Dialogue

Our investigations are proceeding to open up new areas in science, and uncertainty exists as to lines of inquiry to pursue, and as to meaning of results obtained. In this section we invite two reviewers to respond to a ground breaking paper. When we receive those comments, we send copies to the original author, who then has opportunity to respond. Then comments and the author's response are published together in this Journal. We hope that this section of dialogue will interest you, will be thought-provoking, that it will help us to think through the hidden assumptions and issues that underlie investigations in this field. [Editor]

Comments on PHOSPHENE IMAGES OF THALAMIC SLEEP RHYTHMS INDUCED BY SELF-HYPNOSIS by Philip Nicholson

Commentary by Mary Jo Peebles-Kleiger, Ph.D.
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I am neither a Neurologist, nor a Neurophysiologist. My area of expertise is Clinical Hypnosis, particularly as practiced within the practice of Psychology. I have some passing familiarity with the practice of Neuropsychology. It is from this base that my perspective and comments originate.

This is a fascinating paper for several reasons. First, the premise, developed with observation, data, logic and circumstantial evidence, that certain closed-eyed, light patterns provide a window into the electrical activity of the brain—-a sort of "shadow box", if you will—is a compelling one. Discovering the neuroanatomical, neurochemical, and neuroelectrical underpinnings of human behaviors has remained a daunting task. Each new technological advance provides just one more spyglass through which to peer into the complicated, buzzing network of our brain. Nicholson's concrete, literal "introspection" technique is a creative and plausible contribution to this endeavor.

Second, although the practice and demonstrated efficacy of hypnosis have been around for several millennia, controversy as to how it works still exists. Nicholson offers an interesting theory for the neuroelectrical underpinnings of that state of heightened absorption and focus that can look like, but is not, sleep. Several recently published articles are apt here. The *International Journal of Clinical and Experimental Hypnosis* devoted two special issues to the topic of hypnosis and pain (Vol. 45, No. 4, October, 1997 and Vol. 46, No 1, January, 1998). Included in the 1998 special issue is an article by Helen Crawford and her research colleagues which details the examination of somatosensory event- related potential correlates of noxious stimulation during "Attend" conditions contrasted with "Hypnotic Analgesia" (HA) conditions.[1] I will quote directly from her article:

> During HA, hypothesized inhibitory processing was evidenced by enhanced N140 in the anterior frontal region and by a prestimulus positive-ongoing contingent cortical potential at Fp1 only. During HA, decreased spatiotemporal perception was evidenced by reduced amplitudes of P200 (bilateral midfrontal and central, and left parietal) and P300 (right midfrontal and central). HA led to highly significant mean reductions in perceived sensory pain and distress. HA is an active process that requires inhibitory effort, dissociated form conscious awareness, where the anterior frontal cortex participates in a topographically specific inhibitory feedback circuit that cooperates in the allocation of thalamocortical activities.[1(pp. 92,93)]

As Nicholson alludes to, Crawford and colleagues have long been interested in the inhibitory capacities of hypnotic subjects, particularly as these capacities relate to pain management.[2(pp. 41-42)] Crawford and Gruzelier also note the significance, to this capacity, of the robust research finding of higher mean theta (in particular, "Class 11 inhibition" theta) in high hypnotizables, both at baseline and during hypnosis.[3] High hypnotizables, successful in reducing or eliminating pain, demonstrate a shift in theta power dominance from the left to the right hemisphere as they are shifting their attention away from painful stimuli.

An interesting fact, relevant to Nicholson's theory, is that "eye fixation" and/or "eye roll" are common (although not universal) hypnotic induction techniques. Eye fixation consists of simply fixating, with eyes open, on an object (close or at some distance), until the eyes close with the aid of suggestion and natural

fatigue. The eye roll consists of holding the head in a "straight-forward" position; looking up towards one's eyebrows; then looking up towards the top of the head; holding the eyes in that position, then slowly closing the eyelids; taking a deep breath and holding that breath; finally, upon releasing the breath, relaxing one's eyes and experiencing a sensation of floating.[4] Both these techniques regularly induce the hypnotic trance state of controlled disattention from irrelevant stimuli. It is interesting how similar these two techniques are to Nicholson's "eye convergence" technique for inducing phosphenes.[2(p. 9)] Of further interest, is Gravitz's report, that occasionally the eye-roll induction can induce a dissociative episode in certain patients.[5] If we consider dissociation the maximum shift of attention away from external cues while still remaining awake, one wonders whether Nicholson's theory of inducing phosphenes (hypothesized to be inducing thalamic spindles) via eye-convergence is relevant here. Namely, are those patients for whom the eye-roll triggers uncontrolled dissociative episodes particularly adept, through neurophysiology or conditioning (but not through conscious control), at inducing and maintaining thalamic spindling. A common clinical intervention for dissociative patients is to teach them greater control over their uncontrolled dissociation, by having them identify auras or triggers for dissociation, by learning how to halt the spin into dissociation, and by learning eventually how to induce and reverse dissociation in a controlled way.

Spiegel believes the capacity for eye-roll (measured in terms of how much sclera shows as the person maintains the up-gaze and slowly closes the lids), is a biological marker, unchanging over time and directly correlated with a person's potential hypnotic responsivity.[4] He states, "The remarkable correlation between the Eye Roll and hypnotizability suggests that trance capacity is either genetically determined or learned so early in life at something like an imprint level that the circuitry is essentially physiological or structural rather than psychological."[4(p. 27)] His "biological marker" theory has met with little support; however, Spiegel remains a respected and talented senior physician-clinician in the field, who has gathered thousands of clinical case examples supporting his beliefs. I wonder whether Nicholson might have thoughts regarding Spiegel's "biological marker" theory, as well as, the relevance of his (Nicholson's) work to the area of pathological dissociation.

Nicholson emphasizes the unique neuroelectrical events that permit a sustained, relaxed attentional focus, without triggering the onset of sleep. Interesting in

this regard is that fact that when working in hypnosis, if a patient "falls asleep" during a clinically-induced trance state, it is usually understood as a "resistance" or as a lapse in the working alliance. In different words, it is understood that the patient's clinical task is to stay attuned to, and focused with the clinician, while at the same time, allowing oneself the suspension of most other reality or environmental orientations. As clinicians, we assume that to fall asleep, the patient has to abandon his/her pinpoint of clinical focus, and instead give oneself over to the sleep process, without maintaining the tension between the two. These clinical observations and Nicholson's theories appear to be in synchrony.

Finally, a third fascinating aspect of Nicholson's work are the potential implications it might hold for a greater understanding of two phenomena: 1) "awareness under surgery,"[6] and 2) the therapeutic action of a relatively new psychological treatment called Eye Movement Desensitization & Reprocessing (EMDR).[7] It has long been understood that the processes of analgesia, amnesia, paralysis, and awareness are independent ones in the complicated operation of chemical anesthesia. Further, many creative research studies have seemed to demonstrate that awareness under anesthesia (usually with amnesia, occasionally without) is a much more common phenomenon than previously realized. I am interested in Nicholson's thoughts about the relevance of his work to the understanding of this phenomenon.

EMDR treatment came onto the scene in the mid-1980's, but only reached high popularity in the past five or so years. It is a remarkably brief, surprisingly effective method of de-energizing, reprocessing, and laying to rest previously intractable traumatic memories. The method consists of targeting a single traumatic scene, identifying a "negative belief' about oneself associated with that scene, and identifying accompanying body sensations. Then, as one holds that trio in mind (image, belief, sensation), one is asked to follow, with saccadic eye movements, the therapist's finger as it goes left to right in rapid succession for several "sets" (about 24 left-to-right movements). As one follows the finger with eye movements, one is to let whatever images, sensations, feelings that arise float across one's mind, like "looking out a train window." By processing sequential bits of traumatic memory in this way, a patient can emerge from a 90 minute session feeling relieved of the burden of ruminative, distressing memories. The effect is typically lasting over time. Shapiro believes the efficacy of the treatment has neurophysiological roots, particularly since the left-right attentional focus appears to be an essential component.[7] Her partic-

ular neurophysiological theory has met with criticism.[8] Again, I am curious whether Nicholson might have some thoughts relevant to her work.

My final reflection and question for Nicholson concerns his interesting statement that, "This kind of wave propagation [excitation spreading horizontally from one TC cell to the next by chemical means], if it were to occur frequently enough and over a long enough period of time, can be expected to stimulate new neuronal growth in the dendritic arbors of those TC cells that often fire synchronously, a mechanism of long-term potentiation that has been found to produce plastic change in the hippocampus."[2(p.38)] I would be interested in Nicholson's reflections on the clinical implications for behavioral change such a statement holds.

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Commentary by Steven Fahrion, Ph.D.

A fundamental conflict underlying societal conceptualization of phenomena in psychophysiologic and especially neurophysiologic research is that of materialism/vitalism. Much of science proceeds as though an event that *can* be explained in terms of physical phenomena alone represents *nothing more than* a materialistic event. Interestingly, the corollary of this perspective has been the assumption that if an event *cannot* be explained in terms of physical phenomena, then it must be assumed that *it does not exist.* (Notice the basic values evident in this byway of the so-called value-free edifice of science).

The "tunnel" image that is commonly experienced in relation to "near-death" presents an example. This experience is attributed by skeptics to "simply the withdrawal of blood flow to the center of the visual cortex as death approaches." In other words, the "tunnel" experience is seen as an epiphenomenon of an event occurring on the material plane (a pattern of blood flow) rather than, for example, as an indication of a pathway or direction that leads the experiencer toward an essentially non-material spiritual transition. Neither does the "skeptical" mode of thought recognize the possibility that a valid *multiply determined* nature may obtain with such an experience, the possibility that the tunnel may represent *both* consolidation of blood flow in the apparatus of the visual field *and* at another level and at the same time, a pathway toward refined transformative experiences. Rather the physical explanation is invoked as the *only* explanation (by Occam's razor) and any resort to a more symbolic or mystical understanding is seen in a perjorative light, as a straining of credulity.

Nicholson's paper[1] is interesting in that it begins with a readily observed (and often ignored) phenomenon and brilliantly traces this observation through a series of levels to specific physical mechanisms. The visual phenomenon is that of internally-generated light sensations or *phosphenes* that are self-induced during quiet, relaxed but expectant visual observation with eyes closed and in the absence of external light. This phenomenon has been dismissed as a [mere] psychological phenomenon.[2] Here we are one step up from raw reductionistic materialism, yet the same basic reductionistic dynamics obtain. One might assume that if phosphenes are [only] a "psychological phenomenon," they represent a kind of *noise* in the visual system that is at the behest of—that is, secondary to—suggestion, expectation, motive and mood, and therefore worthy of dismissal as a real phenomenon in its own right.

While it might have been easy to take the easy route of dismissing the phenomenon at hand or of simply succumbing to a mystical interpretation of the event, Nicholson instead pursued the meaning of the phosphene quarry by fitting together precise observational details with accepted neurophysiologic knowledge, looking for convergences and conflicts. In this process he was at once engaged in a phenomenologic methodologic, hypothesis-seeking study, as well as a controlled single–case mechanisms-testing project. As this process proceeded to conclusion, let us examine what he did find, what we can learn from the conclusions and their implications, and from his methods of proceeding, that might lend support to our general efforts in the field of subtle energies and energy medicine research.

Visual Phosphene Mechanisms

Drawing upon neurophysiologic research and his own detailed observations, Nicholson describes subjective awareness and consensual evidence from the work of others for various possible correlated neural activities induced by deliberate generation of two types of phosphenes. His personal observations and knowledge of human visual-system characteristics integrate and extend understanding of possible neurophysiologic mechanisms underlying not only diverse observations in the mystical literature, but also certain MRI effects on vision, the process of moving from normal waking consciousness to states of consciousness associated with meditation, and self-regulation of pain control.

Nicholson begins by describing a specific, readily practiced autohypnotic procedure that produces two different types of phosphenes: (1) a sequence of repeated "ringed" or annular visual images that appear to "recede into the distance" of the visual field, and that are then followed, as relaxation proceeds, by (2) images that are generally amorphous waves that expand in various ways. Many individuals who engage in meditative practices have reported similar experiences, without forming an explanation of their observations or without even considering what might be learned about the nervous system from such observations.

In contrast, Nicholson applies a detailed knowledge of the visual system to precisely correlate his subjective observations with neurophysiologic fact. After

noting his hypothesis that phosphene displays are related to sleep-wake transitions, he reviews current consensual conceptual understandings of the mechanisms controlling the transition from waking to slow-wave, non-rapid-eye-movement sleep (NREMS). In this conceptualization, NREMS derives from the interaction and convergence of three brain-electrical rhythms in thalamocortical circuits. These include sequentially (1) intermittent fast activity (desynchronization) associated with sensory processing, then (2) initiation of "sleep" spindles in the reticular nucleus of the thalamus, and propagation of spindle activity to the cortex by thalamocortical relay cells, representing the signature of (3) non-REM sleep through activation of the cortical slow rhythm (of less than one cycle per second), which together with its interaction with spindle activity generates the 1-4 cps delta rhythms of deep sleep.

The receding annular phosphenes appear closely correlated in temporal characteristics with thalamic spindle bursts as they are propagated to the lateral geniculate nucleus (LGN). The LGN is comprised of a bilateral semi-cone of neural material which from a variety of evidences, when stimulated in its ventral and lateral aspects, creates a circular peripheral visual field disturbance experienced as a ring of light. As this disturbance is propagated toward the point of the neural cone it then provides an experience of receding movement. Similarly, Nicholson adduces evidence for a neural connection between amorphous expanding phosphene images and delta wave production.

It is impossible to portray in this brief summary the author's presentation of the exquisite details concerning, and the support for, a hypothesized relationship between this visual behavior and proposed patterns of neural activity, which must be left to the article itself. Instead let us turn our attention to the implications of this work.

Pain Control

Nicholson has noted another convergence of sensory and physiologic data in which he has learned to use the phosphene-generating state to support increased ability to control pain. He notes the usual pattern is that thalamic spindle bursts disrupt sensory inputs modulated through the thalamus, inducing a loss

of consciousness. Yet from the author's experience (as a matter of technique) the possibility exists of extending the delay between initiation of spindle bursts in the thalamus and global spindle bursts at the cortex. The importance of this observation is that it may provide a rationale whereby a visually aware brain may maintain consciousness while the non-visual, i.e. pain-sensing thalamic pathways are entrained by synchronous sleep rhythms, reducing or eliminating pain sensations. Based on the author's neurophysiologic understanding, this mechanism could explain observed hypnotic analgesia through dissociation of transmission of visual and nocioceptive sensation by different thalamic pathways.

Hypothesis Seeking

Nicholson has presented a closely-reasoned, fact-studded paper which despite its speculative nature demonstrates an integrity of inquiry that separates it from many speculative pieces. Nonetheless, one is left to raise certain questions in relation to this inquiry. For example, one can not but wonder about the role of the auditory processing system in relation to phosphenes, especially since the author notes that he is able to count the time course of annular phosphenes as "1001 . . . 1002 . . ., etc." Evidently it is possible for him to interact with auditory channels without disturbing the visual channels of the phosphene mechanism. Yet he is unable to engage in such a counting process without interference with the amorphous expanding phosphenes that he associates with delta production. Some elucidation of this difference would be of interest.

In the main, it is difficult to quarrel with this article, representing as it does a virtual *tour de force* example of scientific approach that is quite different from the much vaunted "randomized controlled study" so often considered exemplary of current state-of-the-art science. In fact this article remains a compacted nexus of hypotheses that hopefully will be usefully pursued in graduate theses. Both for the creative quality of its investigatory methodology, its invaluable, emergent, testable neurophysiologic hypotheses and its potentially valuable practical suggestions with regard to control of the effects of nocioceptive stimuli, from a scientific perspective this paper richly deserves the attention that it will hopefully receive. Still, a final question remains: from a mystical perspective

is the annular phosphene ring *nothing more* than the propagation of neural activation through a cone- shaped pan of the visual system? Or, as in the case of the "near-death tunnel" experience is it possible that cross-cultural mystical participation in the *sapphire, the blue pearl, the wheel, the lotus blossom* can represent at once a pattern of neural activation through such structures *and,* with equal validity at a sociologic level of analysis, a signpost to a (hypnogogic) portal leading to spiritual experiences of beauty beyond measure?

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Response by Philip Nicholson

I'd like to thank Drs. Peebles-Kleiger and Fahrion for their close reading of my work and for their insightful comments which stimulated my own thoughts in productive new directions. In this response, I'd like to address the more specific questions before turning to the important philosophical issue raised by Dr. Fahrion.

Why Spindle Waves Are Countable While Delta Waves Are Not

It seems contradictory to claim, as I do, that meditators must learn to keep the mind clear of distracting thoughts in order to induce phosphenes, but also claim that one should be able to split off some small portion of consciousness to engage in a silent "self-talk," counting seconds while watch phosphenes. This splitting turns out to be relatively easy to do, even during seizure-like paroxysms. Even as a novice meditator, one is instructed to "watch the breathing" while keeping the mind clear," which involves this same splitting process. My inability to count delta waves is more a function of the wave characteristics than of maintaining a particular kind of awareness. Spindle waves are easy to count because they involve a stereotyped series of pulses with well-defined borders that arrive at predictable intervals and follow predictable trajectories; by contrast, delta waves arrive at unpredictable times and locations and expand in several directions simultaneously. An analogy that illustrates the different challenges posed by putting these two events in words is the difference between describing a relatively coherent smoke-ring as it leaves the mouth of a smoker versus a cloud of smoke released by an open-mouth exhalation, or, similarly, the difference between describing a tornado cloud versus a wispy billow of cummulus—in both examples, words are easier to attach to the well-formed than to the unformed entity.

The Neural Correlates of Hypnotic Analgesia

I am indebted to Dr. Peebles for referring me to the recent article by Crawford *et al.*,[1] which I had not yet read. Those researchers compared scalp measure-

ments of somatosensory event-related potentials when the same subjects attended to a noxious stimulus applied to the left hand and when hypnotic analgesia (HA) had been induced. They found that, during HA, the scalp potentials were consistent with there being a significant increase in neuronal activity in the ipsalateral anterior cortex (electrode Fp1) milliseconds *before* the stimulus was applied, after which there was a decrease in neuronal activity in the midfrontal (F3, F4) and central (C3, C4) electrodes which monitor the somatosensory and motor cortices. The subjects ranked the pain as significantly less intense during HA.

Crawford *et. al.* propose that the prestimulus increase in neuronal activity in the left prefrontal cortex (Fp1) can be interpreted as reflecting activation of a "supervisory, attention control system of the anterior frontal cortext" that is the neural correlate of a psychological phenomenon they call "active disattention." The anticipatory (pre-stimulus) increase activates "a topographically specific inhibitory feedback circuit that cooperates in the allocation of thalamocortical activities" to produce the decreases in neuronal activity observed in the somatosensory and motor cortices. It is important to note that the right parietal cortex (P4), a region heavily involved in visual processing, did not register the decreases observed in the midfrontal and central electrodes during HA. (No data about the occipital cortices is available, since these regions were not monitored.)

As Dr. Peebles suggests, the Crawford data is consistent with my theory that self-inducing thalamic sleep rhythm phosphenes by keeping the attention fixated on the visual field has the effect of selectively enhancing neuron activity in the vision-related thalamus while at the same time dampening neuron activity in the non-visual thalamus (because the synchronous sleep waves block normal signal-processing). Thus my paper provides a specific mechanism that might be responsible for the "allocation of thalamocortical activities" that produces the pattern of neuronal activity reported by Crawford *et. al.* However, despite the compatibility between these two papers, I confess it seems unlikely to me that *all* cases of HA would involve thalamic gating by the sort of sleep-related mechanism I propose. I have no expertise in the field of clinical hypnosis—in fact, I've never been hypnotized at someone else's direction—but I would guess that hypnotic analgesia evoked by a therapist's instructions is different in some respects—certainly it would seem to involve less time, less effort, and less

concomitant self-awareness than my own self-administered induction. I suspect hypnosis will someday be shown to involve several variants which share some mechanisms in common—eye movements and attentive fixation, perhaps—but differ in other respects. That is how researchers now regard anesthesia, as Dr. Peebles points out. It is also the generally-accepted view about sleep, as a passage from a text on brainstem control of behavioral states suggests: Steriade and McCarley write that the generation of rapid-eye-movement sleep (REM) depends, as do other behavioral states in their own way, on "relatively discrete 'physiological modules,' REM sleep components, that become active in concert because they share a common mechanism(s) of excitability modulation."[2(p.341)]

Retention of Consciousness During Anesthesia

I do not know enough about this subject to respond to Dr. Peebles' question other than by referring again to the 1997 study by Contreras and Steriade.[3] These researchers were surprised to discover that brain waves usually associated with conscious states were also faintly present during anesthesia and during NREMS. They do not to speculate about the significance of this finding, but it would seem to suggest at least a possibility that some level of awareness might be sustainable during two states in which brain activity is minimal. Dr. Peebles' comments certainly motivate me to learn more about the issue.

Physical and Psychological Effects of Repeated Phosphene Induction

Dr. Peebles asks whether there are clinical implications for behavioral change that can be extrapolated from my proposal that repeated induction of phosphenes stimulates new neuronal growth in the visual pathways. The passage she refers to was deleted from the original manuscript she reviewed in order to save space in the published version, so I'm delighted to have an opportunity to summarize the hypothesis here. The basic issue is how to account for the fact that, if mystics self-induce phosphene visions over a long enough period of time, many find that the phosphene changes color from green (or yellow-green) to blue (or violet). In Hindu and Tibetan Buddhist traditions, this shift from green to blue circular images (*chakras*) is

attributed to increased proficiency at meditation and also to an increase in spiritual merit. My hypothesis suggests that the color change is merely a function of frequent repetition.

In the original article, I propose that eye movements and attentive fixation enhance the excitability of geniculate cells in various ways, and that, in response to this abnormal and sustained stimulation, the thalamocortical (TC) cells in the lateral geniculate nucleus (LGN) release significantly greater amounts of neurotransmitter during phosphene induction than they would release during normal NREMS. If so, there will be an abnormal conjunction of two events: (1) the firing of TC cells in response to the pulse of the cortical slow wave of NREMS arriving via corticothalamic projections, and, (2) the presence of an excess of neurotransmitter in the synaptic junctions that linking afferent ganglion axons with their TC cell targets. The excess of neurotransmitter will decrease the control exerted by the cortical slow wave pulses and by the cells' normal inhibitory mechanisms, thereby creating a potential for excitation to spread by chemical means from one TC cell to the next.

A pharmacological model of wave propagation in sheets of cells not linked by local projections has been proposed by Burgi and Grzywacz.[4] This model is of interest for our discussion, because computer simulations using this model produce graphic images of expanding waves that bear a striking resemblance to the amorphous expanding phosphene clouds. In the Burgi-Grzywacz model, a wave of excitation can spread by chemical means alone when potassium ions accumulate in extracellular space while calcium and sodium ions are decreasing. This development increases the chemical gradient between the inside and outside of cells and thus affects the likelihood that cells will depolarize. Once one cells fires and ejects potassium ions into extracellular space, the chemical gradient of neighboring cells is increased, which causes them to depolarize as well. In this manner, a wave of depolarization is initiated. The wave expands across the sheet of cells following a Poisson probability distribution. Eventually, as more and more calcium ions accumulate in extracellular space, this accumulation causes longer after-hyperpolarizations to occur in regions where many cells have already discharged once, a development which prevents the expanding wave from turning back on itself and reactivating regions through which it has already passed.

If this kind of chemically-driven wave propagation were to occur frequently enough and over a long enough period, it is reasonable to expect that new neuronal growth would begin to sprout in the dendritic arbors of TC cells in the LGN, a process which has been shown to produce plastic change in the visual cortex[5] and in the hippocampus.[6] This new neuronal growth in the geniculate laminae will not be organized in relation to the ganglion cell axons that bring afferent sensory signals to the laminae, and it will not be organized in relation to the corticogeniculate projections through which the cortical slow wave pulsations arrive during NREMS; rather, it can be expected to sprout in an unregulated manner. There is, however, a prediction we can make about this unregulated growth. Based on the observation that phosphene visions tend to change color from green to blue over time, we can infer that the new growth must eventually extend deep enough below the superficial laminae (5 and 6) to contact the dendritic arbors of TC cells in the inner laminae (4 and 3), because these inner laminae are the only ones containing cells that encode wavelengths associated with blue.

Dr. Peebles asks if this potentiation of new connections between nerve cells has any clinical implications for behavioral change. My response is that it would seem to me that engaging in behaviors known to potentiate abnormal patterns of new neuronal growth is a risky strategy, one likely to generate unpredictable and possibly negative consequences, not only in the LGN, but throughout the visual system, including the hippocampus, which is the ultimate recipient of visual signals. The hippocampus also happens to be one of the easiest structures to "kindle." Kindling is a method of lowering the seizure threshold of a neuron assembly by administering a low-level stimulus, one that would not ordinarily produce a paroxysmal discharge, repeatedly over a long period until the low-level stimulus has become enough to trigger a seizure. I think that there is some reason for concern that the sort of phosphene induction I have practiced in the past may have had the effect of kindling hippocampal cells. But this is speculative, and it must be weighed against the possibility that this kind of abnormal neuronal sprouting may be an important prerequisite for triggering mystical visions described in Hindu and Tibetan Buddhist yoga texts as the blue ajna chakra, the pearly blue bindu, and the sahasrara, or "lotus of a thousand petals."

The Role of Dissociation in Mystical Vision

Dr. Peebles cites a case reported by Gravitz[7] that raises the question of whether there is some connection between the eye-roll technique for hypnotic induction and pathological dissociation. In the case cited, a patient who had experienced visual hallucinations and delusions prior to hospitalization reacted with those same symptoms when a medical student tried to use the eye-roll technique to hypnotize the patient. In my opinion, the circumstances of that case are complex and susceptible to too many alternative explanations for it to serve as an index case for our discussion. I suggest we shift to a question I'm more qualified to discuss, which is how dissociation might contribute to the generation of mystical visions. I'm now in the process of writing two articles relevant to this topic—articles intended as sequels to the piece on self-induction of thalamic sleep rhythms (already published) and which will collectively present a general theory about the origins of mystical visions. I'd like to sketch here a few points which will be presented in more detail in those as-yet unpublished articles, because I think this will further our discussion of dissociation.

In one article, I continue my approach of analyzing phosphene spatiotemporal characteristics—in this case, analyzing a set of ecstatic, paroxysmal images that resemble accounts of rapturous visions in the mystical literature. My analysis concludes that the following sequence of events must have been taking place at the neurophysiological level for the phosphenes to have acquired their unusual characteristics through the action of wholly endogenous processes: at the outset, (1) the usual induction of thalamic spindles and delta waves, as described in the published paper, but then a new development, (2) destabilization of the sleep rhythm oscillators, caused by feedback from a hyperexcited cortex, and (3) emergence of spike-and-wave complexes, a pattern of brain waves characteristic of some forms of epileptic seizure, (4) failure of the thalamic seizure to generalize across the cortical mantle, as would normally be expected, then (5) precipitation of a remote seizure focus in the left hippocampus, and, finally, (6) propagation from the left hippocampus to other mesotemporal regions, bilaterally, culminating in a partial "seizure." I put "seizure" in quotes because, strictly speaking, this paroxysm did not qualify as a "seizure," since it remained under voluntary control at all stages: by relaxing my attentive fixation I could make the paroxysmal phosphenes (and the muscle spasms) disappear,

and by refocusing, make them reappear. Nor was the paroxysm, strictly speaking, a symptom of "epilepsy," since physicians restrict that diagnosis to cases where seizures occur regularly and where a cause can be identified; in my case, no evidence of epileptic activity was uncovered by a neurological exam.

In a third article, the last in the series, I look at the lives of historical mystics, to be more specific, at a subgroup of mystics who did not show any obvious signs of epilepsy at the time they began their spiritual quest, and who eventually became well-known for having self-induced ecstatic visions of light. (There is another subgroup of mystics to whom visions came unbidden, which implies a different etiology, a subject beyond the scope of the present discussion.) In the lives of the mystics who self-induced visions, I found that six factors are usually present: (1) a major trauma suffered before the age of 8, (2) a major crisis in adult life in the year preceding the first vision, (3) symptoms of depression during that year of crisis, including withdrawal from society, (4) a deliberate perseverance in behaviors that induce fatigue, sleep loss, and sensory-deprivation, often with deliberate intent to punish or "mortify" the body, and, finally, (5) the sustained practice of prayer or meditation which is often explicitly described as involving immobile expectancy, stereotyped eye movements, and intent concentration.

Many of these same factors—childhood trauma, current life crisis, depressive symptoms, and self-infliction of pain—also characterize adult survivors of child abuse or childhood trauma who then go on to develop post-traumatic stress disorders (PTSD).[8-11] PTSD survivors often exhibit an impressive ability to withdraw into themselves and to dissociate themselves from their environment. The correlation of childhood trauma with adult dissociation suggests that dissociation is readily learned by infants and young children who are forced to find a way to escape overwhelming stressors using only a limited repertoire of defensive strategies. The flooding of neurotransmitters and excess glutamate released by exposure to severe or sustained trauma at a very young age may kill neurons in the hippocampus, an irreversible change which would undermine the person's capacity for "self-soothing," that is, the capacity to modulate responses to intense stimulation or anxious feelings. This deficit helps explain why such a person might be motivated in adult life to retain a defense mechanism like dissociation that is relatively primitive and blunt-edged but which was

effective during earlier crises. It also explains why a few persons with this deficit might want to go a step further and make dissociation a guiding principle of their adult lives, becoming, in effect, single-minded seekers of self-transcendence who want to soothe the pain of their existence by immersing themselves in something that is Wholly Other. I believe this is the basic psychological profile that leads people to persevere in the kinds of extreme behaviors that trigger ecstatic, paroxysmal visions of light.

Eye-Movements, Hypnotic Induction, and the Hippocampus

I am not qualified to comment on the neurophysiology of the eye-roll induction technique, nor about the Eye Movement Desensitization and Reprocessing (EMDR) technique, but, as regards the latter, I cannot resist offering one highly speculative observation that may be relevant to the issue of neurological mechanisms. I mentioned earlier that the etiology of paroxysmal visions of light appears to involve an incipient seizure in the thalamus which dies out, at which time a seizure appears at a remote focus in the hippocampus. This tandem appearance suggests that the paroxysmal discharges generated in the visual-relay cells of the thalamus are somehow communicated through the post-geniculate visual pathways to the hippocampus, the ultimate recipient of visual signals. The hippocampus governs the conversion of visual signals and their limbic associations into long-term memory. While the mechanisms involved in this conversion are still not well understood, it has been proposed that one ingredient is hippocampal reprocessing of the day's events during REM sleep. If this theory is substantiated, one might argue that EMDR works as follows: the therapist choreographs the patient's eye movements, visually registering the fact that the patient is in a safe, supportive environment, while, at the same time, the patient, by recalling the original fear experience, is re-activating the sensory regions that were mobilized in that original trauma. When the hippocampus processes the combined material later that night during REM sleep (in which the eyes keep moving back and forth), the old memory traces of fear are amalgamated with new visual signals which record nothing in the visual field that would produce fear. Once this revised, assimilated version is stored in memory, it may become difficult to retrieve the older version in its original form.

Do Reductionist Explanations of Visions Exclude Other Explanations?

Dr. Fahrion poses the most difficult and troublesome question of all, which is this: If we were to possess a complete description of all the physical mechanisms that cause people to see mystical visions, how should we think about the "felt quality" of those visionary experiences? Dr. Fahrion realizes that my paper could be interpreted in a reductionist way, that it could be interpreted to mean that "the sapphire, the blue pearl, the wheel, the lotus blossom" are "nothing more than a propagation of neural activation," rather than perceptions worthy of esthetic appreciation, celebration, symbolic elaboration, or perhaps even veneration. Someone whose accepts the epistemological position that only physical events exist might well argue that the mystical visions I've described are "nothing but" evidence of self-stimulation, even though, given the present state of knowledge, it requires a considerable leap of faith to embrace such a strong form of materialism.

Despite decades of philosophical argument, no one has been able to gain general acceptance for a conceptual framework that establishes a logical relationship between physical events and the subjective or "felt" quality of our human experience. Moreover, in recent years there has been an accumulation of evidence showing that mental events can affect human physiology in dramatic ways, a development which undermines the strict materialist position that mental events are merely epiphenomenal with no real causal effect. For these reasons, and because I value mystical experience, I agree with Dr. Fahrion that we ought to analyze mystical phenomena using several levels of analysis—not only studying physical processes, as I've done here, but also studying psychological and sociological phenomena that have emerged from the interaction of physical processes but which cannot be adequately explained using physical concepts, and, finally, by relinquishing the analytic approach altogether in order to appreciate the "flow" of mystical transport. But having said this, I'd like to add an important qualification: I think this tripartite approach can be fruitfully applied to some kinds of visions but not to others. Let me explain . . .

When I ask myself—"Is the visionary experience as important to me as it was (or is) to mystics who were (or are) ignorant of the underlying physical

causes?"—I realize my answer must be, "No." When mystics do not know how physical mechanisms produce visions, they can reasonably conclude that what they have seen represents some kind of special selection, some special message that empowers them—or commands them—to undertake some important action in the world. I cannot justify the same conclusion, knowing, as I do, that I was myself the sole cause of that vision.

Despite this way of thinking, I continue to hope, like Dr. Fahrion, that it will be possible to evolve a "New Mysticism," one in which the mystic is able to achieve serenity and be inspired to pursue social goods *without having to deny the scientific knowledge that the visions are in fact self-generated.* If this kind of transformulation can occur, then the New Mysticism may continue to play a constructive role in the human future. I think this ideal might be achievable for mystics who contemplate the phosphene images that appear during calm meditation. These images seem to be relatively harmless as well as beautiful and awe-inspiring; why not celebrate their ethereal presence, if one feels so inclined? We know the physical causes of conception and birth, but that doesn't stop us from thinking of birth as an emotionally and morally compelling experience for everyone involved. The same multi-dimensional quality can characterize the mystical visions produced by stable sleep rhythms.

My attitude about the value of visions changes, however, when the issue becomes whether or not a person should deliberately engage in behaviors that are known to stimulate cortical cells to a hyperexcitable state, and then, knowing that this makes a seizure likely, goes ahead and induces phosphenes until a seizure occurs. That strategy is definitely not harmless! I did it, but, in retrospect, it wasn't a good idea. With each paroxysm, chemicals are released that kill neurons or cause new neuronal growth in abnormal patterns. If the seizures are induced repeatedly and over a long enough time, the threshold for seizure generation drops lower and lower. The exitotoxic damage of certain neuron assemblies will almost certainly create epileptic lesions where none existed before. At this point, the seizures will no longer be subject to voluntary control; the mystic has acquired a self-inflicted epilepsy. If we return to the issue posed by Dr. Fahrion with this scenario in mind, it is more difficult to conclude that seizure-induced experiences of ineffable beauty and globalized meaningfulness are worth the price a would-be mystic must pay, that price

being a set of symptoms often seen in patients with temporal lobe epilepsy and known as an "interictal behavioral syndrome."[12-13] Some cultures have developed social institutions that encourage the mystic to engage in repeated seizures while letting other people take over those social roles the mystic can no longer perform, but this is seldom true in our own culture. I know that, in my own case, because of what I've found in my research, I've decided to stop inducing paroxysmal phosphenes, and I'd advise others to do the same.

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