

# ***ASK THE DOCTOR(S)!***

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## ***Is it possible to have such Seizures WITHOUT an abnormal EEG??***

***Response by Mike Martelli, Ph.D***

In observing general clinical and neurologic practice, I have seen epilepsy both overdiagnosed (esp. from psychogenic forms, but also from other physiologic etiologies), and UNDERDIAGNOSED (esp. for difficult to diagnose types like TLE and frontal lobe seizures, which often require sleep deprivation, 24 hour monitoring, special montages, depth recordings, clinical correlation, etc.). Also, there are a very large number of organic conditions that can imitate epilepsy, (The main differential diagnoses for physiologic events that present as seizures include autonomic disorders, cardiac events, cerebrovascular disease, drug toxicity, metabolic disorders, migraines and sleep disorders) and that require good differential diagnostic and neurologic and general medical knowledge and skills. My observations are that a good differential diagnostic work up for the more difficult to diagnose epilepsies, as well as the potential organic "imitators" of epilepsy is NOT typically performed, and often not performed at the specialty services (esp. when there is psychological disturbance or vulnerability).

I am including some tables from "Imitators of Epilepsy" re: these differentials. In my experience, if clear EEG evidence is not found, the assumption is typically "psychogenic", with only a little attention paid to a few of the more common other organic rule outs. Also, I see many cases where prolactin levels, etc., are not recorded, even sometimes when it is asked for, with reliance only on EEG (sleep deprived usually only when specifically requested, even though epilepsy has various subtypes for which EEG has variable specificity (see the cool table below). Ruling out epilepsy should include, in cases like this, observation & clinical symptom correlation, EEG video monitoring, post seizure blood prolactin levels, placebo or suggestion induction; familial epilepsy risk, responsiveness to anti-seizure medication; neurologic history, etc.). Secondly, physiological syndromes (e.g., cataplexy, transient ischemic attacks, syncope — as noted above, and in table below) must be ruled out. In addition, I might wonder about the possibility of increased vulnerability to effects of more subtle epileptogenic activity (e.g., variable slow wave activity) due to neurologic vulnerabilities (i.e., recent head injury, HTN, etc.)...

Finally, the psychogenic type of Non-Epileptic Seizures (NES) are diagnosed by analyzing the patient's history. A number of signs suggesting psychogenic rather than epileptic episodes, including frequent episodes unaffected by anticonvulsants, coexistence of psychological symptoms or associated psychiatric disease or

vulnerabilities (e.g., anxiety, depression, inappropriate affect or lack of concern, somatization or hysterical personality traits, childhood abuse / trauma, a history of poor adjustment or under achievement, abnormal interaction with significant others, and the presence of emotional triggers.

And, in cases where there is neurologic history consistent with seizures, and psychologic factors indicating predisposition to psychogenic seizures or influence, the likelihood of both organic seizures and conditioning of true seizures to non-epileptic organic imitators and /or even psychogenic manifestations may be high.

Also (and again) differentiation of nonepileptic seizures is much more difficult for partial seizures versus tonic/clonic seizures. The diagnosis of seizures and non-epileptic seizures (NES: both organic and nonorganic) is ultimately probabilistic and fallible. (e.g., for a few summary paragraphs about NES, see: Heilbronner, R.L., Martelli, M.F., Nicholson, K., Zasler, N.D. Heilbronner, R.L. (2002). Masquerades of Brain Injury. Part IV: Functional Disorders. The Journal of Controversial Medical Claims, 9, 3, 1-7., available/ downloadable at <http://villamartelli.com>

Best,

MFM

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### **Selected Tables Excerpted and Adapted FROM:**

**Fisher, R.S., ed, (1994). Imitators of Epilepsy. New York: Demos Publishers.**

#### CHAPTERS

1. Introduction to Spells
2. Seizures That Do Not Look Like Seizures
3. Electroencephalography and the Diagnosis of Epilepsy
4. Serum Prolactin in the Diagnosis of Epilepsy
5. Syncope
6. Cerebrovascular Imitators of Epilepsy
7. Migraine and Epilepsy
8. Sleep Disorders That Imitate Epilepsy
9. Movement Disorders That Imitate Epilepsy
10. Endocrine Imitators of Epilepsy
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12. Dizziness and Vertigo As Imitators of Epilepsy
13. Psychiatric Imitators of Epilepsy
14. Psychogenic Seizures
15. Episodic Dyscontrol and Malingering

16. Hyperventilation
17. Imitators of Epilepsy in Children
18. Approach to the Diagnosis of Possible Seizures

### **Table 1-1. Imitators of Epilepsy**

#### SYSTEMIC (NONNEUROLOGIC) DISORDERS

##### SYNCOPE

- Respiratory syncope
- Hypotensive syncope
- Cardiac syncope
- Circulatory syncope
- Reflex syncope

##### DIRZINESS AND VERTIGO

##### ENDOCRINE DISORDERS

- Hypoglycemia
- Growth hormone excess
- Thyroid disease
- Hyperglycemia Tetany
- Pheochromocytoma
- Carcinoid
- Paroxysmal dysautonomia
- Menstrual disorders

##### TOXIC AND METABOLIC DISORDERS

- Waxing and waning delirium
- Alcohol or drug-related syndromes

##### GASTRIC REFLUX OR ESOPHAGEAL

##### RECURRENT ABDOMINAL PAIN, CHILDREN

##### NEUROLOGIC DISORDERS

##### REACTIVE SEIZURES

##### CEREBROVASCULAR ATTACKS

- Transient ischemic attacks
- Transient global amnesia
- Drop attacks

##### COMPLICATED MIGRAINE

##### SLEEP DISORDERS

- Narcolepsy
- Idiopathic hypersomnolence
- Isolated cataplexy
- Sleep apnea
- Somnambulism
- Hypnogenic paroxysmal dystonia
- Night terrors
- Enuresis Bruxism

- Paroxysmal arousals
- Periodic movements of sleep
- REM behavior disorder

#### INTERMITTENT MOVEMENT DISORDERS

- Chorea and athetosis
- Ballisms
- Paroxysmal ataxia
- Tics and Tourette's syndrome
- Dystonia
- Paroxysmal dyskinesia
- Paroxysmal choreoacanthocytosis
- Torticollis
- Restless legs (periodic movements of sleep)
- Blepharospasm
- Hemifacial spasm
- Meige's syndrome
- Tardive Dyskinesia
- Cramps and spasms
- Isaac's syndrome and stiff-man
- Myoclonus
- Asterixis
- Startle disease (hyperekplexia)
- Tremor

#### INTERMITTENT INTRACRANIAL HYPERTENSION

#### PAROXYSMAL MULTIPLE SCLEROSIS

#### PSYCHIATRIC DISORDERS

#### CONVERSION REACTIONS

- Psychogenic seizures
- Somatic delusional disorder
- Elective mutism

#### EPISODIC DYSCONTROL

#### PANIC ATTACKS

#### HYPERVENTILATION EPISODES

#### MALINGERING

#### DISSOCIATIVE STATES

- Psychogenic fugue
- Multiple personality disorders
- Depersonalization

#### DEPRESSION

#### PSYCHOSES

#### SELF-MUTILATORY BEHAVIOR

#### CULTURE-BOUND SYNDROMES

**Table 1-2. Epidemiology of Spells**

## 2A. Yearly Incidence of Some Episodic Events

Event	Reference
Incidence/100,000	
Syncope	Savage et al. 1985 (30)
3,000	
Dizziness	Sloane 1989 (40)
2,600	
Migraine, headache	Linet and Stewart 1984 (31)
730	
Epilepsy	Hauser 1990 (19)
50	
Transient ischemic attack	Lai et al. 1990 (33)
23	
Transient global amnesia.	Hodges 1991 (32)
3	

## 2B. Prevalence of Some Episodic Events

Event	Prevalence/100,000	Reference
Sleep disorders	38,000	Bixler et al. 1979 (35)
Migraine, headache	15,000	Linet and Stewart 1984 (31)
Epilepsy	2,500	Hauser et al. 1991 (21)
Transient ischemic attack	660	Fratiglioni et al. 1989 (34)
Psychogenic seizures	150	Gumnit and Gates 1986 (37)

Incidence: new cases per 100,000 population per year; prevalence: total cases per 100,000 population at any one time. Figures are approximations, for general comparisons. Actual incidence and prevalence depends on specific populations and studies.

**3-4. The Usefulness of EEG in Various Epileptic Conditions**

Syndrome	Overall Usefulness	False Negatives	False Positives	Comments
Neonatal convulsions	+++	Normal in benign forms	±	Most valuable in Differentiation of severe & benign forms

West syndrome (Infantile spasms)	++++	May occur (normal but very high voltage)	±	Hypsarrhythmic pattern practically diagnostic	
Febrile convulsions	+	Normal EEG the rule	Abnormal EEG suggestive of afebrile seizures	Value of EEG lies in predominantly normal findings	
Lennox-Gastaut syndrome	+++	Almost negligible	+	(difficult differential diagnosis)	A diagnosis grossly based upon EEG. Sleep record helpful
Primary generalized epilepsy (in general)	+++	Almost negligible	+	(seizure free relatives, other conditions)	Often sleep record needed for EEG efficacy, photic stimulation helpful
a) pure petit mal absences	+ +++	No petit mal absence without spike-waves			Extremely reliable correlation
b) juvenile myoclonus epilepsy	+ +		+		Sleep record, photic stim helpful
		?			
Benign Rolandic epilepsy	+++	Almost negligible	++	(seizure free despite predisposition)	Often sleep record needed for EEG evidence
Benign Occipital epilepsy	++	+	++	(seizure free despite predisposition)	
Temporal Lobe epilepsy	++	+		(repeat records often needed)	Sleep record a necessity. EEG DX may require patience, persistence, novel montages
Frontal Lobe Epilepsy	+	+			Difficult EEG Diagnosis (frequently missed)
Motor Cortex epilepsy	0-++	May be ++			A weak spot in EEG Diagnosis
					?

**Table 2-1. Common Atypical Manifestations of Seizures**

Clinical Behavior	Brain Area Involved (Citations)
Vocalization	Dominant or nondominant hemisphere Temporal lobe (23) Frontal lobe (6)
Thrashing	Medial or orbito-frontal lobe (15)
Expressive Aphasia	Broca's area Prefrontal lobe Supplementary motor area (40)
Receptive Aphasia	Dominant or nondominant posterior parietal (41) Dominant or nondominant posterior parietal (41)

Dreamy states, confusion	Parahippocampal and fusiform gyrus (40)
Formed visual hallucinations	Occipital association cortex (29)
	Mesio-temporal structures (29)
Complex auditory hallucinations	Lateral or mesio-temporal lobes

**Table 5-3. Signs and Symptoms for Differentiating Seizures and Syncope**

<u>Finding</u>	<u>Syncope</u>	<u>Seizure</u>
Prodromal Symptoms	common, lightheaded or dizzy, sweating, pallor, typically a dimming of vision	less common, very brief aura
Onset of Symptoms	gradual onset and progression	sudden and quick
Vital Functions	depressed pulse, heart rate or respirations	rapid heart rate, elevated blood pressure
Position or Posture	usually occurs in upright position or with exertion	occurs in any position
Motor Activity	lie motionless or mild limited clonic or myoclonic jerks	prominent tonic, clonic or myoclonic jerks, some automatisms
Recovery of Consciousness	prompt	slow
Incontinence	uncommon	more common

**Table 7-2. Interactions of Migraine and Epilepsy**

- Mistaken diagnoses
- Basilar artery migraine
- Postictal migraine
- Migraine-triggered seizure
- Epileptogenic lesion from migraine
- Overlap syndromes
  - Benign occipital and rolandic epilepsy
  - Migrainous and convulsive hemiplegia
  - Mitochondrial encephalomyopathy

**Table 7-3. Factors in Differential Diagnosis: Epilepsy Versus Migraine**

**Favors Epilepsy:**

- Partial motor seizures
- Recurrent spontaneous tonic-clonic seizures

- Seizures during sleep
- Interictal spikes, sharp waves and spike-waves
- Photoc provocation
- Sudden onset

**Favors Migraine:**

- Recurrent spontaneous headaches
- Photophobia
- Scotoma
- Simple visual hallucinations
- Provoked by diet
- Responds to "migraine" medications
- Gradual onset

**Common to Both:**

- Positive family history
- Confusion
- Stupor
- Loss of consciousness
- GI upset
- Flushing and autonomic symptoms
- Vertiginous dysequilibrium
- Complex visual hallucinations
- Uncinate (smell/taste) auras
- Transient focal neurological deficits
- Provoked by hormonal changes
- Occasional CSF abnormalities
- Abnormal EEG during attack
- Responds to "anticonvulsants"
- Anxiety and depression

**Table 9-1. Motor Imitators of Epilepsy**

- Chorea and athetosis
- Ballismus
- Paroxysmal ataxia
- Tics and Tourette's syndrome
- Dystonia
- Paroxysmal dystonic choreoathetosis
- Paroxysmal kinesiogenic choreoathetosis
- Torticollis
- Restless legs (periodic movements of sleep)
- Blepharospasm
- Hemifacial spasm
- Meige's syndrome
- Tardive dyskinesia
- Akathisia



- Cramps and spasms
- Issac's syndrome and stiff-man
- Myoclonus Asterixis
- Startle disease (hyperekplexia)
- Tremor

**Table 9-2. Paroxysmal Choreoathetosis vs. Epilepsy**

	PDC	PKC	Epilepsy
Prevalence	Very rare	Rare	Common
Family history	+++	++	+
Usual age of onset	0-5	5-20	1-35
Sex predominance	Male	Male	Equal
Duration of movements	Mins-hours	Secs-Mins	Secs-Mins
Precipitated by movements	-	++++	+
Ability to suppress	+++	+++	+
EEG during episode	WNL	WNL	Abnormal
Antiepileptic response+	+++	+++	+

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Ability to suppress	+++	+++	+
EEG during episode	WNL	WNL	Abnormal
Antiepileptic response	+	+++	++++

**TABLE 15-2. Criteria for Intermittent Explosive Disorders**

Discrete episodes of loss of control

Assault or destruction of property during episodes  
 Minimal or no provocation  
 Behavior disproportionate to triggers  
 Not due to other psychiatric causes  
 Onset/remission in minutes to hours  
 Subsequent regret  
 Normal impulsivity between episodes  
 Prodromal affective/autonomic symptoms  
 Partial amnesia syndrome, except as correlated to associated conditions, such as occasional tumors or epilepsy

**TABLE 18-1. Imitators of Epilepsy**

- Seizure
- Vasovagalsyncope
- Hypovolemic or hypotensive syncope
- Cardiac arrhythmias
- Circulatory obstruction
- Transient ischemic attacks
- Transient global amnesia
- Vasospastic migraine
- Vertigo
- Hypoglycemia
- Gastric reflux or esophageal spasm
- Sleep disorders
- Breathholding spells
- Waxing and waning delirium
- Alcohol or drug-related syndromes
- Intermittent movement disorders
- Intermittent intracranial hypertension
- Conversion reactions (hysteria)
- Panic attacks
- Hyperventilation episodes
- Depression
- Malingering

<b>TABLE 18-3. Useful Physical Exam Maneuvers in Diagnosis of Spells</b>	
<b>Maneuver</b>	<b>Condition</b>

<ul style="list-style-type: none"> <li>• Orthostatic blood pressures</li> <li>• Listen for bruits</li> <li>• Heart sounds</li> <li>• Check for nystagmus</li> <li>• Nylen-Barany</li> <li>• Hyperventilation</li> <li>• Observe for sleepiness</li> <li>• Tics, tremors, chorea</li> <li>• Mental status exam</li> <li>• Nonphysiological findings</li> <li>• Psychiatric screen</li> </ul>	<ul style="list-style-type: none"> <li>• Syncope</li> <li>• Cerebrovascular disease</li> <li>• Arrhythmias, embolic sources</li> <li>• Vestibular disease</li> <li>• Benign positional vertigo</li> <li>• Hyperventilation spells</li> <li>• Hypersomnia</li> <li>• Movement disorder</li> <li>• Delirium</li> <li>• Functional disorder</li> <li>• Affective or thought disorder</li> </ul>
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**TABLE 8-2. Sleep Conditions Imitating Epilepsy**

Daytime sleep attacks	Enuresis
Cataplexy	Bruxism
Hypnagogic hallucinations	Periodic movements of sleep
Sleep paralysis	Paroxysmal dystonias
Night terrors	REM behavior disorder Sleepwalking

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***How do I find Information on (Mercury Amaglams and Cognitive Functioning..... (or anything else...))??***

***Response by Mike Martelli, Ph.D***

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I strongly recommend some very useful literature search tricks.

Begin with the basics. . .

Below, in the first group, are ref's produced in a 10 sec. Entrez Pubmed (Nat. Lib. Med) search

<http://www.ncbi.nlm.nih.gov/entrez/query.fcgi>

using keywords "Amalgam and cognitive"

In the second group, I used "Mercury and cognitive".

Getting more serious, I would spend at least 10 minutes, or even an hour, and really explore the literature using conceptual search skills and apply available search knowledge. For example, modifying keywords (e.g., cognition, intellectual, memory, attention, neuropsychological) should lead to some more refs. . . subsequent narrowing would help identify the most relevant, although I like to also maintain personal control and do the narrowing visually, from broader searches, as the more selective searches can miss several relevant papers. Also, displaying the abstracts, versus just titles, will help prevent missing many relevant papers that simply don't have completely descriptive titles. Also, there are numerous other databases, including several good ones and several semi-adequate ones (PsychFirst, Variations of Nat. Lib Med and PubMed, Psychabstracts, Findarticles.com, etc.) and using multiple databases always expands the number of references (these are available in guide articles ref'd below). In addition, identifying the major relevant journals, which can usually be accomplished using a combination of Google, and common Trends, in any area (e.g., Neurotoxicology, Ann Rev Pharmacol and Toxicology, Arch Toxicology, Res in Toxicology . . . ) will allow visiting the Websites of the journals, where you can perform topical searching of abstracts . . . which, again, seems to always produce some papers which do not show up in Pubmed, PsychFirst, etc., list searches. Finally, and I certainly recommend this, visit my website, or Ken Popes Website (kspope.com) or Pauline Wallins website (<http://www.drwallin.com>) where you will find guides on efficient literature searches. If I can recommend my website, I link guides from Ken Pope, Pauline Wallin, myself and several others that address this and related topics for psychology and medicine on

<http://villamartelli.com>

under the "General Internet Resources" section.

These strategies and guides are really useful, but do not seem to be used in most published research papers, or even many comprehensive reviews.

Best,

MFM

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Keywords:

Amalgam and Cognitive

Factor-Litvak P, Hasselgren G, Jacobs D, Begg M, Kline J, Geier J, Mervish N, Schoenholtz S, Graziano J.

Mercury derived from dental amalgams and neuropsychologic function.

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Environ Res. 2000 Sep;84(1):1-11.

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## ***Isn't Awareness Always Necessary for TBI Rehabilitation?***

***By Mike Martelli, PhD***

***Concussion Care Centre of VA, Pinnacle Rehabilitation & Tree of Life***

Conventional wisdom, and the guiding principle for virtually all cognitive and neuropsychological rehabilitation efforts, assumes that awareness of impairments is a necessary first step in the rehabilitation of such residual brain injury deficits as memory, problem solving and self control difficulties. Although this assumption is logically and intuitively appealing, it can at the same time restrict & block creative rehabilitation interventions. Further, improperly applied, it can even undermine the higher level purpose of optimizing adaptive behavior.

For example, inexperienced therapists often blindly attempt to increase awareness without evaluation of a persons self esteem, abstract or higher level reasoning, or general coping abilities. They might employ confrontational strategies, without realizing that unawareness may be due to concrete thinking and/or may be is a coping defense that protects a fragile self esteem or prevents overwhelming feelings of helplessness. By breaking down denial, or increasing awareness without first offering new coping strategies, significant distress and emotional and behavioral deterioration can be produced. From a stress and coping perspective, old, ineffective strategies should be replaced with newer, more effective ones. However, ineffective coping is often better than no coping, and coping strategies should never be removed without replacement.

A striking example of overzealous attempts to increase awareness is that of a 23 year old severe brain injury survivor who was unaware of the extent of his residual memory, mobility and behavior problems and seemed to cope with job and friend loss through excessive drinking. He initially was referred to a clinical psychologist

who immediately proscribed drinking, and enlisted family support to restrict purchase of alcohol. Within one week of forced sobriety, the survivor attempted to commit suicide and almost succeeded. Fortunately, his treatment provider enlisted additional help, and the new treatment plan called for specific assistance in coping with loss of job and friends, setting incremental goals for finding new friends, improving chances of sustaining reasonable employment through stepwise efforts, finding new, constructive activities to replace previous drinking, and education regarding drinking after brain injury. With simple encouragement and supportive counseling, drinking gradually reduced to infrequent.

Further, there are many instances where significant improvements in behavior and coping be achieved without much or any increase in awareness. Consider a recent example. A 47 year old New York executive who sustained a severe TBI almost 10 years ago had been treated at the best brain injury rehab centers in the country. Despite this, and despite having received very specific, targeted, aggressive treatment for unawareness, he continued to interact with others in an insulting, impatient, aggressive, too frequently dictatorial and sometimes abusive manner. Initial treatment efforts with this clinician involved trying to identify his red flag situations (change in schedule, having to wait for a request, etc.), teaching him to recognize these, and respond with a strategic self corrective procedure. After a lot of practice and consistent staff intervention to help make it become a habit, this strategy showed some benefit. However, it was still a multiple-step procedure with room for error and misuse, it did not work on bad days or when he was already angry or for new situations that did not exactly fit with the red flag list, and, even though it was an easy procedure, he had trouble remembering, especially when angry. Subsequently, a much simpler and easier approach was tried. This approach did not require awareness, did not even confront the survivor with any unpleasant realizations about personal weaknesses and instead allowed jokes about his New York style, which was present before his injury. With practice, this strategy was soon adopted as a habit that worked pretty effectively in most situations. The strategy, essentially is as follows: "Smile", explained as "Always smile when talking with others. The more important the communication, or the stronger you feel, the more you smile".

In this second example, a simple habit, smiling, was increased. Importantly, smiling tends to produce pleasant interactions. It is incompatible with insult, aggression and abuse. By its increased presence, it tended to produce noticeably mellowed responses that were more pleasant and less impatient, aggressive or dictatorial in style. As a result, this survivor received more satisfactory responses from others, which gave him less reason to be angry, and more reason to smile. As time passed, the positive responses from others and the more satisfactory responses to his requests rewarded his smiling, which tended to increase even more.

Finally, there are situations where limited cognitive abilities, including concrete thinking, may leave persons unable to adequately increase awareness of problem behaviors. A recent illustrative example is that of a fairly bright 40 year old severe TBI survivor who had some unusual residual "holes" in cognitive abilities. He was treated for three years at some of the better brain injury rehabilitation centers, for three years, and was transferred about without improvement in persistent childish behaviors and lack of self control. Attempts to increase awareness as a prerequisite step to modifying behavior failed, as did simple behavioral modification



programs aimed at rewarding more adaptive behaviors. At his fourth treatment program, it was finally determined, after several months, that an unusual and concrete thinking style, combined with his premorbid personality style, made increasing awareness a near impossibility. As a result, his treatment program was modified considerably. Rather than emphasizing awareness, which was apparently too complex, behavioral interpretations were simplified and paired with dichotomous "good" or "bad" labels. Explanations and attempts to appreciate his behavior beyond this were curtailed. A list of "bad" behaviors along with alternatively desirable "good" behaviors was devised and reviewed extensively. Rewards were assigned for increasing ratios of good to bad behaviors and staff provided encouragement and praise for increasing semblance of "good" behavior. In a short period of time, a pattern of consistent increases in appropriate behavior, and decreases in inappropriate behaviors, was observed.

In summary, although increasing awareness about deficits and maladaptive coping that results from brain injury is important, and although it is often a logical or necessary first step in modifying ineffective behavior, it is not always necessary. In fact, there are many situations where efforts to increase awareness before new coping strategies are acquired will cause increased emotional distress and catastrophic fears to the point that more harm than good can occur. Further, there are situations where it is infinitely easier to change a behavior than change awareness. Finally, there are situations where limited cognitive abilities, including concrete thinking, may leave persons unable to increase awareness of problem behaviors. In these situations, simplifying expectations and rewarding desirable behaviors, in order to build desirable habits, is a more effective alternative to increasing awareness.

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## ***What is EEG Biofeedback and can it help with Remediation of Post Concussive Symptoms?***

*Michael F. Martelli, PhD and Nathan D. Zasler, MD*

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Cognitive and emotional dysfunction, fatigue and disordered initiation and maintenance of sleep represent common impairments that frequently persist following traumatic brain injury (TBI). These have historically presented formidable challenges to the field of brain injury rehabilitation. The practice of clinical psychophysiology / applied biofeedback, which is predicated on the provision of enhanced feedback about pathophysiological processes in order to increase control over and restore normal function, has well documented clinical efficacy for musculoskeletal and vascular responses. A promising new application to EEG activity arose in the early 1970's with replicated demonstrations of efficacy in first suppressing seizures in animal and human subjects exposed to seizure inducing jet fuel, as later to poorly controlled and drug resistant epileptics. More recently, clinical report, case study evidence and initial group study data supporting the utility of EEG

biofeedback training for attentional disorders has appeared, with emerging evidence suggesting utility in treatment of learning disabilities and such emotional symptoms as depression and anxiety, as well as fatigue and sleep disorders. Finally, some preliminary evidence is emerging which also suggests that EEG Biofeedback training may be useful for simultaneously reducing frequently seen abnormalities in EEG patterns and remediating persistent cognitive, emotional, fatigue and sleep related problems following TBI. Initial findings by the authors from three subjects with persistent post concussive symptoms following TBI who underwent serial EEG Biofeedback training, Relaxation training and Cranial Electrical Stimulation (CES - microcurrent to the scalp, which produces increased alpha brain waves) offer support for moderate self rated symptom improvement, with findings of greater self reported symptom resolution following both EEG Biofeedback training and Cranial Electrical Stimulation versus a tape based relaxation procedure. These initial results are promising and further investigation is being conducted

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## ***How Do You Provide Helpful Feedback to A Patient When Their Doctor Decides They Have a Primary Diagnosis of Somatoform Disorder***

*(i.e., their physical symptoms have a psychological and not physical cause)?*

*Michael F. Martelli, PhD*

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First of all, it should be emphasized that feedback in the absence of ongoing treatment is probably never that useful with an entrenched defense system like somatoform disorder. Therefore, feedback is usually best offered as part of a multidisciplinary evaluation and treatment program, during which part of the treatment is education about psychological contributions, effect of stress on physical function, etc. Importantly, dualistic, "either / or" conceptualizations of functional or organic disorder by treating professionals are usually counterproductive to understanding somatoform disorders and effectively communicating and intervening with persons with this type of disorder. Simple feedback from a psychologist who suggests the need for psychologic based intervention will usually not be very effective. Also, directly attributing symptoms to psychological causes is never as fruitful as talking about stress making physical symptoms worse, and establishing an incremental

approach to appreciating psychological contributions to physical symptoms and incrementally reducing psychological influence.

As part of a multidisciplinary treatment program, or, alternately, as part of treatment by a fairly crafty psychologist treating individually (e.g., many good pain management specialists are skilled at making feedback about psychophysiological patterns very palatable) there are numerous methods, all with a common denominator of psychophysiological interaction, and typically including such things as biofeedback assisted demonstrations to show effect of stress on physiologic arousal, lots of psychoeducation about the Type I / Type II stress responses and physiologic channel arousal (including diagrams of heart rate, BP, muscle arousal, breathing, EEG desynchronization, glucose, etc., and explanations of Sabre Tooth Tigers), patient vulnerability (injury, genetic, environmental, etc) X sensitized physiologic system stress response, etc..... the key is often (but not always) an emphasis on not challenging veracity or legitimacy or organicity of physical symptoms, and instead, focusing on what controls them (i.e., not taking away the persons defenses until they have been given new coping skills, with the latter replacing the former).

Depending on the primary belief system and biases of the person with Somatoform Disorder, most are usually amenable to an ischemia psychophysiological explanation (e.g., pain increased arousal and sympathetic arousal in muscles, stress and vigilance (etc.) prevented parasympathetic rebound and a hyperaroused pattern, which has led to low grade ischemia and atrophy in muscles that prevents healing and contributes to reinjury during normal activities..... healing requires graduated increases in activity, combined with decreased anxiety and necessarily required associated decrease in somatic vigilance, etc. This author has written some useful patient information on this subject that can be accessed from the Villa Martelli Disability Resources Webpage at <http://go.to/MFMartelliPhD>, and clicking on the "USEFUL MODEL/ METHODOLOGY FOR REHABILITATION..." Link.

Some other useful reading might be John Sarno's stuff, the Physiatrist (Physical Medicine and Rehabilitation (PM&R) physician) from NYU who has published a few books and been featured on national news magazine shows. He is demonstrating 85% pain problem resolution in chronic low back pain patients via his Tension Myositis explanation (stress producing ischemia/ hypoxia in muscles) and reeducation program, which basically instructs persons to reduce stress, not let it get to their muscles, and just begin increasing activity without worrying about pain. Many of his patients appear to have somatoform disorders, and he is touting a very high success rate, and enlisting a ton of support from celebrities (e.g., producer of 20/20, Howard Stern, etc.) - although this probably suggests some nonspecific treatment factors associated with his personality, but certainly seems to indicate that he is capable of producing the two necessary ingredients for good psychotherapy outlined in Bergin and Garfield's old text - a credible rationale and a believable ritual.

Also, there was a special issue on Hysteria in Rehabilitation, edited by RW Teasell in *NeuroRehabilitation: An Interdisciplinary Journal*, Vol. 8, No 3 (May, 1997) that would probably be very helpful, and even included a nice model for treatment of conversion disorder (i.e., offering of a credible psychophysiological explanation for symptoms, combined with a double bind communication about expected recovery and time frame - something

like, this is the explanation of your symptoms and you will get in this amount of time, and if not, the rest is all psychological).

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## ***What is Post Traumatic Amnesia?***

***On 20/20 last night, Halle Berry's explanation for her hit and run is that she has total amnesia for the accident. She claims she drove herself home because she didn't realize she'd been in an accident until her fiance came home over an hour later and asked her why her head was bleeding and her car was dented. She alleged that she only had a vague idea that something may have happened at the intersection. The announcer said experts confirmed that this type of amnesia was common after a head injury. Is this possible, or is this just another trumped up story in an attempt to get out of trouble?***

**(General Question submitted by several patients, in several formats)**

*Answer by: Michael F. Martelli, PhD*

*Concussion Care Centre, Pinnacle Rehabilitation and Tree of Life*

*Virginia Commonwealth University / Medical College of Virginia*

Well, I guess like many persons, I tend to be skeptical when persons commit crimes (i.e., leave the scene in a hit and run accident) and then deny accountability or responsibility. And, I tend to be too fascinated by celebrities and by interesting clinical syndromes. So I understand your question. However, I do not know the facts. I can only cautiously speculate, in the interest of educating interested persons and clarifying information about Post Traumatic Amnesia (PTA).

Apparently, Ms. Berry is claiming that she was in Post Traumatic Amnesia after her accident. Post Traumatic Amnesia (PTA) is a period of disturbed episodic memory following trauma to the brain (ranging from blunt force hits to sudden acceleration and deceleration) often associated with confusion and disorientation. Following milder injury, people seem fairly normal but are unable to store any new memories. That is, they have amnesia for events that occur during the period of PTA (i.e., are not able to remember afterward), until they

recover their ability to store new memories. The length of PTA is one of the major indicators of severity of brain injury (i.e., mild  $\leq$  24hrs; moderate  $\leq$  1 wk; severe  $\geq$  1 wk). Clearly, if Halle Berry was in PTA after her accident (i.e., mild,  $<$  24hrs) she could have driven herself home. A person can leave the scene of an accident whether in PTA or not. The only difference is that, when in PTA, they will not remember it later. That means she could leave the scene of the accident while in PTA, and then not remember it when she cleared (i.e., continuous memory returned). Notably, PTA is not global dementia with deterioration of intellectual skills or personality - rather, it is a failure in ability to consolidate or store new memories.

Is it possible Ms. Berry did not know she was in an accident at the time of the accident? Although it is certainly possible, it should be noted that pain, blood, and so on are usually not mistaken, especially if a person is awake and alert enough to drive. Even if she was amnesic for the accident, the residual signs like pain and blood should have alerted her to the fact, and it is the typical human response to try to orient oneself and fill in any missing memory gaps in the case of such uncertainties. Even if she was amnesic for the accident immediately afterwards, and even if she was not able to retain new information, the typical response, based on observations of lots of people in PTA, would be that she would keep asking the obvious questions and looking for answers, despite learning and losing the answers repeatedly.

But, please understand, it would be extremely irresponsible for this professional, or any other, to make any assumptions on this limited data and speculation. Also, we are not always very good at predicting behavior, even with all of the facts.

By the way, my favorite clinical example regarding PTA is as follows. I had a patient who was in PTA in the hospital when his wife, and a female friend (with whom I later learned he was having a current affair), both visited. I subsequently found out that she was very fearful that he might die and so she chanced showing up - but he lived as it was only a mild. He, per the girlfriend (and my observation) acted completely appropriately, despite her great fear that because he could remember nothing for more than 20-30 seconds, he would slip up.

After he cleared, he was told the story. He nearly had a panic attack, thinking that if he could not remember the incident, he must have slipped up. He was sure he did. He didn't believe the girlfriend, or his neuropsychologist. He was afraid to see his wife, etc. But, he did not slip up, his wife knew nothing, and in treatment, we collaborated on a plan that involved cleaning up his act. His marriage survived, got stronger, etc. But he, to this day, suspects he slipped up and that others either missed it or cut him a break.

I also have another interesting example. In April, 1994, a neuropsychologist friend had a fairly serious car accident, sustained a complicated (mild bleed) mild TBI, had 11 hours of PTA, and was informed subsequently that not only was he *not* unconscious for 20 minutes like he had mistakenly assumed, but that at about one minute post accident, the first person who arrived fought with him to keep him from chasing after the other guy in the accident while he was muttering that the other guy hit him. He did not remember any of this, of course, and erroneously assumed he was unconscious. He had about 5 seconds of retrograde amnesia (RA) and,

again, 11 hours PTA. Now, if the guy who arrived had not been really big, and had he not had help, and had he not had a soothing voice, I suspect the neuropsychologist would have gone after the kid who hit him and who knows what he might have done that could have resulted in a legal charge. Clearly, I don't think any action he may have taken while in PTA would have reversed the PTA. (BTW, he returned to work, half days, after two days of hospitalization and two days at home, and returned to full time in three weeks)

Further, based on the Police report (which was apparently based on the non-head injured driver's exclusive report), the neuropsychologist assumed, (because of RA, PTA) that he was at fault. At the hearing, it was determined, based on pictures and deduction after the other driver's testimony did not fit the pictures, making the police report impossible) that the other driver was in fact at fault, as apparently the neuropsychologist thought while in PTA (but not afterwards), and charges were dropped. (No the neuropsychologist didn't sue the other driver, and even refused to follow advice of the neuropsychologist he was required to see in order to return to work, who encouraged him to sue and only wanted to talk about forensic issues, implications, etc. - what a profession!)

So, this story adds evidence to the fact of often observed phenomenon of people in PTA looking and acting pretty normally to noncritical observers, but not being able to recall any of the events afterwards.

Regarding any professionals offering opinions about this case after only hearing a few news accounts, I think this suggests preconceived ideas and making inferences on insufficient data. This is unwise for any professional. It should be noted that, just as in real clinical practice, practitioners have biases. Confirmatory bias (selective attention and interpretation) tends to justify biased opinions. This is probably much more prevalent in clinical decision making that most are willing to recognize. Halle's story is like a projective test, and people, on limited data, allow their biases to rule, and then use data to support their biased opinions.

Again, I am responding to this question only to help educate and clarify. I do not intend to offer any specific clinical opinions about Halle Berry.

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## ***Do Things Happen For a Reason?***

***Mike Martelli, Ph.D***

*[From Dr. Joel Finkelstein, a New York, Type A individual who successfully challenged numerous health obstacles and chronic conditions to*

*achieve a thriving chiropractic practice and school owner until surviving a severe TBI approximately 7 years ago]*

"I never believed that things happen randomly.

My philosophy my whole life was that things happen for a reason...my motto ' No guts no glory!!!'

...but I can find no reason for my injury

Which statement is true:

Do things happen for a reason, or do they not?" JBF

I think both statements are true. For most things, we have at least some control, and many things seem to happen to make us discover and use our control in situations where we thought we had none. However, some things seem to happen where there is no control and no meaning, and these are random. Perhaps it is the goal of life to discover meaning where there is meaning and control what can be controlled, but to accept that some things are random and outside our control, and discover that we still control our reactions to these things....and perhaps that is the real meaning of life.....going Slow is 10 times harder than going Fast, and accomplishing smaller things when we have more obstacles is also 10 times harder - so the motto and meaning is still true for "No guts, no glory!!!" because it takes 10 times the guts to go slow and accomplish baby step goals. In fact, it's an even tougher challenge with more obstacles and a completely new and different set of rules.

For most of your life, your meaning was "I can do it anyway." Now, your meaning is derived from learning to master the hardest challenge ever - the challenge where patience is required and slow movement is the key.