subparoxysmal, will be relayed to retrohippocampal regions but also relayed contralaterally via the dorsal hippocampal commissure (DHC) to the left H, as shown here. Among the regions which receive DHC projections, and thus receive the excitatory signals from the right H, is the left EC; it then relays this excitation into the DG and fields CA3 and CA1. This means the increased output from the right H, which is generated by rhythmical spiking, will also affect the left H, and, as a result, the same signals will register in the visual cortices in both hemispheres. These signals generated by rhythmical activity will not have any spatially-specific patterns, so the increase of excitatory signals will register in the visual cortices as a uniform brightening of both halves of the visual field. Two other important items are also included in this drawing. An arrow labeled VHC that runs from field CA3 of the right H to the DG/CA3 border of the left H identifies a structure called the ventral hippocampal commissure (VHC), a direct, monosynaptic, interhippocampal projection that is present in monkeys but still disputed in humans. The VHC is in a position to add just enough excitatory input to trigger an outbreak of paroxysmal activity in a small number of granule cells at the hippocampal pole, the region receiving the most intense convergence of excitatory signals (see Figure 10). Also note the position of the attention-driven, septohippocampal circuit, shown here only in relation to the left EC-H loop. When attention is mobilized, as it commonly is during meditation, cells in the prefrontal cortices (PFC) stimulate the septum, and the septum, in turn, stimulates cells in the superficial layers of the extreme rostral pole of the H to release acetylcholine. The acetylcholine increases the excitability of pyramidal cells in CA3 and granule cells (see Figure 10).

active, so its findings do not exclude the possibility that the outcome might have been different if the rats were in a NREMS state at the time the stimulation was delivered.

During the behavioral states of resting immobility or NREMS, pyramidal cells in field CA3 of the H fire synchronous sharp waves (SPWs) at intermittent intervals that range between 0.02 to 3 Hz. These SPWs enhance the excitability of neurons in all but one subregion of the EC-H circuit.^{68,69} Granule cells in the DG are stimulated by SPWs arriving via “feedback” projections from CA3.⁷⁰ In response to SPWs, neuron excitability in the EC-H loop can increase by as much as 200% to 500%—or even 900%, if, as in this case, the target cells in the EC-H loop are also being excited by receipt of afferent sensory stimuli at the time the SPWs arrive.^{68,69} The generation of intrinsic synchronous SPWs during NREMS might make the H more vulnerable to destabilizing influences than during a waking state.

One potential source of destabilization is compromise of the barrier to propagation of excitatory SPWs. This barrier is normally interposed at the superficial layers of the EC. When SPWs reach the deep layers of the EC, the cells located there send signals through their ascending, intra-entorhinal projections that inhibit cells in the superficial layers of the EC.⁷⁰ If it were not for this barrier, SPWs would be relayed back into field CA3 where they originated, thereby establishing a reverberating loop of positive feedforward excitation with obvious epileptogenic potential. But the superficial layers of the EC are also the sole path through which afferent sensory signals are channeled into the H. If afferent sensory signals—or, in this case, visual signals generated by a seizure in CTC circuits—are bombarding the superficial layers of the EC with excitatory signals that have the rhythmical pattern of cortical fast runs, this might compromise the normal dampening effect that the EC exerts on SPWs. As noted by Steriade et al., the rhythms of fast runs are similar to those found in the mesotemporal regions during partial seizures: “The seizure epoch characterized by fast runs . . . resembles the stereotyped fast rhythm (~10-20 Hz) reported in human temporal lobe epilepsy that may spread to perihippocampal structures and cingulate cortex. . . .”^{58(p.1477)} Other studies have also reported similar rhythms.^{64-66,72} The barrage of afferent visual signals relayed into the H from the EC might also interfere with another process that dampens the excitatory effect of SPWs during a NREMS state. The SPWs alternate with ‘dentate

spikes,' which are large-amplitude, short-duration field potentials in DG granule cells that temporarily inhibit the pyramidal cells in CA3 and CA3/4 that fire the SPWs, so that the firing of dentate spikes produces a transient decrease in H output.^{64,71,72} The normal dampening effect of dentate spikes could be compromised by the receipt of afferent visual signals from CTC circuits.

Another wholly-independent mechanism that might generate rhythmical activity in the H during NREMS is activation of high-frequency 'ripples'.⁷³ Siapas and Wilson have shown that high-frequency 'ripples' in field CA1 of the H "co-occur" in close temporal synchrony with stage 2 NREMS spindles recorded in the cortex, which implies that this "co-occurrence of spindle-ripple episodes" is driven by some "common external factor."⁷⁴ Siapas and Wilson speculate that the common external driver of this co-occurrence might be the brainstem reticular formation (or some kind of interaction between the prefrontal cortices and the H). The importance of this finding of spindle-ripple co-occurrence for our purposes is that it raises the possibility that an accelerated barrage of high-frequency ripples might be generated in the H in synchrony with the accelerated firing of spindle bursts by the RTN in response to driving by cortical SW complexes. Thus a co-occurrence of accelerated spindles and accelerated ripples might have occurred in this case at the point when the author observed the dark, fast-paced annuli—and, as noted above, it would have then been superceded by cortical fast runs and the "stereotyped fast rhythm (~10-20 Hz) reported in human temporal lobe epilepsy."⁵⁸

The build-up of interictal spiking in the right H would also have the paradoxical effect of suppressing paroxysmal discharges in the H where it arises.⁷⁵⁻⁷⁷ This is consistent with the observation, mentioned earlier, that outbreaks of paroxysmal firing most often begin in the contralateral H.^{62,63,75-77} This explains why, after the author observed a gradual brightening of the visual field, this effect persisted for so long before the appearance of the next phosphene images associated with an outbreak of paroxysmal activity.

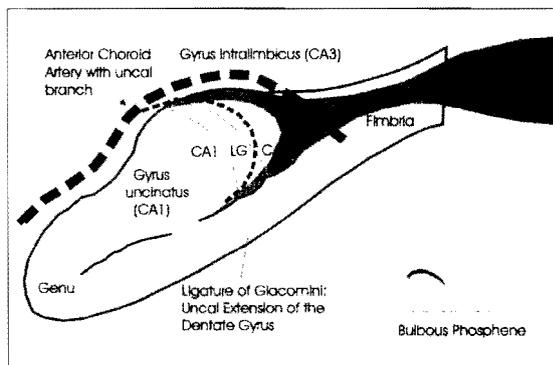
Origins of the Bulbous Phosphene

We can reasonably infer that the build-up of rhythmical firing took place in the right H, even though the destabilizing processes discussed above would be

occurring in both hemispheres. This inference is based on the observation that the bulbous image appeared in the right half of the visual field. Since the right half of the visual field is represented by neural assemblies in the left hemisphere, the processes that generated the bulbous image must have occurred there. To generate an object-like image, signals must contain spatially-specific information, but all spatially-specific signals will be obliterated in fields CA3 and CA1 of the right H which is consumed by rhythmical firing. If we conclude that the spatially-specific signals generating the bulbous image must come from the left H, then the rhythmical activity must occur in the right H.

This analysis of hippocampal processing of spatially-specific and spatially-diffuse signals is based on a theory of “entorhinal-hippocampal (EC-H) dialogue” proposed by Buzsáki and colleagues^{78,79}. It is important to understand the main ideas in their theory, because these will turn out to be crucial for understanding how a spatially-specific image like the bulbous phosphene can be generated inside the H. In this theory, hippocampal output is produced by the interaction of information processed by two very different kinds of circuits that together compose the EC-H loop. There is, on one hand, an extensive network of pyramidal neurons in field CA3, each of which is linked by many local projections; because of the local interaction, the CA3 circuit cannot preserve “spatially-specific” signals received from the EC. There is, however, a “spatially-specific” circuit which is constituted by the reciprocal projections that form a direct link between the EC and field CA1. The function of the spatially-specific EC-CA1 circuit is to implement a ‘matching’ process that reimposes the spatial information contained in the afferent sensory signals that originally entered the EC. When the EC sends its signals into both CA3 and CA1, the spatially-specific signals are lost in CA3 but preserved by keeping the spatially-specific signals oscillating back and forth in the reciprocal projections that link CA1 and the EC. The output of cells in field CA3, which has lost the spatially-specific information, is referred downstream to field CA1, where it is reintegrated with the spatially-specific patterns still active in the EC-CA1 circuit. Thus the original spatial patterns of afferent sensory signals are preserved intact. In the discussion that follows, we consider what would happen if there were no afferent visual signals arriving from the retina, and instead there was a differential activation of cell groups within the H that generated a ‘facsimile’ of a spatial pattern. Before addressing that issue, we need to find out more about the bulbous image.

Figure 10. Origins of the bulbous image. This schematic drawing of the human H, adapted from a series of drawings in an anatomical atlas by Duvenoy⁸¹ and from analyses of hippocampal function by Gloor,⁷⁵ shows how the medial bend of the H ('genu') points the anterior region (uncus) back toward the posterior H. At the rostral pole of the uncus



H is a snub-nosed cone. This polar region is called the gyrus intralimbicus (GI).^{78,81} The GI contains a subgroup of cells from field CA3 and from the hilar region (CA3/4). Its associated CA1 subfield, called the gyrus uncinatus, has been pulled out of normal alignment with CA3 by having been stretched around the genu. Focusing attention during meditation will have the effect of selectively enhancing neuron activity at the GI pole relative to other regions of the H. The attention-driven septohippocampal projections terminate primarily in the superficial layers of the GI pole—the alveus and the stratum oriens.⁷⁵ When the terminals are stimulated, acetylcholine is released. The acetylcholine inhibits the interneurons that inhibit CA3 pyramidal cells and granule cells, so the result in both cases is an increase in cell excitability. The region of maximum acetylcholine release—and thus of maximum cell excitability—has the same geometrical shape as the hollow, bulbous phosphene described by the author. (See the text for an discussion of how this selective activation of cells at the GI pole results in the generation of a visual image.)

The unusual shape of the bulbous image closely resembles the peculiar, 'thumb-like' shape of the extreme rostral pole of the anterior (uncus) H, called the gyrus intralimbicus (GI), as shown in Figure 10.^{75,81,82} To our knowledge, there is no other anatomical structure in the visual pathways that has this same shape. The GI contains a small subgroup of field CA3 and CA3/4 (hilar) neurons which has been physically separated from the rest of field CA3 by the medial bend of the H (genu). The field CA1 neurons associated with that CA3 subfield in the GI are located nearby in the gyrus uncinatus. Interposed between this CA3 subfield in the GI and the associated CA1 subfield is the uncus extension of the DG, called the Ligature of Giacomini (LG).^{75,81,82}

The GI pole can be activated by the exercise of attention, a potentially important point since meditation involves a strong inward focus of attention. This focus mobilizes neuron assemblies in the prefrontal cortex, which then

stimulate neurons in the medial septum that send excitatory signals to the GI pole. The septohippocampal terminals are densely packed in the most superficial layers of the GI—that is, in the alvear sheet and in the stratum oriens—and also in the anterior extension of the DC, the Ligature of Giacomini.^{75,81,82} As shown in Figure 10, this distribution of septohippocampal terminals forms a hollow, bulb-like cone.

Attention-driven septohippocampal stimulation of the GI triggers a release of acetylcholine in this bulb-like conical structure which produces two important effects: (1) the acetylcholine inhibits the interneurons that inhibit CA3 cells, which increases their excitability, and (2) it excites the muscarinic receptors of GABAergic interneurons in the Ligature of Giacomini, which enhances the excitability of the granule cells closest to the GI pole.^{80,83-87} In normal circumstances, the function of this attention-driven septohippocampal stimulation would be to facilitate the processing of afferent sensory signals, but, in this case, without any external signals to process, the effect of acetylcholine release will be selective disinhibition of the field CA3 neurons at the GI pole and the adjacent granule cells in the Ligature of Giacomini.⁸⁷ There will not be a comparable disinhibition in the rest of the field CA3 neurons located in the middle and posterior regions of the H, because there are few, if any, septohippocampal terminals located there. Nor will there be a disinhibition of granule cells in the rest of the DC. We propose that this selective increase in the excitability of CA3 and DG cells in the GI pole, when referred downstream to field CA1 and processed in the EC-CA1 circuit, will emerge in a form that, once it registers in visual awareness, will be decoded as the spatially-specific pattern of a hollow, ‘thumb-shaped’ bulb.

These signals generated inside the H do not have the same kind of “spatially-specific” coordinates as would be generated during normal, retinal-based perception, and this presents a problem: how does this information get registered in visual awareness? In retina-based perception, an external object triggers retinal light receptors that map position and direction using an egocentric topography, that is, using coordinates that map the object from the point-of-view of the perceiving subject. But when visual signals are processed inside the H, they are mapped in a different way: instead of recording events using egocentric coordinates, the H processes visual signals using an ‘allocentric’ map—one that records position and direction as coordinates and vectors on an abstract

geometric grid with no egocentric reference.⁸⁸⁻⁹⁴ Therefore, the signals that generate the bulbous phosphene will initially be recorded in allocentric coordinates—and no image will appear in the egocentric visual field until the signals have been referred back to the visual cortices and registered there. This can happen in two ways: (1) field CA1 sends signals directly to the parahippocampal cortices (PHC) and the perirhinal cortices (PRC), which then relay the signals to neocortical areas via an extensive network of back-projections, or (2) if field CA1 sends its output to the EC, which sends it on to the PHC and PRC for relay backward. This means there is a co-activation of cells in field CA1, in the EC, in the PHC and PRC regions, and in the neocortical areas.⁸⁸⁻⁹⁴ This coactivation is thought to be one of the mechanisms involved in the laying down of long-term memories. It is also thought to become active during recall of a memory, where the cells that contributed to the original experience (and to the laying down of the long-term memory) are reactivated.^{93,94}

Several new studies point to ways that allocentric spatial information generated inside the H could be relayed back to the visual cortices via the PHC or PRC. There is now new evidence that the visual cortices can acquire *new* information after visual signals have been processed by the H and returned via the PHC/PRC back-projections, information that was not part of any afferent visual signal, but rather is incorporated as an 'added value' to the original visual signals.⁹² Recent research also shows that, when subjects recall a visual memory, the neurons in the H, EC, PHC and visual cortices that are reactivated in concert may have the capacity to tune in selectively to particular categories of content in the visual scene being presented.⁹⁵ This is an unexpected finding—that vision-related cells in these regions of the mesotemporal cortex can be category-selective—and it has at least two important implications: "One is that the hippocampus carries more than just relational or spatial information (although the hippocampus had the largest proportion of cells selective for the category of 'spatial scenes'). A related implication is that the hippocampus has more than just a modulation or consolidation effect on cortex; instead, it carries complex visual information."^{96(p.856)}

These recent research findings challenge the conventional view that H processing of visual signals does not generate any 'value added' when the H signals are returned via back-projections to the visual cortices. This suggests that it might be possible—and, indeed, given the author's observations, that it

clearly can happen—that a pattern of signals which has spatially-specific allocentric coordinates acquired in the H can be preserved when sent via back-projections to the visual cortices, and that receipt of this new set of spatially-specific signals in the visual cortices would be registered in awareness ‘as if’ it were an external object perceived by the retinal receptors and mapped in the egocentric coordinates of normal perception.

Another anatomical factor to be considered is that output from CA1 fields in the uncus H (and thus from the GI pole) is segregated from the output of CA1 fields in the middle and posterior H when it is sent to the PHC and PRC for relay to the neocortex: signals from the GI pole are sent to area TH in the PHC, while signals from the middle H are sent to area TF and signals from the posterior H to area TEav.⁹⁷ This segregation of CA1 signals might explain why the intense firing of cells at the GI pole appears to be segregated as a locus of intense activation amid a much larger field with no spatially-specific signals present—thus producing the epiphenomenal image of a bulbous glow floating in a large expanse of bright blue.

The Origin of the Phosphene Rays

Since the phosphene rays appear at the same place in the upper right quadrant of the visual field as the bulbous image, we can infer that the rays were also generated by neural events taking place in the EC-H loop of the contralateral (left) hemisphere. The initial appearance of a cluster of three rays rising less than halfway to the perimeter of vision, and the subsequent realignment of the rays in which they double in number, extend to the perimeter of vision, and fan farther apart, can all be explained based on the anatomy of intrahippocampal circuits and on computer simulations of signal propagation in a model of intrahippocampal circuitry.⁹⁸⁻¹⁰²

Earlier we explained how the left EC-H loop received a stream of excitatory signals referred contralaterally from the right H, but it is highly unlikely that the image of three thin, discrete, phosphene rays was triggered by this kind of excitatory stimulation received in the left EC. Projections from the EC to the DC have been shown to diverge spatially so that the terminals are widely distributed along the longitudinal axis of the DG.⁷⁹ Also, Yeckel and Berger have

shown that when granule cells discharge in response to excitatory signals relayed by the EC, there is a “frequency facilitation,” that is, “a progressive increase in the number of active granule cells distributed over a wider spatial extent of the dentate gyrus.”¹⁰³ A similar spatial dispersion occurs when granule cells are discharged antidromically by feedback from CA3 cells.¹⁰⁴ Therefore, we can conclude that the stream of subparoxysmal, spatially-diffuse signals relayed to the left EC from the right H were not sufficient in this case to stimulate an outbreak of paroxysmal discharges limited to only three granule cells. Some additional factor would have had to be added into the mix—something that would limit the paroxysmal discharge to those three granule cells.

Stimulation studies in rats show that applying an electrode to the fibers of the ventral hippocampal commissure (VHC) never discharges more than three granule cells, no matter how strong a stimulus is applied.^{75,105-107} This would suggest that the human equivalent of the VHC might have added the extra stimulation that triggered the outbreak of paroxysmal discharges in three granule cells in this case. This scenario is also consistent with the general principle that, during the transition to a partial seizure in patients with mesotemporal epilepsy, the initial outbreak of paroxysmal activity occurs most often in the H contralateral to where the build-up of rhythmical activity occurs.^{62,63} There is, however, a major problem with the proposition that a VHC signal determined the pattern of paroxysmal outbreak in this case: while the presence of a functioning VHC has been demonstrated in monkeys, no one has been able to provide convincing evidence that there is such a structure in humans.

The Phosphene Rays Imply Existence of a Functioning VHC in Humans

In rats, the fibers of the VHC originate from pyramidal cells in the most proximal part of field CA3 (i.e., that part closest to the DG, designated by CA3c) and from the related CA3/4 cells in the hilar region; the fibers terminate in the inner third of the molecular layer of the contralateral DG, where their arrival has an excitatory effect.¹⁰⁵⁻¹⁰⁸ The reason why stimulation of the VHC in rats discharges a maximum of three granule cells is that VHC terminals innervate only a very narrow zone of cells that extends only 50 microns (μm) into the inner molecular layer of the DG and another 50 μm in the subgranular layer.¹⁰⁸

In monkeys, the VHC is proportionally much smaller than in rats, and it connects only the anterior (uncal) regions of the H, not the entire longitudinal extension; in other respects, the VHC connections in rats and monkeys are similar.^{72,82,108-111} The strongest evidence in support of a functioning VHC in humans comes from a depth electrode study of epileptic patients awaiting surgery by Spencer et. al. who found that 20% of spontaneous seizures that originated in a H in one hemisphere spread first to the contralateral H before activating any of the ipsilateral temporal cortices.¹¹² This pattern is consistent with propagation through a conduit analogous to the monkey VHC but not consistent with spread through the alternative structure, the dorsal hippocampal commissure (DHC). The DHC is a bundle of many fibers that originate in several different retrohippocampal regions and project to the contralateral homologues. Some DHC fibers also project to the contralateral EC (which is how the rhythmical activity in the right H gets relayed to the left EC-H loop). But for seizure discharges to propagate contralaterally from one H to the other via the DHC, the ipsilateral temporal cortices would first have to be activated. This activation did not appear in 20% of the seizures in the study by Spencer et al.

The conclusion that a subgroup of seizures spread contralaterally via a VHC projection has been challenged by several researchers. Studies of EEG coherence and phase relationships did not find evidence of linear relationships between seizure activity occurring in the right and left mesotemporal regions, as might be expected if there were a direct interhippocampal projection.¹¹³⁻¹¹⁹ Also, in depth electrode studies performed in epileptic patients awaiting surgery, Wilson et al. stimulated the anterior H (where VHC fibers would be) with a single shock but were not able to evoke discharges in the contralateral H.¹²⁰⁻¹²² In the synopsis of their findings, Wilson et al. did add an important qualification to their conclusion that there was no longer a functional VHC in humans: if there were a VHC projection in humans, they noted, and if this fiber were to follow the same course as in the monkey, it would be so long that action potentials moving along the fiber might become so attenuated by the time they reached the contralateral H that “. . . the resulting field potential would be of such a long duration and such low amplitude that it could not be detected. Indeed, such an input might be below the threshold for local synaptic activation of a response, unless other factors intervened such as temporal summation occurring with repetitive stimulation.”^{122(p.185)} Finally, in an anatomical study

of the human brain undertaken to resolve the controversy about the existence of the VHC, Gloor et al. found a few thinly-myelinated fibers which crossed the midline at the same place where the VHC fibers cross in monkeys, but being unable to trace the fibers to the sites of origin or termination, and, having noted the oblique trajectory of the fibers, Gloor et al. concluded that the fibers were more likely part of the H decussation than a remnant of the human VHC.¹²³ They did, however, acknowledge that they could only guess about the functions of these fibers: “It remains possible. . . that a small remnant of a ventral hippocampal commissure exists at this level in the human brain.”^{123(p.1253)}

The phosphene rays offer new evidence in support of the hypothesis that humans must have at least some functioning remnant of the VHC projection. As we discussed earlier, the unusual pattern in which only three granule cells participated in the initial outbreak of paroxysmal activity cannot be explained by the build-up of rhythmical activity in the right H, since excitatory stimulation relayed into the left H via the EC would have triggered a large number of granule cells—but the rhythmical activity in CA3 of the right H could have stimulated the cells that gave rise to a VHC projection to send a constant stream of excitatory signals. Moreover, we know that these VHC signals would arrive in the uncus region of the contralateral H and stimulate granule cells near the GI pole (in the Ligature of Giacomini)—a region that was also being stimulated so strongly by being the focus of attention that it was generating a bulbous phosphene glow in the visual field. Based on the rat stimulation studies, we can reasonably infer that stimulation of a VHC fiber would not trigger more than three granule cells; therefore, we conclude that VHC input must have been the crucial stimulus in this case. Note that this kind of temporal summation of excitatory signals converging on the apical dendrites of granule cells near the VHC terminal is precisely the type of contingency anticipated by Wilson et al. when they wondered if a VHC input might be “below the threshold for local synaptic activation of a response, unless other factors intervened such as temporal summation occurring with repetitive stimulation.”^{122(p.185)}

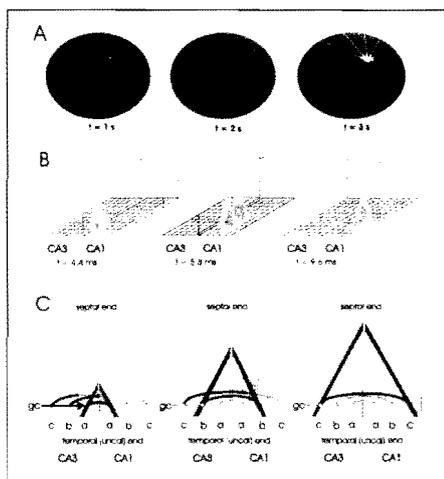
Transformations in the Phosphene Rays and Intrahippocampal Circuitry

Studies by Acsády et al. have shown that discharge of a single granule cell is capable of discharging a single pyramidal cell in field CA3, even though, as

we've mentioned earlier in this article, cells in field CA3 are linked by many local interconnections.¹²⁴ The one-to-one discharge can occur because granule cell mossy fibers innervate many more inhibitory interneurons in CA3 than pyramidal cells, in fact, there are often several mossy fibers that converge on a single interneuron. This circuitry creates a strong feedforward inhibition of pyramidal cells in CA3, an inhibition which can "efficiently suppress recurrent excitation in the CA3 collaterals but allow for the selective discharge of a few CA3 pyramidal cells."^{124(p.3399)} If there is a selective discharge of three granule cells by VHC input, as we propose, what will happen to the visual image of three rays as the action potentials set in motion by the granule cell discharges propagate via mossy fibers to field CA3, then propagate from CA3 to CA1 via Schaffer collateral projections, and then get relayed back to the visual cortices?

Figure 11 presents our hypothesis for how the initial display of phosphene rays could be generated by the registration of granule cell discharges in the allocentric spatial coordinates of field CA1 in the left H, and also shows how these initial factors also explain the further transformations in the rays—the doubling in number, the extensions in length, and the fanning further apart. The information shown here is adapted from a paper by Bernard et al. that describes the results of simulations performed using a computer model of intrahippocampal circuitry.¹⁰² This model incorporates the findings reported by several anatomical studies, and, in our drawings, we have added information about the distribution of longitudinal association strips in CA3 and CA1 adapted from Ishizuka et al. and Deadwyler and Hampson.⁹⁸⁻¹⁰⁰ The basic argument, which is presented in detail in the legend to Figure 11, is that the discharge of one granule cell discharges one pyramidal cell in each subsection of field CA3. The action potential set in motion by the discharge of that CA3 cell moves in two directions: first, it activates other CA3 cells that are interconnected in a longitudinal association strip, and, second, all of these activated CA3 cells send signals via their axons (called Schaffer collaterals) to target cells in CA1. Receipt of these signals in CA1 activates cells in the longitudinal association strip in CA1 which has a reverse 'mirror-image' diagonal to the strip in CA3. Because Schaffer collaterals differ in length, the signals from CA3 arrive at different times in different subfields of CA1, which means, over time, different longitudinal association strips get activated. As we explain in the Figure 11 legend, this differential activation can account for the changes in the length of the phosphene rays, for their doubling in number in the second display, and for their fanning apart.

Figure 11. Origins of the phosphene rays. A. Transformations of the phosphene rays at one second intervals. B. A schematic drawing of the topography of field CA1 represented as a 2D rectangle with a grid of identical 50 μm squares in computer simulations of Schaffer collateral function by Bernard et al.¹⁰² The researchers counted the number of spikes generated in each square of the CA1 grid after a simulated stimulation of DG granule cells. An important outcome of the computer simulations by Bernard et al. is the finding that stimulation of a single granule cell will evoke one spike-peak in CA1 at 4.4 milliseconds (ms), then, without additional input from the granule cell, will evoke two spike-peaks at 5.8 ms. This doubling effect parallels the author's observations of a doubling in the number of phosphene rays, even though a much longer time frame—a matter of successive seconds rather than milliseconds—was required to register the changes in visual awareness. At 9.6 ms, the same initial granule cell discharge generated a larger spike-peak with a wide base that started spreading apart in all directions. The neural events that are thought to cause these changes are described next. C. The sequential activation of intrahippocampal circuits by a granule cell discharge, as described by Bernard et al. An action potential fired by a granule cell



moves along a mossy fiber axon that traverses CA3 in a proximodistal direction, making synapses with one pyramidal cell in each of the three CA3 subfields (proximal CA3c, mid-region CA3b, and distal CA3a).^{98,111} The pyramidal cells in each subfield of CA3 activate other CA3 cells that are interconnected with it along a longitudinal association strip that runs in a diagonal pattern across CA3.^{99,101} The pyramidal cells in each of these three CA3 longitudinal association strips send Schaffer collateral projections to target cells in CA1, and those target cells are also arrayed in longitudinal association strips with the reverse-image diagonal, forming, in effect, a chevron-like pair.^{99,101} As a result of this chevron pattern, the Schaffer collateral axons running from CA3a to CA1a connection are much shorter than those forming the CA3c-CA1c connection.^{99,101} The conduction velocity of mossy fibers (0.67 meters/second) is faster than the conduction velocity of Schaffer collaterals (0.5 meters/second). This difference in speed, combined with the differences in the lengths of the Schaffer collaterals, means that an action potential moving along a mossy fiber activates all of its target cells in CA3 before any action potentials moving through Schaffer collaterals reach their target cells in CA1. Based on findings of Bernard et al., we can infer that the following sequence of events might have occurred while the author was observing the phosphene rays: (1) the last cell in CA3 to be discharged by granule cell input was the first to trigger a discharge in CA1, or, to be more specific, a pyramidal cell in CA3a evoked the first discharge in CA1a (via the shortest Schaffer collateral), and other cells in CA3a that were interconnected in a longitudinal association strip also evoked discharges in the paired longitudinal association strip in CA1a; (2) the second cell in CA3 to be discharged by the granule cell, located in the middle (CA3b) subfield, with activation of the paired longitudinal association strips in CA3b and CA1b in the same manner described for CA3a-CA1a; and (3) the first cell in CA3 to be discharged by the granule cell, located in proximal CA3c, is the last to trigger a discharge in CA1c because of the long length of that particular Schaffer collateral connection, and here too, as in the preceding discharges, the longitudinal association strips in CA3c and CA1c are activated in sequence. There are several points worth emphasizing. The longitudinal association fibers activated in CA1 become progressively longer as the zone of activation moves from CA1a to CA1b to CA1c. This progression might account for the change observed in the phosphene rays from a short length extending only halfway to the perimeter of vision to full extension to the perimeter. Also, the first spike-peak in CA1a occurs close to the CA1 border with CA3, which suggests that the spike-peak might be spatially-constrained from expanding in the direction—and this spatial confinement might account for the author's observation that one of the first phosphene rays had an odd leftward bend. In sum, based on two sources—the computer simulations of intrahippocampal circuits by Bernard et al., and a theory about the distribution of longitudinal associational strips by Deadwyler and Hampson,¹⁰¹ we can offer an explanation, on an ad hoc basis, for how discharge of three granule cells in the left DG could generate the following phosphene epiphenomena: (1) a columnar or ray-like image that reflects activation of cells in the CA1 longitudinal association strips; (2) changes in the length of the rays that reflect changes in the lengths of the longitudinal association strips; (3) the leftward bend in the tallest ray of the initial presentation, which reflects a spatial constraint that impedes the longitudinal association strip at the CA1-CA3 border; (4) the doubling in number of rays in the second display, which reflects the doubling in the number of spike peaks observed in the computer simulations that occurs for unknown reasons; (5) the fanning apart of the long rays, which reflects the moving apart of the two spike peaks observed in the computer simulations that also occurs for unknown reasons; and (6) the appearance of opaque triangles at the bases of the rays in the third display, which suggests an overlapping of the bases of the large spike-peaks or the multiple longitudinal association strips activated in CA1c.

The phosphene image of six rays fully fanned apart remained in the visual field for about ten to twelve seconds, which is consistent with the time that would be required for propagation of ictal discharges from the left H to the right H—a mean interval of 8.7 seconds with a range of 1 to 51 seconds.^{124,125}

PART IV: SYMPTOMS OF BILATERAL MESOTEMPORAL SEIZURE

Origin of the Lightning-Like Flashes

The dull-white, spatially-diffuse phosphene flashes covered less than half of the visual field and seemed to erupt in either the right or left side of the visual field at irregular intervals. The timing seemed to vary from near-simultaneity of bursts in opposite halves of the visual field, on one hand, to delays between successive bursts that lasted a full second or longer, on the other hand. These patterns can be explained as paroxysmal population discharges of granule cells in which the bursts occur independently in the right and left DG. The paroxysmal population discharges will be followed by after hyperpolarizations that can vary in duration but last a minimum of a half-second, the time required for granule cells to recover their capacity to fire.¹²⁶ Given these intervals, population discharges of granule cells that occur independently in the two hemispheres could generate a succession of flashes with the timing observed, that is, from near-simultaneity in both halves of the visual field to intervals in which no flash appear for almost a full second.

Auditory, Psychic, and Sensorimotor Symptoms

The photoparoxym was accompanied by a loud buzzing and crackling sound reminiscent of an electrical circuit ‘shorting out.’ Similar symptoms were reported in a study by Ketter et al. of procaine-induced stimulation of neuron hyperexcitability in the anterior limbic region of volunteer subjects, where 28% (9/32) of subjects reported seeing “unformed visual hallucinations (lights or colors reported as intense or very intense),” and 90% (29/32) described hallucinations of “unformed buzzing, ringing, or electronic sounds.”¹²⁷

Psychic symptoms reported by the author include: (1) a feeling of awe mixed with fear, which suggests that the seizure probably spread to the

amygdala;^{25,78,127} (2) an orgasmic sensation not focused on the genital region, which suggests that the seizure must have spread to the septum;¹²⁸⁻¹³⁰ and, (3) feelings of euphoria that occurred after the seizure ended. Euphoria is rarely reported as a symptom in the medical literature on epilepsy,¹³¹ but it was cited by 28% (9/28) of the volunteer subjects in the experiment of procaine-induced stimulation of the limbic area by Ketter et al.¹²⁷ Euphoria after a limbic seizure may result from release of endogenous opioids.¹³²

There was also a bilateral myoclonus manifesting as small movements in many different muscle groups, especially in the extremities and in the face. This pattern suggests a cortical reflex myoclonus, which is often associated with partial seizures.¹³³ Since the general excitability of the sensorimotor cortices is regulated by cingulate cortex, and since the cingulate cortex receives input from the amygdala via projections from the anterior nuclei of the thalamus, the spread of the bilateral mesotemporal seizure to the amygdala would activate a cortical reflex myoclonus.¹³⁴

The illusion that there was a 'current of energy' that was 'ascending' in the body suggests that the mesotemporal seizure may have reactivated hypersynchronous fast run activity in CTC circuits. Cortical fast runs would drive sensorimotor relay cells in the thalamus to fire in an expanding wave, just as they drove visual relay cells in the LGN to generate the visual image of a radiating spray. If the same kind of wave that moved dorsoventrally down the LGN to generate the image of a spray were to move similarly down the sensorimotor nuclei in the thalamus, this would generate the illusion of an *ascending* current. The reason for this is that the lateral ventral thalamic nucleus (VL) which projects to the precentral motor cortex and the posterior ventral nucleus (VP) which projects to the postcentral sensory motor cortex—both have a somatotopic organization in which the regions of the body are represented in a continuous sequence with the lower extremities represented in the dorsal region and the head area represented ventrally.¹³⁵⁻¹⁴⁰ This means that a continuous wave of cortically-driven discharges moving dorsoventrally through the VL and VP would first stimulate TC cells representing the lower body (toes, knees, hip, and trunk), then cells representing the middle regions (shoulder, elbow, wrist, hand, fingers, neck, and scalp), and only then cells representing the upper regions (brow, eyes, eyelids, face, lips, jaw, tongue and throat). By contrast, the sequence of body representation in the precentral and

postcentral sensorimotor cortices becomes discontinuous at the extreme lateral convexity where the representation of the face begins,¹³⁵ so the wave that creates the illusion of an ascending 'current' cannot originate in the sensorimotor cortices. This explanation of the illusory 'current' as a cortically-driven discharge of TC cells in the VL and VP nuclei is consistent with the finding that VL metabolism is elevated in both focal and in generalized epilepsy, and also with the finding that there is an elevation of metabolism in the specific thalamic nuclei which are functionally connected with cortices engulfed by hypersynchronous seizures, which would apply to both the VL and VP in the author's case, given that involuntary bilateral myoclonus was clearly present.^{141,142}

• • •

CORRESPONDENCE: Philip T. Nicholson • 52 Norfolk Road • Chestnut Hill, MA 02467-1832 • pnicholson@earthlink.net

REFERENCES & NOTES

1. J. Gonda, *The Vision of the Vedic Poets* (Mouton & Co., The Hague, Netherlands, 1963).
2. R. G. Wasson, *Soma: Divine Mushroom of Immortality* (Harcourt Brace Jovanovich, New York, NY, 1971).
3. S. Radhakrishnan, Ed., *The Principal Upanishads* (Humanities Press International, Atlantic Highlands, NJ, 1992).
4. D. R. Kinsley, *Hinduism: A Cultural Perspective*, 2nd Edition (Prentice Hall: Englewood Cliffs, NJ, 1993).
5. R. S. Mishra, *The Textbook of Yoga Psychology: The Definitive Translation and Interpretation of Patanjali's Yogasutras* (Crown Publishers, New York, NY, 1987).
6. G. Feuerstein, *The Yogasutras of Patanjali* (Inner Traditions International, Rochester, VT, 1989).
7. G. Krishna, *Kundalini: The Evolutionary Energy in Man* (Shambhala, Boulder, CO, 1967).
8. L. Silburn, *Kundalini: Energy of the Depths* (State University of New York Press, Albany, NY, 1988).
9. S. Muktananda, *Play of Consciousness* (SYDA Foundation, South Fallsburg, NY, 1978).
10. E. Conze, *Buddhism: Its Essence and Development* (Harper & Row, New York, NY, 1959).
11. D. Cozort, *Highest Yoga Tantra: An Introduction to the Esoteric Buddhism of Tibet* (Snow Lion Publications, Ithaca, NY, 1986).
12. R. A. F. Thurman, *Essential Tibetan Buddhism* (Harper Collins, New York, NY, 1995).
13. G. H. Mullin, Transl. & Ed., *Tsongkhapa's Six Yogas of Naropa* (Snow Lion Publications, Ithaca, NY, 1996).
14. W. Y. Evans-Wentz, Transl. & Ed., *Tibetan Yoga and Secret Doctrines* (Oxford University Press, London, 1958).

15. D. P. Brown, The Stages of Meditation in Cross-Cultural Perspective, In *Transformations of Consciousness* (K. Wilber, I. Engler & D. Brown, Eds., Shambhala Press, Boston, MA, 1986).
16. E. Jacobson, *Progressive Relaxation* (University of Chicago Press, Chicago, IL, 1938).
17. J. H. Schultz & W. Luthe, *Autogenic Training: A Physiological Approach to Psychotherapy* (Grune & Stratton, New York, NY, 1969).
18. H. Benson, *The Relaxation Response* (Morrow, New York, NY, 1975).
19. M. L. Frus & M. Lund, Stress Convulsions, *Archives of Neurology* **31** (1974), pp. 155-159.
20. M. L. Friis, Stress Convulsions, In *Comprehensive Epileptology* (M. Dam & L. Gram, Eds., Ravens Press, New York, NY, 1990), pp. 247-250.
21. M. S. George, T. A. Ketter, B. A. Parekh, B. Horwitz, P. Herscovitch & R. Post, Brain Activity During Transient Sadness and Happiness in Healthy Women, *American Journal of Psychiatry* **152** (1995), pp. 341-351.
22. P. Loiseau, Seizure Precipitants, In *Epilepsy: A Comprehensive Textbook*, Vol. 1 (J. Engel, Jr. & T.A. Pedley, Eds., Lippincott-Raven Publishers: Philadelphia, PA, 1997), pp. 93-97.
23. M. S. Myslobodsky, Pro- and Anti-convulsant Effects of Stress: The Role of Neuroactive Steroids, *Neuroscience and Biobehavioral Reviews* **17** (1993), pp. 129-139.
24. J. D. Bremner, P. Randall, T. M. Scott, R. A. Bronen, J. P. Seibyl, S. M. Southwick, R. C. Delaney, G. McCarthy, D. S. Charney & R. B. Innis, MRI-Based Measurement of Hippo Campal Volume in Patients with Combat Related Posttraumatic Stress Disorder, *American Journal of Psychiatry* **152** (1995), pp. 973-981.
25. S. L. Rauch, B. A. van der Kolk, R. E. Fisler, N. M. Alpert, S. P. Orr, C. R. Savage, A. J. Fischman, M. A. Jenike & R. K. Pitman, A Symptom Provocation Study of Posttraumatic Stress Disorder Using Positron Emission Tomography and Script-Driven Imagery, *Archives of General Psychiatry* **53** (1996), pp. 387-390.
26. A. M. Magariños, J. M. G. Verdugo & B. S McEwen, Chronic Stress Alters Synaptic Terminal Structure in Hippocampus, *Proceedings of the National Academy of Sciences USA (Neurobiology)* **94** (1997), pp. 14002-14008.
27. C. H. Gunderson, P. B. Dunne & T. L. Feyer, Sleep Deprivation Seizures, *Neurology* **23** (1973), pp. 679-686.
28. P. Rajna & J. Veres, Correlations Between Night Sleep Duration and Seizure Frequency in Temporal Lobe Epilepsy, *Epilepsia* **34,3** (1993), pp. 574-579.
29. I. Lorenzo, J. Ramos, C. Arce, M. A. Guevara & M. Corsi-Cabrera, Effect of Total Sleep Deprivation on Reaction Time and Waking EEG Activity in Man, *Sleep* **18,5** (1995), pp. 346-354.
30. L. Bettendorf, M. Sallanon-Moulin, M. Touret, P. Wins, I. Margineanu & F. Schoffeniels, Paradoxical Sleep Deprivation Increases Content of Glutamate and Glutamine in Rat Cerebral Cortex, *Sleep* **19,1** (1996), pp. 65-71.
31. D. P. Brunner, D. J. Dijk & A. A. Borbély, Repeated Partial Sleep Deprivation Progressively Changes the EEG During Sleep and Wakefulness, *Sleep* **16,2** (1993), pp. 100-113.
32. D. C. Hesdorffer & C. M. Verity, Risk Factors, In *Epilepsy: A Comprehensive Textbook*, Vol. 1 (J. Engel, Jr. & T. A. Pedley, Eds., Lippincott-Raven Publishers, Philadelphia, 1997), pp. 59-67.
33. R. Leproult, G. Copinschi, O. Buxton & E. Van Cauter, Sleep Loss Results in an Elevation of Cortisol Levels the Next Evening, *Sleep* **20,10** (1997), pp. 865-870.

34. P. Wolf, Isolated Seizures, In J. Engel, Jr. & T. A. Pedley, Eds., *Epilepsy: A Comprehensive Textbook*, Vol. 1 (Lippincott-Raven Publishers, Philadelphia, PA, 1997), pp. 2475-2481.
35. A. Daurat, A. Aguirre, J. Forer & O. Benoit, Disruption of Sleep Recovery After 36 Hours of Exposure to Moderately Bright Light, *Sleep* **20**,5 (1997), pp. 352-358.
36. S. T. Katz, Ed., *Mysticism and Language* (Oxford University Press, Oxford, 1992).
37. R. K. C. Forman, Ed., *The Problem of Pure Consciousness* (Oxford University Press, Oxford, 1990).
38. M. Steriade & R. W. McCarley, *Brainstem Control of Wakefulness and Sleep* (Plenum Press, New York, NY, 1990).
39. D. A. McCormick & H. C. Pape, Properties of a Hyperpolarization-Activated Cation Current and Its Role in Rhythmic Oscillation in Thalamocortical Relay Neurons, *Journal of Physiology* (London) **431** (1990), pp. 291-318.
40. M. Steriade, D. Contreras, R. Curro Dossi & A. Nunez, The Slow (< 1 Hz) Oscillation in Reticular Thalamic and Thalamocortical Neurons: Scenario of Sleep Rhythm Generation in Interacting Thalamic and Neocortical Networks, *Journal of Neuroscience* **13**,8 (1993), pp. 3284-3299.
41. M. von Krosigk, T. Bal & D. A. McCormick, Cellular Mechanisms of a Synchronized Oscillation in the Thalamus, *Science* **291** (1993), pp. 361-364.
42. M. Steriade, D. A. McCormick & T. Sejnowski, Thalamocortical Oscillations in the Sleeping and Aroused Brain, *Science* **262** (1993), pp. 679-685.
43. 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.
44. W. E. Le Gros Clark, The Laminar Organization and Cell Content of the Lateral Geniculate Body in the Monkey, *Journal of Anatomy* **75** (1940-1941), pp. 419-433.
45. M. Connolly & D. Van Essen, Representation of the Visual Field in Parvicellular and Magnocellular Layers of the Lateral Geniculate Nucleus in the Macaque Monkey, *Journal of Comparative Neurology* **226** (1984), pp. 544-564.
46. J. G. Malpelli & R. H. Baker, The Representation of the Visual Field in the Lateral Geniculate Nucleus of the Macaca Mulatta, *Journal of Comparative Neurology* **161** (1975), pp. 569-594.
47. D. A. Coulter, Thalamocortical Anatomy and Physiology, In *Epilepsy: A Comprehensive Textbook*, Vol. I (J. Engel, Jr. & T. A. Pedley, Eds., Lippincott-Raven, Philadelphia, PA, 1997), pp. 341-351.
48. 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.
49. P. T. Nicholson, Phosphene Images of Thalamic Sleep Rhythms Induced by Self-Hypnosis, *Subtle Energies and Energy Medicine* **7**,2 (1996), pp. 111-148.
50. M. Knoll, Subjective Light Patterns Spectroscopy in the Encephalographic Frequency Range, *Nature* **184** (1959), pp. 110-126.
51. 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 Neurologica* **23** (1963), pp. 201-236.
52. B. C. Motter, M. A. Steinmetz, C. J. Duffy & V. B. Mountcastle, Functional Properties of Parietal Visual Neurons: Mechanisms of Directionality Along a Single Axis, *Journal of Neuroscience* **7**,1 (1987), pp. 154-176.

53. 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.
54. M. Steriade & D. Contreras, Relations Between Cortical and Thalamic Cellular Events During Transition from Sleep Patterns to Paroxysmal Activity, *Journal of Neuroscience* **15**,1 (1995), pp. 623-642.
55. 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.
56. W. W. Lytton, D. Contreras, A. Destexhe A & M. Steriade M., Dynamic Interactions Determine Partial Thalamic Quiescence in a Computer Network Model of Spike-and-Wave Seizures, *Journal of Neurophysiology* **77** (1997), pp. 1679-1696.
57. M. Steriade & D. Contreras, Spike-Wave Complexes and Fast Components of Cortically Generated Seizures: I. Role of the Neocortex, *Journal of Neurophysiology* **80**,3 (1998), pp. 1439-1455.
58. M. Steriade, F. Arnzica, D. Neckelmann & I. Timofeev, Spike-Wave Complexes and Fast Components of Cortically Generated Seizures. II. Extracellular Patterns, *Journal of Neurophysiology* **80**,3 (1998), pp.1456-1479.
59. D. Neckelmann, F. Arnzica & M. Steriade, Spike-Wave Complexes and Fast Components of Cortically Generated Seizures: III. Synchronizing Mechanisms, *Journal of Neurophysiology* **80**,3 (1998), pp. 1480-1494.
60. I. Timofeev, F. Grenier & M. Steriade, Spike-Wave Complexes and Fast Components of Cortically Generated Seizures: IV. Paroxysmal Fast Runs in Cortical and Thalamic Neurons, *Journal of Neurophysiology* **80**,3 (1998), pp. 1495-1513.
61. M. A. Persinger, Striking EEG Profiles from Single Episodes of Glossolalia and Transcendental Meditation, *Perceptual and Motor Skills* **58**,1 (1984), pp. 127-133.
62. J. Engel, Jr., M. A. Dichter & P. A. Schwartzkroin, Basic Mechanisms of Human Epilepsy, In *Epilepsy: A Comprehensive Textbook*, Vol. I (J. Engel Jr. & T. A. Pedley, Eds., Lippincott-Raven: Philadelphia, PA, 1997), pp. 499-512.
63. P. D. Williamson & J. Engel, Jr., Complex Partial Seizures, In J. Engel Jr. & T. A. Pedley, (Eds.), *Epilepsy: A Comprehensive Textbook*, Vol. I (Lippincott-Raven, Philadelphia, PA, 1997), pp. 557-566.
64. A. Bragin, G. Jandó, Z. Nádasdy, M. van Landeghen & G. Buzsáki, Dentate EEG Spikes and Associated Interneuronal Population Bursts in the Hippocampal Hilar Region of the Rat, *Journal of Neurophysiology* **73**,4 (1995), pp. 1691-1705.
65. D. Paré, M. deCurtis & R. Llinás, Role of the Hippocampal-Entorhinal Loop in Temporal Lobe Epilepsy: Extra- and Intra-cellular Study in the Isolated Guinea Pig Brain In Vitro, *Journal of Neurology* **12**,5 (1992), pp. 1867-1881.
66. M. Barbarosie & M. Avoli M., CA3-Driven Hippocampal-Entorhinal Loop Controls Rather Than Sustains In Vitro Limbic Seizures, *Journal of Neuroscience* **17**,23 (1997), pp. 9308-9314.
67. A. Kandel, A. Bragin, D. Carpi & G. Buzsáki, Lack of Hippocampal Involvement in a Rat Model of Petit Mal Epilepsy, *Epilepsy Research* **23** (1996), pp. 123-127.
68. G. Buzsáki G., Generation of Hippocampal EEG Patterns, In Issacson and Pribham, *The Hippocampus*, Vol. 3 (Plenum Press, New York, NY, 1986), pp. 137-167.
69. J. J. Chrobak & G. Buzsáki, Selective Activation of Deep Layer (V - VI) Retrohippocampal Cortical Neurons During Hippocampal Sharp Waves in the Behaving Rat, *Journal of Neuroscience* **14**,10 (1994), pp. 6160-6170.

70. M. Penttonen, A. Kamondi, A. Sik, L. Acsády L & G. Buzsáki G., Feed-Forward and Feed-Back Activation of the Dentate Gyrus In Vivo During Dentate Spikes and Sharp Wave Bursts, *Hippocampus* **7** (1997), pp. 437-450.
71. A. Kamondi, L. Acsády & G. Buzsáki G., Dendritic Spikes are Enhanced by Cooperative Network Activity in the Intact Hippocampus, *Journal of Neuroscience* **18,10** (1998), pp. 3919-3928.
72. J. L. Stringer, J. M. Williamson & E.W. Lothman, Induction of Paroxysmal Discharges in the Dentate Gyrus: Frequency Dependence and Relationship to Afterdischarge Production, *Journal of Neurophysiology* **62,1** (1989), pp. 126-135.
73. A. Bragin, J. Engel, Jr., C. L. Wilson, I. Fried & G. Buzsáki G., High-Frequency Oscillations in Human Brain, *Hippocampus* **9** (1999), pp. 137-142.
74. A. G. Siapas & M. A. Wilson, Coordinated Interactions Between Hippocampal Ripples and Cortical Spindles During Slow-Wave Sleep, *Neuron* **21** (1998), pp. 1123-1128.
75. P. Gloor, *The Temporal Lobe and Limbic System* (Oxford University Press: London, 1997), p. 865
76. B. W. Colder, R. C. Frysinger, C. L. Wilson, R. M. Harper & J. Engel, Jr., Decreased Neuronal Burst Discharge Near Site of Seizure Onset in Epileptic Human Temporal Lobes, *Epilepsia* **37,2** (1996a), pp. 113-121.
77. B. W. Colder, C. L. Wilson, R. C. Frysinger, L. C. Chao, R. M. Harper & J. Engel, Jr., Neuronal Synchrony in Relation to Burst Discharge in Epileptic Human Temporal Lobes, *Journal of Neurophysiology* **75,6** (1996b), pp. 2496-2508.
78. G. Buzsáki, M. Penttonen, A. Bragin, Z. Nádasdy & J. J. Chrobak, Possible Physiological Role of the Perforant Path-CA1 Projection, *Hippocampus* **5** (1995), pp. 141-146.
79. G. Buzsáki, The Hippocampal-Neocortical Dialogue, *Cerebral Cortex* **6** (1996), pp. 81-92.
80. C. Buhusi & N. A. Schmajuk, Attention, Configuration, and Hippocampal Function, *Hippocampus* **6** (1996), pp. 621-642.
81. H. M. Duvernoy, *The Human Hippocampus: An Atlas of Applied Anatomy* (J. F. Bergmann Verlag, Munich, 1988), p. 159.
82. D. L. Rosene & G. W. Van Hoesen, The Hippocampal Formation of the Primate Brain, In *Cerebral Cortex, Vol.6: Further Aspects of Cortical Function, Including Hippocampus* (E. G. Jones & A. Peters A, Eds., Plenum Press, New York, NY, 1987), pp. 345-457.
83. D. G. Amaral & J. Kutz, An Analysis of the Origins of the Cholinergic and Noncholinergic Septal Projections to the Hippocampal Formation of the Rat, *Journal of Comparative Neurology* **240** (1985), pp. 37-59.
84. A. I. Gulyás, L. Seress, K. Tóth, L. Acsády, M. Anral & T. F. Freund, Septal GABAergic Neurons Innervate Inhibitory Interneurons in the Hippocampus of the Macaque Monkey, *Neuroscience* **41,2/3** (1991), pp. 381-390.
85. L. J. Reece & P. A. Schwartzkroin, Effects of Cholinergic Agonists on Two Non-Pyramidal Cell Types in Rat Hippocampal Slices, *Brain Research* **566** (1991), pp. 115-126.
86. P. A. Schwartzkroin & D. C. McIntyre, Limbic Anatomy and Physiology, In *Epilepsy: A Comprehensive Textbook*, Vol. 1 (J. Engel Jr. & T. A. Pedley, Eds., Lippincott-Raven, Philadelphia, PA, 1997), pp. 323-337.
87. N. Hájos, E. C. S. Papp, L. Acsády, A. I. Levey & T. F. Freund, Distinct Interneuron Types Express M2 Muscarinic Receptor Immunoreactivity on Their Dendrites or Axon Terminals in the Hippocampus, *Neuroscience* **82,2** (1998), pp. 355-376.

88. W. A. Suzuki & D. G. Amaral, Topographic Organization of the Reciprocal Connections Between the Monkey Entorhinal Cortex and the Perirhinal and Parahippocampal Cortices, *Journal of Neuroscience* **14,3** (1994), pp. 1856-1877.
89. G. J. Blatt & D. L. Rosene, Organization of Direct Hippocampal Efferent Projections to the Cerebral Cortex of the Rhesus Monkey: Projections from CA1, Subiculum, and Subiculum to the Temporal Lobe, *Journal of Comparative Neurology* **392** (1998), pp. 92-114.
90. R. D. Burwell & D. G. Amaral, Perirhinal and Postrhinal Cortices of the Rat: Interconnectivity and Connections with the Entorhinal Cortex, *Journal of Comparative Neurology* **391** (1998), pp. 293-321.
91. R. D. Burrell, The Parahippocampal Region: Corticocortical Connectivity, *Annals of the New York Academy of Science* **911** (2000), pp. 25-42.
92. P. Lavenex & D. G. Amaral, Hippocampal-Neocortical Interaction: A Hierarchy of Associativity, *Hippocampus* **10** (2000), pp. 420-430.
93. E. Save & B. Poucet, Hippocampal-Parietal Cortical Interactions in Spatial Cognition, *Hippocampus* **10** (2000), pp. 491-499.
94. E. T. Rolls, Hippocampal-Cortical and Cortico-Cortical Backprojections, *Hippocampus* **10** (2000), pp. 380-388.
95. G. Kreiman, C. Koch & I. Fried, Imagery Neurons in the Human Brain, *Nature* **408** (16 Nov 2000), pp. 357-361.
96. C. G. Gross, Coding for Visual Categories in the Human Brain, *Nature Neuroscience* **3,9** (Sept. 2000), pp. 855-856.
97. M. Yukie, Connections Between the Medial Temporal Cortex and the CA1 Subfield of the Hippocampal Formation in the Japanese Monkey, *Journal of Comparative Neurology* **423** (2000), pp. 282-298.
98. N. Tamamaki & Y. Nojyo, Crossing Fiber Arrays in the Rat Hippocampus as Demonstrated by Three-Dimensional Reconstruction, *Journal of Comparative Neurology* **303** (1991), pp. 435-442.
99. N. Ishizuka, J. Weber & D. G. Amaral, Organization of Intrahippocampal Projections Originating From CA3 Pyramidal Cells in the Rat, *Journal of Comparative Neurology* **295** (1990), pp. 580-623.
100. X-G. Li, P. Somogyi, A. Ylinen & G. Buzsáki, The Hippocampal CA3 Network: An In Vivo Intracellular Labeling Study, *Journal of Comparative Neurology* **339** (1994), pp. 181-208.
101. S. A. Deadwyler & R. F. Hampson, Anatomical Model of Hippocampal Encoding of Spatial Information, *Hippocampus* **9** (1999), pp. 397-412.
102. C. Bernard, R. C. Cannon, Y. Ben An & H. V. Wheal, Model of Spatio-Temporal Propagation of Action Potentials in the Schaffer Collateral Pathway of the CA1 Area of the Rat Hippocampus, *Hippocampus* **7** (1997), pp. 58-72.
103. M. F. Yeckel & T. W. Berger, Spatial Distribution of Potentiated Synapses in Hippocampus: Dependence on Cellular Mechanisms and Network Properties, *Journal of Neuroscience* **18,1** (1998), pp. 438-450.
104. K. Wu, K. J. Canning & L. S. Leung, Functional Interconnections Between CA3 and the Dentate Gyrus Revealed by Current Source Density Analysis, *Hippocampus* **8** (1998), pp. 217-230.
105. S. A. Deadwyler, I. R. West, C. W. Cotman & G. S. Lynch, A Neurophysiological Analysis of Commissural Projections to Dentate Gyrus of the Rat, *Journal of Neurophysiology* **38** (1975a), pp. 167-184.

106. F. W. Lothman, Biological Consequences of Repeated Seizures. In *Epilepsy: A Comprehensive Textbook*, Vol. 1 (J. Engel Jr. & T. A. Pedley, Eds., Lippincott-Raven, Philadelphia, PA, 1997), pp. 481-497.
107. M. W. Jung, S. I. Wiener & B. L. McNaughton, Comparison of Spatial Firing Characteristics of Units in Dorsal and Ventral Hippocampus of the Rat, *Journal of Neuroscience* **14** (1994), pp. 7347-7356.
108. A. Deadwyler, J. R. West, C. W. Cotman & G. Lynch, Physiological Studies of the Reciprocal Connections Between the Hippocampus and Entorhinal Cortex, *Experimental Neurology* **49** (1975b), pp. 35-57.
109. E. W. Lothman, E. H. Bertram, III & J. L. Stringer, Functional Anatomy of Hippocampal Seizures, *Progress in Neurobiology* **37** (1991), pp. 1-82.
110. D. G. Amaral, R. Insausti & W. M. Cowan, The Commissural Connections of the Monkey Hippocampal Formation, *Journal of Comparative Neurology* **225** (1984), pp. 307-306.
111. S. Demeter, D. L. Rosene & G. W. Van Hoesen, Interhemispheric Pathways of the Hippocampal Formation, Presubiculum, and Entorhinal and Posterior Parahippocampal Cortices in the Rhesus Monkey: The Structure and Organization of the Hippocampal Commissures, *Journal of Comparative Neurology* **233** (1985), pp. 30-47.
112. S. S. Spencer, P. D. Williamson, D. D. Spencer & R. H. Mattson, Human Hippocampal Seizure Spread Studied by Depth and Subdural Recording: the Hippocampal Commissure, *Epilepsia* **28**,5 (1987), pp. 479-489.
113. N. J. I. Mars & F. H. Lopes da Silva, Propagation of Seizure Activity in Kindled Dogs, *Electroencephalography and Clinical Neurophysiology* **56** (1983), pp. 194-209.
114. N. J. I. Mars, P. M. Thompson & R. J. Wilkens, Spread of Epileptic Seizure Activity in Humans, *Epilepsia* **26** (1985), pp. 85-94.
115. J. Gotman, Measurement of Small Time Differences Between EEG Channels: Method and Application to Epileptic Seizure Propagation, *Electroencephalography and Clinical Neurophysiology* **56** (1983), pp. 501-514.
116. J. Gotman, Interhemispheric Interactions in Seizures of Focal Onset: Data From Human Intracranial Recordings, *Electroencephalography and Clinical Neurophysiology* **67** (1987), pp. 120-133.
117. J. P. Lieb, J. Engel, Jr. & T. L. Babb, Interhemispheric Propagation Time of Human Hippocampal Seizures: I. Relationship to Surgical Outcomes, *Epilepsia* **27** (1986a), pp. 286-293.
118. J. P. Lieb & T. L. Babb, Interhemispheric Propagation Time of Human Hippocampal Seizures: II. Relationship to Pathology and Cell Density, *Epilepsia* **27** (1986b), pp. 294-300.
119. L. P. Lieb, T. L. Babb, J. Engel, Jr. & T. Darcey T., Propagation Pathways of Interhemispheric Seizure Discharges in Human Versus Animal Hippocampal Epilepsy, In *Fundamental Mechanisms of Human Brain Function: Opportunities for Direct Investigation in Association with the Surgical Treatment of Epilepsy* (J. Engel, Jr., G. A. Ojemann, H. O. Luders & P. D. Williamson, Eds., Raven Press, New York, NY, 1987), pp. 165-170.
120. C. L. Wilson, M. Isokawa, T. L. Babb, J. Engle, Jr., L. D. Cahan & P. H. Crandall, A Comparative View of Local and Interhemispheric Limbic Pathways in Humans: An Evoked Potential Analysis, In *Fundamental Mechanisms of Human Brain Function: Opportunities for Direct Investigation in Association with the Surgical Treatment of Epilepsy* (J. Engel, Jr., G. A. Ojemann, H. O. Luders & P. D. Williamson, Eds., Raven Press, London, 1987), pp. 27-38.

121. C. L. Wilson, M. Isokawa, T. L. Babb & P. H. Crandall, Functional Connections in the Human Temporal Lobe. I. Analysis of Limbic System Pathways Using Neuronal Responses Evoked by Electrical Stimulation, *Experimental Brain Research* **82** (1990), pp. 279-292.
122. C. L. Wilson, M. Isokawa, T. L. Babb, P. H. Crandall, M. F. Levesque & J. Engle, Jr., Functional Connections in the Human Temporal Lobe. II. Evidence for a Loss of Functional Linkage Between Contralateral Limbic Structures, *Experimental Brain Research* **85** (1991), pp. 174-187.
123. P. Gloor, V. Salanov, A. Olivier & L. F. Quesney, The Human Dorsal Hippocampal Commissure: An Anatomically Identifiable and Functional Pathway, *Brain* **116** (1993), pp. 1249-1273.
124. L. Acsády, A. Kamondi, A. Sik, T. Freund & G. Buzsáki G., GABAergic Cells Are the Major Postsynaptic Targets of Mossy Fibers in the Rat Hippocampus, *Journal of Neuroscience* **18,9** (1998), pp. 3386-3403.
125. S. S. Spencer & D. D. Spencer, Entorhinal-Hippocampal Interactions in Medial Temporal Lobe Epilepsy, *Epilepsia* **35,4** (1994), pp. 721-727.
126. J. L. Stringer & E. W. Lothman, Reverberatory Seizure Discharges in Hippocampal-Parahippocampal Circuits, *Experimental Neurology* **116** (1992), pp. 198-203.
127. T. A. Ketter, P. J. Andreason, M. S. George, C. Lee, D. S. Gill, P. I. Parekh, M. W. Willis, P. Herscovitch & R. M. Post, Anterior Paralimbic Mediation of Procaine-Induced Emotional and Psychosensory Experiences, *Archives of General Psychiatry* **53** (1996), pp. 59-69.
128. J. R. Stevens, V. H. Mark, R. Erwin, P. Pacheco & K. Suematsu K., Deep Temporal Stimulation in Man, *Archives of Neurology* **21** (1969), pp. 157-169.
129. R. G. Hearh, Pleasure and Brain Activity in Man, *Journal of Nervous and Mental Disease* **154,1** (1972), pp. 3-18.
130. J. D. Liporace & M. R. Sperling, Simple Autonomic Seizures, In *Epilepsy: A Comprehensive Textbook*, Vol. 1 (J. Engel Jr. & T. A. Pedley, (Eds., Lippincott-Raven, Philadelphia, PA, 1997), pp. 549-555.
131. D. R. Fish, Psychic Seizures, In *Epilepsy: A Comprehensive Textbook*, Vol. 1 (J. Engel Jr. & T. A. Pedley, Eds., Lippincott-Raven, Philadelphia, PA, 1997), pp. 543-548.
132. S. B. Bausch & C. Chavkin C., Changes in Hippocampal Circuitry After Pilocarpine-Induced Seizures as Revealed by Opioid Receptor Distribution and Activation, *Journal of Neuroscience* **17,1** (1997), pp. 477-492.
133. M. Hallet, Myoclonus and Myoclonic Syndromes, In *Epilepsy: A Comprehensive Textbook*, Vol. 1 (J. Engel Jr. & T. A. Pedley, Eds., Lippincott-Raven, Philadelphia, 1997), pp. 2717-2723.
134. D. G. Amaral, J. L. Price, A. Pitkänen & T. S. Carmichael, Anatomical Organization of the Primate Amygdaloid Complex, In *The Amygdala* (J. P. Aggleton, Ed., Wiley-Liss, New York, NY, 1992), pp. 1-66.
135. W. Penfield & H. Jasper, *Epilepsy and the Functional Anatomy of the Human Brain* (Little, Brown & Company, Boston, MA, 1954).
136. C. Darian-Smith, I. Darian-Smith & S. S. Cheema, Thalamic Projections to Sensorimotor Cortex in the Macaque Monkey: Use of Multiple Retrograde Fluorescent Tracers, *Journal of Comparative Neurology* **299** (1990), pp. 17-46.
137. W. Kahie, H. Leonhardt & W. Platzer, *Color Atlas/Text of Human Anatomy*, Vol. 3 *Nervous System and Sensory Organs* (Georg Thieme Verlag, Stuttgart, 1993), p. 376.
138. J. L. Vitek, J. Ashe, M. R. DeLong & Y. Kaneoke, Microstimulation of Primate Motor Thalamus: Somatotopic Organization and Differential Distribution of Evoked Responses Among Subnuclei, *Journal of Neurophysiology* **75,6** (1996), pp. 2486-2495.

139. P. Kotagal & H. O. Lüders, Simple Motor Seizures, In *Epilepsy: A Comprehensive Textbook*, Vol. 1 (J. Engel Jr. & T. A. Pedley, Eds., Lippincott-Raven, Philadelphia, PA, 1997), pp. 525-532.
140. P. C. Van Ness, R. P. Lesser & M. S. Duchowny, Simple Sensory Seizures, In *Epilepsy: A Comprehensive Textbook*, Vol. 1 (J. Engel Jr. & T. A. Pedley, Eds., Lippincott Raven, Philadelphia, PA, 1997), pp. 533-542.
141. J. A. Detre, D. C. Alsop, G. K. Aguirre & M. R. Sperling, Coupling of Cortical and Thalamic Ictal Activity in Human Partial Epilepsy: Demonstration by Functional Magnetic Resonance Imaging, *Epilepsia* **37**,7 (1996), pp. 657-661.
142. C. Redecker, C. Bruhl, G. Hagemann, O. Binus & O. W. Witte, Coupling of Cortical and Thalamic Metabolism in Experimentally Induced Visual and Somatosensory Focal Epilepsy, *Epilepsy Research* **27** (1997), pp. 127-137.

∞ ∞ ∞