Theoretical

EMPIRICAL STUDIES OF MEDITATION: *DOES A SLEEP RHYTHM HYPOTHESIS EXPLAIN THE DATA?*

Philip T. Nicholson

Abstract

This article reviews the findings of important empirical studies of meditators and shows that these findings can be explained by the hypothesis that meditation is generated by induction of slow wave sleep rhythms. This hypothesis explains why radionucleide imaging (PET, SPECT, and fMRI) studies report increases in neuronal activity in the thalamus (where sleep rhythms are generated) and in the hippocampus (which receives a barrage of vision-related signals caused by manipulations of attention and sleep rhythm activity). It also explains the diverse findings of EEG/QEEG studies, for example, the observed short-term increases in alpha band frequencies and coherence, the subsequent shifts to slower theta/delta frequencies, and the reports of a sudden frequency-splitting and amplitude-doubling concurrent with ecstatic raptures. The author suggests that existing studies of meditation do not account for the likelihood that the theta/delta frequency distribution associated with meditation can be generated by two very different mechanisms: (1) by induction of a drowsy, hypnagogic state ("stage 1" NREMS), an experience familiar to many people and thus easily achievable by novice meditators, and alternatively, (2) by inducing the full progression of thalamic sleep rhythms, an option available only to advanced meditators who are able to move beyond "stage 1" NREMS to induce thalamic spindle-burst typical of "stage 2" NREMS, then beyond that to induce delta waves typical of "stage 3" NREMS. These thalamic delta waves, after augmentation by intracortical circuits, register in the cortical EEG as low-theta/high-delta band activity, making it easy to mistake the underlying mechanism as stage 1 NREMS.

KEYWORDS: Hippocampus, meditation, sleep, thalamus, epilepsy, religion and medicine.

INTRODUCTION

The fundamental problem impeding progress in the scientific study of meditation is not a dearth of experimental data about meditation and its effects on the brain and body; the problem is lack of a suitable theory that explains the experimental data in terms of causal mechanisms. In a companion paper published in a previous issue,[1] we described a set of kundalini-like phosphene images, and, based on analysis of the phosphene spatiotemporal characteristics, proposed the hypothesis that all of the images in this sequence can be explained by (1) the voluntary induction of brain rhythms that would normally appear only during a transition from waking to non-rapid-eye-movement sleep (NREMS), or (2) destabilization of sleep rhythm oscillators and emergence of tandem seizures in two different regions of the brain.[1] In this paper, we review the findings of electroencephalographic (EEG) studies and radionucleide imaging (PET, SPECT & fMRI) studies of meditation and hypnosis in order to assess how well the sleep rhythm hypothesis accounts for the published data.

EEG STUDIES OF MEDITATION

When a human subject is immobile and resting with eyes closed, avoiding active cognition ('keeping the mind empty'), the dominant frequency in the cortical EEG is 'waking occipital alpha,' that is, brain waves with frequencies of about 8 cycles per second (Hz), which is in the lower range of the alpha band (8-12 Hz), and amplitudes of about 20 microvolts (μV), distributed over the posterior (occipital) regions of the scalp. This EEG pattern is the baseline observed in virtually all studies of meditation and hypnosis. Once subjects begin to meditate, the EEG frequencies can become either faster or slower, a paradoxical divergence for which there is still no explanation. We propose that the sleep rhythm hypothesis can explain both changes in terms of a single mechanism, resolving the apparent contradiction by showing that the different outcomes represent different stages in the transition from a waking state to NREMS.

EEG Studies Reporting Frequency Increases

When meditation begins, the waking occipital alpha waves may be 'enhanced' in any of several ways: (1) by increases in the *frequencies* of the alpha waves,

that is, a shift from low alpha (8 Hz) to high alpha (11-12 Hz) or low beta (13-14 Hz);[2-4] (2) by increases in the *coherence* of alpha activity in anterior and posterior electrodes and in frontal electrodes;[5] or (3) by a *doubling of amplitudes* from the 20 μV associated with waking occipital alpha to amplitudes of 40 to 50 μV[4] or even higher—50 to 100 μV.[3,6,7]

The amplitude-doubling can occur at radically different time periods in the meditation session, as shown in Figure 1: two studies show an early shift to amplitude-doubling, but several other studies show a longer time spent in the alpha band before the amplitude-doubling appears, or a steady increase in EEG frequencies up to the point when amplitude-doubling occurs.[2,3,7-9]

The study of Indian yogis by Anand *et al.* showed that high-amplitude alpha waves were not blocked, as normal waking alpha would be, when the subject was exposed to sensory stimuli—opening the eyes, hearing loud sounds, or having a hand placed in freezing water.[3] A similar finding was reported in studies of Zen practitioners by Kasamatsu and Hirai[4] and by Kasamatsu *et al.*[6] Some researchers report that the changes in alpha band activity occur in the first minute after meditation begins. These findings imply that there must be (1) some kind of endogenous 'generator' that can be mobilized quickly—in less than a minute—by an inward focus of attention, (2) that this attention-induced generator must be powerful enough to interfere with the processing of afferent sensory signals, and (3) that the generator must be able to distribute alpha band excitation to the central and forward regions of the cortex, since the alpha activity was observed to spread forward from the occipital region and to be coherent over most of the scalp.

These changes in alpha band activity—and the identity of the endogenous generator that instigates the changes—can be explained by the sleep rhythm hypothesis. In the companion article, our analysis of meditation-induced phosphene images shows how meditation, a complex behavior that combines physical relaxation and an inward orientation of attention, can induce synchronous sleep rhythms that would normally occur only during the transition from waking to NREMS.[1] The sleep rhythm oscillators constitute an endogenous 'generator' that can be mobilized by manipulation of attention to provide the physiological 'energy' to set in motion a complex chain of neural events. One of the most important neural assembles involved

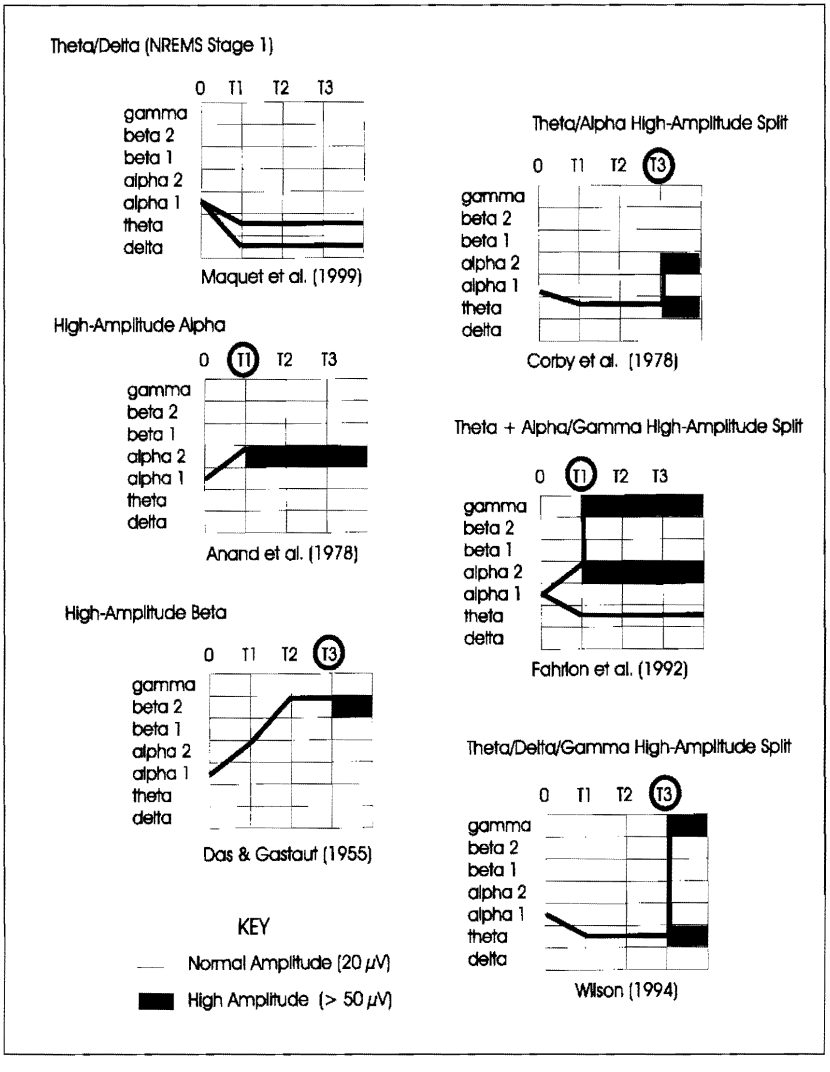


Figure 1. *A Comparison of EEG Changes During Meditation in Six Studies. The theta/delta EEG pattern typical of stage 1 NREMS is shown in the upper left corner. The rest of the charts illustrate cases of amplitude-doubling, some of which also display a 'splitting' in the frequency bands. Note the differences in the timing of the amplitude-doubling in Anand et al.[3] and Fahrion et al.,[7] where the doubling occurs almost immediately after meditation begins (at Time 1 [T1]), with the cases reported by Das and Gastaut,[2] Corby et al.,[8] and Wilson,[9] where the doubling (and splitting) occurs after a longer period of meditation (at T3).*

in the transition to NREMS is the thalamic recticular nucleus (RTN). During the transition to NREMS, the drowsy intermediate state ('stage 1' NREMS) is *usually* eclipsed by loss of consciousness, inaugurating 'stage 2' NREMS. At this point, the RTN has shifted from firing the single spikes associated with the processing of afferent sensory signals during wakefulness to firing its own synchronous bursts of spikes that wax and wane, creating a characteristic 'spindle' pattern in the EEG. Spindle bursts recorded in the thalamus have frequencies that range between 6 to 14 Hz, but sleep spindles detected in the cortical EEG have frequencies that vary within a more narrow range of 12 and 14 Hz. Thus spindle wave activity in the scalp EEG, where it is most often measured, falls into the upper range of the alpha band (8 to 12 Hz) and the lower range of the beta band (13 to 17 Hz).

If the shift from a normal waking state to a meditative state of consciousness is being driven by attention-induced sleep spindles, we would expect to see the baseline pattern of waking occipital alpha (8 Hz) shift to alpha/beta (12 to 14 Hz). This change has been observed in many studies of meditators. Also, we would expect to see the high-alpha activity appear in the central and forward electrodes of the scalp EEG, because RTN sleep spindles are transmitted first to the central and frontal cortices and later to the posterior regions, so that, in effect, the posterior cortices "go to sleep last."[10-12] Thus the appearance of alpha band activity outside of the occipital region, and the coherence of the alpha activity over most of the scalp, is consistent with the cortical activity being driven by subcortical sleep spindles. The timing of the changes in alpha band activity can also be explained by the sleep rhythm hypothesis: as we describe in the companion paper,[1] the RTN fires only 3 to 5 spindle bursts during stage 2 NREMS and then stops automatically. This is usually enough to implement a full synchronization of the cortices and elicit a shift to the delta band activity characteristic of 'stage 3' NREMS. Since the time interval between successive spindle waves is only 5 seconds, the total amount of time necessary to complete a volley of 3 to 5 spindle bursts is only 15 to 25 seconds. Thus the RTN spindle wave generator can distribute high-alpha band activity across the cortex in less than a minute, the time interval mentioned in two studies.[4-5]

But if meditation-induced enhancements of alpha band activity are driven by RTN-generated spindle waves, as we suggest, why don't we see reports of stage

2 sleep spindles in EEG studies of meditators? This doesn't happen, and, if it did, the subject whose EEG showed sleep spindles would be dropped from the study on the grounds that he or she had fallen asleep in the normal sense of the term and thus had slipped out of the meditative state. The sleep rhythm theory suggests that the thalamic sleep spindles become indistinguishable from alpha band activity if the subject is meditating rather than falling asleep in the normal manner.

The view proposed in the companion paper is that meditation is a hybrid state of consciousness in which the attention centers and the posterior, visual cortices are still 'awake' even though neuron assemblies in the rest of the body become so calm that the body's own physiological monitors are 'tricked' into activating the neural assemblies that initiate a transition to NREMS. In this view, meditation involves behaviors that preserve the excitability of neurons in the vision-related and attention-related cortices so that they are not entrained, as they would be during a normal transition to NREMS, by the synchronous pulsations of slow wave sleep rhythms. The reasons why neurons in the attention-related and vision-related cortices preserve their ability to fire independently is presented briefly in the companion article and examined at greater length in an earlier work by the present author.[13] The basic proposition is that keeping the eyes converged and the attention fixated on the center of the visual field facilitates neurons in the visual cortices so that they remain in a highly excitable state. This prevents their entrainment by synchronous spindle waves relayed from the thalamus. Because cells in the visual cortices retain their ability to discharge, recover, and discharge again, the spindle waves register in the visual cortices as if they were afferent sensory signals of colored annuli moving away from the viewer.

This analysis is consistent with theories about sleep-wake mechanisms proposed by Steriade and McCarley, who point out that each behavioral state is supported by activation of neurons in several "modules" that could, in theory, be activated in abnormal combinations if the patterns of neuron excitability were somehow altered by behavioral manipulations: "The argument is straightforward: increasing the excitability of neurons in a particular brain region or nucleus will increase the probability of behaviors or physiological functions controlled or mediated by that region. . . . We thus see REM sleep as composed of relatively discrete 'physiological modules,' REM sleep components, that become

active in concert because they share a common mechanism(s) of excitability."[1(p.342)] Similarly, NREMS (and waking) are supported by different patterns of neuron pool excitability. The sleep rhythm hypothesis of meditation proposes that meditation involves activation of the physiological modules that govern NREMS in concert with the physiological modules that support spatial attention during a waking state.

One important consequence of the attention-related and vision-related cortices being still 'awake' at a time when the rest of the body is, technically speaking, 'asleep,' is that, in this condition, the RTN spindle bursts are likely to merge indistinguishably into the alpha band activity registered by the cortical EEG. Alpha waves are thought to result from oscillations of neurons in intracortical networks, not from subcortical processes.[15] When spindle bursts from the RTN reach their cortical targets, these signals are likely to stimulate an increase of neural activity in those intracortical networks which will increase the frequencies of the alpha waves registered by the cortical EEG. Also, alpha waves wax and wane in a manner that closely resembles the pattern of RTN spindle bursts, making it difficult to distinguish RTN-generated spindle waves from cortically-generated alpha waves.[16] Therefore, if our hypothesis that the RTN drives alpha band activity during meditation is correct, we can expect to see the following patterns: (1) an appearance of RTN-driven alpha wave activity in the central and forward electrodes within one minute; (2) a shift in RTN-driven alpha band frequencies from the lower range of 8 Hz to the upper range of 11 to 12 Hz, a frequency range that overlaps spindle wave frequencies in the scalp EEG; (3) an RTN-driven increase in the coherence of alpha band activity in all electrodes; and (4) an RTN-driven persistence of alpha activity despite exposure to sensory stimuli. All of these patterns have been reported in EEG studies of experienced meditators. This suggests that the sleep rhythm hypothesis can account for the enhancements of waking occipital alpha reported by EEG studies of experienced meditators, but what about the other studies in which meditation produced a slowing of the EEG? Can the sleep rhythm hypothesis also explain this?

Studies Reporting Decreases in EEG Frequencies

A second EEG pattern often observed in meditators, healers, and hypnotized subjects is a slowing of the EEG in which waking occipital alpha is replaced

by theta (4-8 Hz) and high-delta (0.5-4 Hz) frequencies distributed over the central and frontal cortices, with alpha rhythms becoming fragmented to the point that they constitute less than half of an epoch.[8,9,17,18] This theta-delta-fragmented-alpha pattern is the conventional EEG criterion for 'stage 1' NREMS, a transitional state of relaxed, drowsy consciousness during which the EEG still shows intermittent desynchronizations that indicate some residual processing of thoughts or afferent sensory signals.[11-13,20,21] In EEG studies that use subjects who are not 'experienced' meditators, there is a prolongation of this stage 1 NREMS pattern for the duration of the experiment. In those studies, if the EEG detects sleep spindles or other signals characteristic of slow wave sleep, the researchers rightfully conclude that the subject has slipped out of meditation/hypnosis and has fallen asleep in the normal sense of the term (stage 2 NREMS), which results in that subject being dropped from the study. But the shift from stage 1 to stage 2 NREMS will have a very different outcome if the study focused on 'experienced' meditators who have learned how to manipulate eye movement and spatial attention to keep the visual cortices 'awake' after the RTN starts firing the sleep spindles characteristic of stage 2 NREMS. In these experienced subjects, the onset of thalamic spindle volleys manifests as changes alpha band activity, specifically, increases in alpha band frequencies, coherences, and amplitudes.

When the RTN stops firing spindle bursts, delta wave activity (0.5 to 4 Hz) will be generated in the circuits that link the thalamus and the cortex, a process described more fully in the companion article.[1] If the subject is meditating, we can expect that thalamic spikes in the delta frequency range will stimulate the intracortical neuron networks that generate alpha (which are being kept 'awake' by behavioral manipulations) to greater activity. In the case of spindle activity, the increase was about 4 Hz (from 8 to 12 Hz). If the same augmentation applied to delta wave activity and its intracortical reverberations, we would expect to find activity in the frequency range of 4.5 to 8 Hz, that is, from high delta through the theta band to low alpha. This means that both experienced and inexperienced meditators may have a cortical EEG that shows a theta/delta predominance—and that the underlying mechanisms producing these brains waves are the same—but that the inexperienced meditators are still in stage 1 NREMS while the experienced meditators have moved on into stage 2 NREMS.

Amplitude-Doubling and 'Splitting' Phenomena

In EEG studies of experienced meditators, there is often a sudden, dramatic doubling of wave amplitudes in one or more of the frequency bands after meditation is underway. Several EEG studies of experienced meditators fit this profile. In a study of a Hindu yogi by Das and Gastaut, the first change after meditation began was an increase in the frequency of occipital alpha from 8 Hz to 12 Hz, followed by increases through the beta band—from low-beta (13-17 Hz) to mid-beta (18-20 Hz) to high beta (20-25 Hz) and then very high beta ('gamma') frequencies (> 25 Hz).[2] The gamma wave amplitudes suddenly doubled from 20 μV to 50 μV in all electrodes. The yogi later identified this point as the peak of ecstasy. In a number of cases involving experienced meditators and healers, the amplitude-doubling occurs simultaneously in two frequency bands. For example, in a study of Zen monks by Kasamatsu and Hirai, the EEG recordings of a few of the older and more experienced monks showed an early increase in alpha wave amplitudes at the outset of meditation, then, after prolonged meditation, there was a sudden split in which rhythmic, high-amplitude theta trains were present at the same time as high-amplitude alpha.[4] A study by Corby *et al.* that compared Tantric yoga practitioners to controls found that 17 out of 20 of the experienced practitioners showed changes in both alpha and theta frequencies that were significantly greater than in the control group.[8] One subject reported having a "near-samadhi" experience at the time the scalp EEG showed a doubling of amplitudes occurring in a 'split' pattern: alpha wave amplitudes surged to 100 μV, and theta wave amplitudes surged contemporaneously to 150 μV.

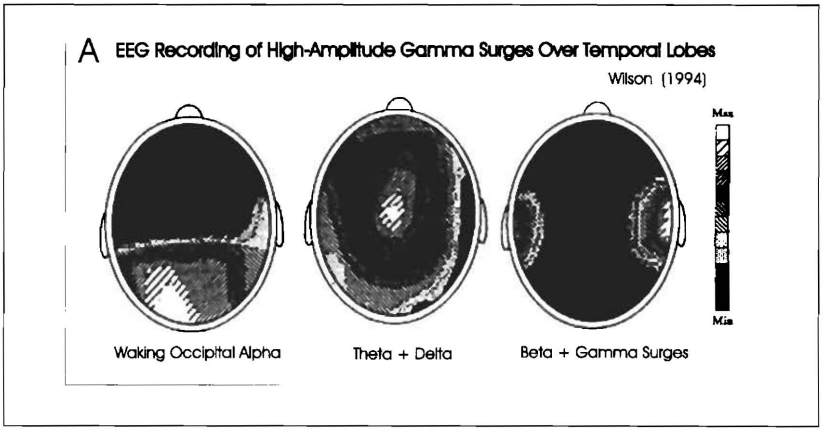
Similar splits involving slower frequencies (theta or alpha) and higher frequencies (gamma) have been reported in more recent studies which were able to make use of quantitative EEG mapping techniques that make it possible to visualize the topographical distribution of gamma surges and 'split' phenomena. In a study of an experienced therapeutic touch healer by Fahrion *et al.*, researchers observed a sudden 'splitting' in which amplitude-doubling occurred simultaneously in both the high-alpha and gamma band frequency bands.[7] The gamma surges reached amplitudes of 100 μV with the maximal amplitudes localized over the temporal lobes in a distinctive 'ear-muff' pattern. Similarly, in an EEG study of an experienced healer by Wilson, the initial EEG pattern was theta-delta predominance that slowed down even more

and became more coherent in the "really low frequencies, like low delta," at which point there was a sudden split between the theta-delta frequencies and gamma band frequencies (64 Hz - 128 Hz) and a near-doubling of normal amplitudes (20 to 39 μV).[9] Wilson also studied a group of subjects who were attending a meditation training seminar that taught them to deepen their trances by listening to audio tapes with binaural beats embedded. After the training was over, Wilson found that "a sudden shift from slow waves to temporal lobe activation occurred in 80% of these subjects, with temporal activation reaching 64 Hz in one female subject."[9] The gamma surges were associated with subjective reports of "ecstatic" or "out-of-the-body" experiences. This high-amplitude activity appeared in one of two distributions, as shown in Figure 2: as a table-top' of theta/delta rhythms surrounding the central vertex (electrode Cz), or as gamma surges localized over one (or both) of the temporal cortices—the 'ear-muff' pattern reported in the earlier study by Fahrion *et al.*[7] More recently, LORETA studies by Lehmann *et al.*[40] and DeLuca and Daly,[19] (in this issue), show a similar pattern of high-amplitude activity localized over the vertex and temporal lobes in experienced Tibetan-Buddhist meditators.

EEG Patterns Associated With Simple Partial Seizures

Wilson initially thought that the gamma surges he was recording were generated by temporal lobe seizures: "When people are really going up into ecstatic or transcendent experiences, I've seen them go up to 120 to 150 μV activity in the temporal lobe. . . . The first time I saw this evidence of temporal activation, I thought the person probably had a temporal lobe seizure, and I continued to think that for some time because of the intensity of that response."[9(p.181)] He later rejected this hypothesis, reasoning that the experiences of ecstasy and transcendence related by his subjects were incompatible with their having seizures. Now more recent studies of symptomatology in patients with simple partial seizures of hippocampal and mesotemporal origin provide new support for a mesotemporal seizure hypothesis.

In a 1997 study, Pacia and Ebersole compared seizure activity in depth electrodes with activity in the scalp EEG with the goal of determining what kinds of intracranial events were associated with ictal scalp rhythms.[22] They found that seizures confined to the hippocampus produced very little change



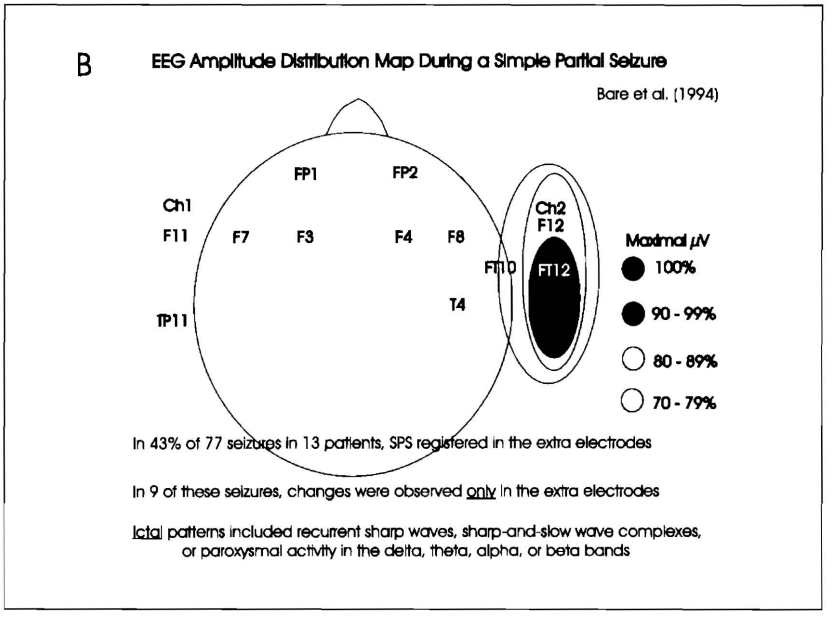


Figure 2. *EEG Changes Associated With Simple Partial Seizures. (A) Topographic maps of EEG frequency distributions recorded by Wilson,*[9] *showing a map of resting occipital alpha, a table-top distribution of slow theta/delta activity at the vertex, and gamma frequency surges localized over the temporal lobes in an 'earmuff' pattern. (B) A map of EEG amplitude distributions during simple partial seizures, adapted from Bare* et al.,[23] *that shows amplitude maxima that were revealed by addition of subtemporal electrodes that are not normally used. Of the 77 seizures in 13 patients, 43% registered in the extra, subtemporal electrodes; in 9 of these seizures, the changes appeared only in the extra electrodes.*

in the scalp EEG, but, if the seizures spread from the hippocampus to the adjacent temporal cortex, then a regular theta-alpha rhythm (5 to 9 Hz) appeared over temporal and subtemporal regions of the scalp, and also over the vertex: "Seizure activity confined to the mesotemporal area can produce an unusual scalp EEG seizure pattern, as we have demonstrated, in which the predominant rhythm recorded from standard 10-20 placements is around the vertex. Although seemingly unlateralized, this type 1B pattern is in fact quite localizing, because a particular cortical orientation is necessary for its generation. Specifically, cortical EEG sources in basal temporal cortex produce a dipolar field with a net vertical orientation. This results in scalp EEG field maxima, positive and negative, being located at the vertex and base of the skull, respectively. Few electrodes record from the latter, except subtemporal and sphenoidal placements."[22(p.650)] This typical rhythm is thought to be driven by a seizure inside the hippocampus. A slightly different rhythm was associated with mesotemporal seizures that began outside the hippocampus in the adjacent mesotemporal cortex. At the onset of these neocortical seizures, "Low-voltage, high-frequency discharges in the beta and 'gamma' range that could be focal or regional were a consistent finding. . . A recent study reported frequencies as high as 120 Hz at the onset of neocortical seizures," and, when these gamma surges occurred, the "focal gamma activity . . . could be missed easily with sparse electrode coverage."[22(p.652)]

During simple partial seizures, the scalp EEG records the greatest wave amplitudes in subtemporal electrodes, that is, in extra electrodes added the normal 10-20 array to cover the most lateral temporal regions, as shown in Figure 2.[23] Note how the amplitude maxima are localized over the temporal lobes in the 'earmuff' pattern described in the studies of experienced meditators and healers.

The sleep rhythm theory described in the companion paper describes how mesotemporal seizures (and ecstatic raptures) can be triggered with surprising ease when sleep rhythm oscillators are destabilized during the transition from stage 1 to stage 2 NREMS. This is consistent with reports in the medical literature that the incidence of partial seizures is high during the transition to NREMS: for example, in a study of 1,116 seizures in 188 epileptic patients, Bazil and Walczak found 30% of complex partial seizures occurred at sleep onset, and, of these, almost 75% occurred during the earliest stages of the

transition to NREMS—25% during the drowsiness of 'stage 1,' and 54% during stage 2 when the RTN fires synchronous spindle bursts.[24] This high incidence of partial seizures at sleep onset may reflect the fact that the power spectra for both spindle waves and delta waves reach their highest levels during the first NREMS episode of a night.[25,26]

A history of self-inducing seizures may explain why the EEGs of some experienced meditators and healers undergo the dramatic amplitude-doubling and frequency splitting reported in the EEG studies. For example, Fahrion et al. reported a very rapid shift from a state of waking occipital alpha to a theta/alpha/gamma split, indeed, the healer-subject said he couldn't maintain the prescribed baseline state of resting alpha when in the presence of a patient he was supposed to heal.[7] Both Colby et al. and Wilson describe subjects who were able to induce gamma surges and amplitude-doubling with relative ease.[8,9] We suggest that, in order to generate this EEG pattern, all of these subjects must have had some pre-existing epileptiform vulnerabilities, or, if not, that they must have had a history of self-inducing sleep rhythms and destabilizing them to trigger seizures. These prior seizures would likely kindle changes in their neural circuitry that facilitate rapid onset of mesotemporal seizure when these individuals used meditation skills to mobilize the RTN spindle generator.

Our analysis of the phosphene imagery in the companion paper[1] implies that even one ecstatic mesotemporal seizure may create some residual disinhibition of hippocampal cells. This 'leak' of excess excitability to retrohippocampal regions could undermine the stability of cells in the mesotemporal region, creating the kind of "sensory-limbic hyperconnection" proposed by Bear as a primary cause of limbic seizures.[27] If so, then the EEG recordings of meditators who show rapid onset of a high-amplitude 'split' EEG pattern strongly suggest that the surges recorded represent the reactivation of an epileptogenic circuit that was already well established before their participation in the EEG study.

Partial Seizures and Anomalous Electrostatic Phenomena

The sleep rhythm hypothesis may help explain an otherwise anomalous finding in an interesting study of meditators published by Green *et al.*[28] The researchers

constructed a copper-walled room in a laboratory to isolate meditators electrically from their environment and compared measurements of body potential surges induced by 'exceptional' healers (non-contact therapeutic touch practitioners) and 'regular' subjects. They found that regular subjects never produced body-potential surges over 4 volts while meditating, whereas exceptional subjects induced many surges above 4 volts. These surges ranged from 4 to 221 volts and lasted from 0.5 seconds to 12.5 seconds with a median duration of 3.6 seconds. The total charge on the body remained constant throughout the surge, and the readings quickly returned to the baseline value once the surge was over. The researchers concluded that the surges must have been produced by an oscillation of charges within the body with nothing emitted into the environment, and they pointed out that "there are as yet no known psychophysiologic or biophysical explanations for such large-magnitude electrical phenomena, 10^3 times greater than EKG voltages, and 10^6 times larger than EEG voltages."[28(p.69)] A possible explanation for these findings is suggested by the sleep rhythm theory of meditation that we've just discussed.

The experienced healers could induce oscillations inside the body that register as surges of body potential by focusing attention inward and activating the RTN spindle generator. If they were to induce a single spindle spike-burst—say, one that lasted for 0.5 seconds—and it took this spindle wave 4 seconds to move through the thalamocortical projections and register in the visual cortices (as a 'receding annulus'), then this timing for the duration of a spindle wave would closely match the 3.6 median duration of the body potential surges $\geq$ 4 volts. If the healer were to induce a full volley of spindles—that is, 3 to 5 bursts every 5 seconds with each wave lasting 4 seconds—the event might take anywhere from 12 to 25 seconds, which encompasses the 12.5 second duration of the body potential surges observed.

The magnitude of the body potential surge produced by this kind of attention-induced spindle generation might be explained as a consequence of the subjects' having 'forcibly' induced spindle bursts frequently while practicing meditation—and by their having pushed sleep rhythm induction to the point of destabilization and seizure generation, as described in the companion paper.[1] By inducing subclinical seizures over many years, these subjects may have realigned their neural circuitry in a manner that facilitates destabilization of sleep rhythm oscillators which then triggers mesotemporal seizures. As noted

above, a simple partial seizure of mesotemporal origin may have few, if any, clinical symptoms, so that the meditators would not be motivated to seek medical treatment for their condition. With this epileptogenic circuitry in place, the simple expedient of focusing attention to mobilize the RTN spindle wave generator would be enough to trigger paroxysmal activity in the mesotemporal region. Based on this hypothesis, we would expect the EEG to show high-amplitude waves with the characteristic table-top or ear-muff distribution of maximum wave amplitudes while the healers are generating the surges in body potential, and this is indeed what occurs.

NUCLEAR IMAGING STUDIES OF MEDITATION

In recent years, EEG studies of meditators have been supplemented by studies using nuclear imaging technologies like positron emission tomography (PET), single photon emission computed tomography (SPECT) and functional magnetic resonance imaging (fMRI). The PET and SPECT devices measure changes in cerebral blood flow which can be attributed to changes in neuronal metabolism in a particular region of the brain. Several PET studies show that a significant increase in metabolic activity in the anterior attention networks of the frontal cortices is induced by the inward orientation of attention associated with hypnosis and with meditation.[17,18,29-32] Particularly interesting for our inquiry are the SPECT studies of Tantric Buddhist meditators by Newberg *et al.*, which show that, in addition to the expected increase in the activation of frontal regions that mediate attention (the dorsal prefrontal cortices), there is increased blood flow to the *thalamus*—where the RTN is located—and to the right medial temporal lobe—where the *hippocampus* is located.[30-32] It is important to add that other PET studies have also reported significant increases in the activation of the hippocampus in meditators and in hypnotized subjects.[17,18]

There is, however, one set of findings that would appear to be incompatible with a sleep rhythm hypothesis. If meditation produces its effects by activating the brain rhythms of NREMS, then neurons in the striate (primary) visual cortices should be activated as well as neurons in the rest of the visual cortices—the extrastriate (secondary and tertiary) visual cortices—and their limbic targets, including the structure that receives the highest representations

of visual signals—the hippocampus. Two PET studies—a study of meditators by Lou *et al.* and a study of hypnotized subjects by Maquet *et al.*—appear to contradict this prediction of the sleep rhythm theory.[17,18] Both of these studies report activation of the extrastriate visual cortices and their limbic targets but no activation of the primary striate visual cortices. This pattern of activation resembles the pattern generated by rapid-eye-movement sleep (REMS).[33-37] Since sleep rhythms are emitted by RTN spindle bursts and relayed via thalamocortical projections to the striate visual cortex, a finding that the striate visual cortex is not activated by meditation contradicts the sleep rhythm hypothesis. There is, however, an important confounding variable that limits the applicability of these two PET studies of meditation and hypnosis.

A REM-like pattern of activation in the visual cortices is also found in studies of mental imagery—and when the protocols of the PET studies of hypnosis and meditation are examined, it can be seen that the tasks they assigned were essentially mental imagery tasks. But, in the present author's experience, most types of meditative practice use imaginary mental imagery as an aid for novices while at the same time advising more advanced practitioners to remain 'detached' from mental distractions by practicing techniques that keep the mind 'empty' of experiential (dream-like) imagery. The importance of this distinction is revealed in a PET study of mental imagery by Kosslyn *et al.*[38] The researchers asked subjects to recall visual images of simple everyday objects that they'd just been shown earlier in the experiment, and they found that, during performance of this assigned task, there was a REM-like pattern of changes in cerebral blood flow, that is, an increase in activation of the extrastriate visual cortices and in the limbic targets of extrastriate projections, including the hippocampus, and no increase of activation in the primary, 'striate' visual cortices or in the thalamus. This mental imagery experiment by Kosslyn *et al.* closely resembles the PET study of meditation by Lou et al. where the subjects were experienced 'Yoga Nidra' meditators but the experimental protocol instructed them to complete their normal 'empty mind' meditation before coming to the laboratory—and during the experiment, they were asked to perform visualizations and other imagination-based tasks. Similarly, in the PET study of hypnosis by Maquet *et al.*, the task assigned to hypnotized subjects was mental recollection of autobiographical scenes. Thus neither of these PET studies is designed to detect what happens during an 'empty mind' meditation.

But by accident the same PET study by Kosslyn *et al.* also showed the pattern of cerebral blood flow associated with 'empty mind' meditation. This happened

before the subjects started the assigned task. While baseline measurements were being taken, the subjects were instructed to lie down, rest with their eyes closed, and "have it black in front of the mind's eye"—essentially the same behaviors involved in meditation except that attention is not fixated (at least not pursuant to instructions). After the experiment was over, the researchers were surprised to find a significant increase in activation of the primary visual cortices during the 'empty mind' baseline period before the experiment began. This finding suggests that the pattern of cerebral blood flow during 'empty mind' meditation will more likely resemble the pattern associated with NREMS than the pattern associated with REMS and mental imagery experiments, if a study were designed to detect the difference.

CONCLUSION

In this paper we have shown that the main results reported in all of the empirical studies of meditators, including those studies that use radionucleide imaging techniques (PET, SPECT, and fMRI) and those that use EEG/QEEG techniques, can be explained in mechanistic terms as a voluntary activation of corticothalamic sleep rhythm oscillators of NREMS stages 1 through 3. When these studies report different (and sometimes contradictory) outcomes, these differences can also be explained by reference to the sleep rhythm hypothesis presented in the companion paper published in a preceding issue.[1] Moreover, the sleep rhythm hypothesis can explain why certain patterns appear in the empirical studies—for example, why EEG studies of advanced meditators typically report sudden shifts to high-amplitude gamma surges at the vertex and temporal regions ("ear-muffs") typical of hippocampal seizures, and why the radionucleide imaging studies typically show that significant increases in metabolic activity specifically in the dorsolateral prefrontal cortex, the thalamus, and the hippocampus, findings for which no one has yet proposed an detailed mechanistic explanation. Thalamic activation is consistent with activation of an RTN spindle burst volley lasting less than one minute, a phenomenon usually associated with stage 2 NREMS, and with activation of the post-thalamic primary striate cortices as reported by Kosslyn *et al.*,[38] the extrastriate visual association cortices, and their limbic targets, including that region where visual signals achieve their highest representation, namely, the hippocampal/post-hippocampal complex.

The ability of human subjects to learn how to induce and sustain sleep rhythms that normally occur during the transition to NREMS—and to do so without the loss of consciousness that would normally occur during this transition—suggests that there must be a hybrid behavioral state of *sleep-conditioned consciousness.* It is important to acknowledge that, at present, the sleep rhythm hypothesis is based on an ad hoc analysis of information that was originally obtained by introspection, which interposes two important methodological flaws that limit the generalizability of these conclusions; nonetheless, it would seem reasonable, given the remarkably detailed and comprehensive explanatory power of the sleep rhythm hypothesis of meditation, to design experimental studies of meditation (and self-hypnosis) with more sensitive measures of the sleep rhythm activity that can take place in subcortical regions and be masked by perturbations in neuron assemblies near the cortical surface.

EPILOGUE

Several new studies of meditation have come to our attention since the time that this paper and its companion article were submitted for publication in May, 2001. These studies are consistent with the analysis of EEG data and radionucleide imaging data we've presented here, thus adding new evidence that supports the sleep rhythm hypothesis of meditation and self-hypnosis.

Four New Studies of Meditators

In a functional magnetic resonance imaging (fMRI) study of meditators using Benson's "relaxation response," Lazar *et al.* found that there were significant increases in signal activity in the forward attentional centers of the dorsolateral prefrontal cortex, in the hippocampal complex, in the temporal cortices, and in the anterior cingulate cortex (ACC), among other regions.[39] This constellation of increased neuronal activity is similar to the SPECT studies by Newberg *et al.*,[30-32] and it is consistent with our sleep rhythm hypothesis.

In two new studies of advanced Tibetan Buddhist meditators using quantitative electroencephalography (QEEG) supplemented by Low-Resolution

Electromagnetic Tomography (LORETA), DeLuca and Daly[19] and Lehmann *et al.*[40] both report that, once the most advanced stage of meditation was attained, high-amplitude gamma activity appeared at either the vertex or the two temporal regions which (i.e., the "ear-muff" pattern) or in both locations. This distribution of high-amplitude activity is the same as that reported in the studies by Fahrion *et al.*[7] and Wilson.[9] Our discussion points out the similarity of these brain wave patterns to those detected during hippocampal seizure.[22,23]

Finally, a new EEG study of kundalini yoga practitioners by Arambula et al.[41] confirms the observations of two earlier studies[4,5] that alpha/beta frequencies that are originally restricted to the occipital region ("waking occipital alpha") spread forward to the central and forward cortices in less than a minute, a phenomenon we attribute to voluntary induction of RTN spindle bursts analogous to spindle bursts that occur during stage 2 NREMS.

An Important Qualification

Nothing we have written here about the nature of the mechanisms that generate high-amplitude gamma surges or surges of body potential should be interpreted as detracting from evidence that encounters with healers may produce genuine healing in patients, even if no detectible energy is actually being transferred between the healer and the patient. Evidence that positive effects can result from these encounters is now becoming available in ever-increasing quantities— as, for example, in the works of Locke and Colligan,[42] Benor,[43] and Sternberg.[44]

If we suppose that, as part of the healing protocol, some healers self-induce a hippocampal seizure in themselves, this strategy might produce an effect on the patient by an indirect means: one of important side-effects of hippocampal seizures is a heightened sense of emotionality and of the meaningfulness of life as well as a sense of personal destiny, all of which might enhance the healer's confidence in his or her efficacy. If this increased self-confidence in the healer then evokes greater confidence in the patient, so that the patient becomes more optimistic about the prospects of a cure, then the healer's increase in self-confidence might have the effect of mobilizing the patient's own inner healing resources and invigorating the so-called "placebo response." In addition, the

healer's meditative stance might help the patient achieve a more relaxed state comparable to the easily-accessible "relaxation response" popularized by Benson.[45] This change might be enough to increase dopamine levels (Kjaer *et al.*[46]) or nitrous oxide levels (Stefano[47]), two biochemical substances which are now being investigated as ingredients that modulate the body's ability to heal itself.

• • •

CORRESPONDENCE: Philip Nicholson • 52 Norfolk Road • Chestnut Hill, MA 02467-1832 • Voice: 617-566-7429 • Fax: 617-738-7634 • Email: pnicholson@earthlink.net

REFERENCES & NOTES

1. P. T. Nicholson, Meditation, Slow Wave Sleep, and Ecstatic Seizures: The Etiology of Kundalini Visions, *Subtle Energies and Energy Medicine* **12**,3 (2001), pp. 183-230.
2. N. N. Das & H. Castaut, Variations De L'Activite Electrique Du Cerveau, Du Coeur, Et Des Muscles Squelettiques Au Cours De La Meditation Et De L'Extase Yogique, *Electroencephalography and Clinical Neurophysiology* Supplement **6** (1995), pp. 211-219.
3. B. K. Anand, C. S. Chhina & B. Singh, Some Aspects of Electroencephalographic Studies in Yogis, *Electroencephalography and Clinical Neurophysiology* **13** (1961), pp. 452-456.
4. A. Kasamatsu & T. Hirai, An Electroencephalographic Study on the Zen Meditation (Zazen), *Folia Psychiatrica et Neurologica Japonica* **20**,4 (1966), pp. 315-336.
5. F. Travis & R. K. Wallace, Autonomic and EEG Patterns During Eyes-Closed Rest and Transcendental Meditation (TM) Practice: The Basis for a Neural Model of TM Practice, *Consciousness and Cognition* **3** (1999), pp. 302-328.
6. A. Kasamatsu, T. Okuma, S. Takenaka, E. Koga, K. Ikeda & H. Sugiyama, The EEG of "Zen" and "Yoga" Practitioners, *Electroencephalography and Clinical Neurophysiology* Supplement **9** (1957), pp. 51-52.
7. S. L. Fahrion, M. Wirkus & P. Pooley, EEG Amplitude, Brain Mapping, & Synchrony In & Between A Bioenergy Practitioners & Client During Healing, *Subtle Energies and Energy Medicine* **3**,1 (1992), pp. 19-52.
8. J. C. Corby, W. T. Roth, V. P. Zarcone, Jr. & B. S. Kopell, Psychophysiological Correlates of the Practice of Tantric Yoga Meditation, *Archives of General Psychiatry* **35** (1978), pp. 571-577.
9. E. Wilson, The Transits of Consciousness, *Subtle Energies and Energy Medicine* **4**,2 (1994), pp. 171-185.
10. K. P. Wright, Jr., P. Badia & A. Wauquier, Topographical and Temporal Patterns of Brain Activity During the Transition from Wakefulness to Sleep, *Sleep* **18**,10 (1995), pp. 880-889.
11. H. Tanaka, M. Hayashi & T. Hori, Statistical Features of Hypnagogic EEG Measured by a New Scoring System, *Sleep* **19**,9 (1996), pp. 731-738.
12. H. Tanaka, M. Hayashi & T. Hori, Topographical Characteristics of Slow Wave Activities During the Transition from Wakefulness to Sleep, *Clinical Neurophysiology* **111** (2000), pp. 417-427.

13. P. T. Nicholson, Phosphene Images of Thalamic Sleep Rhythms Induced by Self-Hypnosis, *Subtle Energies and Energy Medicine* **7**,2 (1996), pp. 111-148.
14. M. Steriade & R. W. McCarley., *Brainstem Control of Wakefulness and Sleep* (Plenum Press, New York, NY, 1990), p. 342.
15. J. S. Barlow, *The Electroencephalogram: Its Patterns and Origins* (MIT Press, Cambridge, MA, 1993).
16. M. Corsi-Cabrera, M. A. Guevara, Y. Del Rio-Portilla, C. Arce & Y. Villaneuva-Hernandez, EEG Bands During Wakefulness, Slow-Wave and Paradoxical Sleep as a Result of Principle Component Analysis in Man, *Sleep* **23**,6 (2000), pp. 738-744.
17. P. Maquet, M. E. Faymonville, C. Degueldre, G. Delfiore, C. Franck, A. Luxen & M. Lamy, Functional Neuroanatomy of the Hypnotic State, *Biological Psychiatry* **45** (1999), pp. 327-333.
18. H. C. Lou, T. W. Kjaer, L. Friberg, C. Wildschiodtz, S. Holm & M. Nowak, A ^{15}O-H_2O PET Study of Meditation and the Resting State of Normal Consciousness, *Human Brain Mapping* **7** (1999), pp. 998-105.
19. John DeLuca & Ray Daly, The Inner Alchemy of Buddhist Tantric Meditation: A QEEG Case Study Using Low Resolution Electromagnetic Tomography (LORETA), *Suble Energies and Energy Medicine* **13**,2 (2002), pp. 65-118.
20. M. Steriade, D. A. McCormick & T. Sejnowski, Thalamocortical Oscillations in the Sleeping and Aroused Brain, *Science* **262** (1993), pp. 679-685.
21. R. Broughton & J. Hasan, Quantitative Topographic Electroencephalographic Mapping During Drowsiness and Sleep Onset, *Journal of Clinical Neurophysiology* **12**,4 (1995), pp. 372-386.
22. S. V. Pacia & J. S. Ebersole, Intracranial Substrates of Scalp Ictal Patterns From Temporal Lobe Foci, *Epilepsia* **38**,6 (1997), pp. 642-654.
23. M. A. Bare, T. H. Burnstine, R. S. Fisher & R. P. Lesser, Electroencephalographic Changes During Simple Partial Seizures, *Epilepsia* **35**,4 (1994), pp. 715-720.
24. C. W. Bazil & T. S. Walczak, Effects of Sleep and Sleep Stage on Epileptic and Nonepileptic Seizures, *Epilepsia* **38**,1 (1997), pp. 56-62.
25. F. Ferrillo, M. Beelke, F. De Carli, M. Cossu, C. Munari, C. Rosadini & L. Nobili, Sleep-EEG Modulation of Interictal Epileptiform Discharges in Adult Partial Epilepsy: A Spectral Analysis Study, *Clinical Neurophysiology* **11** (2000), pp. 916-923.
26. F. Ferrillo, M. Beelke & L. Nobili, Sleep EEG Synchronization Mechanisms and Activation of Interictal Epileptic Spikes, *Clinical Neurophysiology* **11**, Supple. 2 (2000), pp. S65-S73.
27. D. M. Bear, Temporal Lobe Epilepsy: A Syndrome of Sensory-Limbic Hyperconnection Cortex, *Cortex* (1979), pp. 357-384.
28. E. E. Green, P. A. Parks, P. M. Guyer, S. L. Fahrion & L. Coyne, Anomalous Electrostatic Phenomena in Exceptional Subjects, *Subtle Energies and Energy Medicine* **2**,3 (1995), pp. 69-94.
29. H. Herzog, V. R. Lee, T. Kuwert, K. H. Langen, E. R. Kops & L. E. Feinendegen, Changed Pattern of Regional Glucose Metabolism During Yoga Meditative Relaxation, *Neuropsychobiology* **23** (1990-1991), pp. 182-187.
30. A. Newberg, A. Alavi, M. Baime, P. D. Mozley & E. d'Aquili, Measurement of Cerebral Blood Flow During the Complex Cognitive Task of Meditation Using HMPAO-SPECT Imaging: A Preliminary Study, *Journal of Nuclear Medicine* **38** (1997), p. 95P.
31. A. Newberg, A. Alavi, M. Baime & M. Pourdehnad, Cerebral Blood Flow During Meditation: Comparison of Different Cognitive Tasks, *European Journal of Nuclear Medicine* **27**,8 (2000), p. 1104 (PS_375).

32. A. Newberg, A. Alavi, M. Baime, M. Pourdehnad, I. Santanna & E. d'Aquili, The Measurement of Regional Cerebral Blood Flow During the Complex Cognitive Task of Meditation: A Preliminary SPECT Study, *Psychiatry Research: Neuroimaging Section* **106** (2001), pp. 113-122.
33. N. Hofle, T. Paus, D. Reutens, P. Fiset, J. Gotman, A. C. Evans & B. E. Jones, Regional Cerebral Blood Flow Changes as a Function of Delta and Spindle Activity During Slow Wave Sleep in Humans, *Journal of Neuroscience* **17**,12 (1997), pp. 4800-4808.
34. P. Maquet, D. Dive, E. Salmon, B. Sadzot, C. Franco, R. Poirrier & C. Franck, Cerebral Glucose Utilization During Stage 2 Sleep in Man, *Brain Research* **571** (1992), pp. 149-153.
35. P. Maquet, Sleep Functions and Cerebral Metabolism, *Behavioral Brain Research* **69** (1995), pp. 75-83.
36. P. Maquet, J-M Peters, J. Aerts, G. Delfiore, C. Degueldre, A. Luxen & C. Franck, Functional Neuroanatomy of Human Rapid-Eye-Movement Sleep and Dreaming, *Nature* **383** (1999), pp. 163-166.
37. A. R. Braun, T. J. Balkin, N.J. Wesensten, F. Gwadry, R. E. Carson, M. Varga, P. Baldwin, C. Belenky & P. Herscovitch, Dissociated Pattern of Activity in Visual Cortices and Their Projections During Human Rapid Eye Movement Sleep, *Science* **279**,2 (1998), pp. 91-95.
38. S. M. Kosslyn, W. L. Thompson, I. J. Kim & N. M. Alpert, Topographical Representations of Mental Images in Primary Visual Cortex, *Nature* **378** (1995), pp. 496-498.
39. S. Lazar, G. Bush, R. Gollub, G. Fricchione, G. Khalsa & H. Benson, Functional Brain Mapping of the Relaxation Response and Meditation, *Neuroreport* **11** (2000), pp. 1581-1585.
40. D. Lehmann, P. Faber, P. Achermann, D. Jeanmonod, L. Gianotti & D. Pizzagalli, Brain Sources of EEG Gamma Frequency During Volitionally Meditation-Induced, Altered States of Consciousness, and Experience of Self, *Psychiatry Research: Neuroimagining Section* **108** (2001), pp. 111-121.
41. P. Arambula, E. Peper, M. Kawakami & K. Gibney, The Physiological Correlates of Kundalini Yoga Meditation: A Study of a Yoga Master, *Applied Psychophysiology and Biofeedback* **26**,2 (2001), pp. 147-153.
42. S. Locke & D. Colligan, *The Healer Within: The New Medicine of Mind and Body* (E. P. Dutton, New York, NY, 1986.).
43. D. Benor, *Healing Research: Holistic Energy Medicine and Spirituality, Vol. I: Research in Healing* (Helix Editions Ltd., Deddington, Oxfordshire, United Kingdom, 1992).
44. E. Sternberg, *The Balance Within: The Science Connecting Health and Emotions* (W. H. Freeman, New York, NY, 2001).
45. H. Benson, *The Relaxation Response* (Morrow & Co., New York, NY, 1975).
46. T. Kjaer, C. Bertelsen, P. Piccini, D. Brooks, J. Alving & H. Lou, Increased Dopamine Tone During Meditation-Induced Change of Consciousness, *Brain Research and Cognitive Brain Research* **13**,2 (2002), pp. 255-259.
47. G. Stefano, Molecular Processes That Have the Potential to be Part of the Placebo Effect and the Relaxation Response, *Proceedings of Science and Mind/Body Medicine Course* (Harvard School of Medicine, Department of Continuing Education, May 3, 2002).

∞ ∞ ∞