Reprint (2-2)2

PMS, EEG, and Photic Stimulation

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Two studies of premenstrual syndrome (PMS), EEG, and photic stimulation have recently been completed at the Royal Postgraduate Medical School, Hammersmith Hospital, London (UK) In a preliminary trial of photic stimulation as a treatment for PMS, seventeen women with PMS were treated with a take-home flashing light device for 15 to 20 minutes per day throughout their cycle. At the end of three months of treatment the median reduction in PMS symptoms for the 17 patients was 76%, and twelve of the 17 patients technically no longer had PMS. Separately, an EEG study of six women with PMS demonstrated that when they were premenstrual their EEGs showed more slow (delta) activity and slower P 300 evoked response than when they were mid-cycle. These results are discussed in the context of other known "slow brainwave" disorders, such as ADD and Minor Head Injury, and various theoretical explanations are proposed.*

Experimental Results

Preliminary Trial of Photic Stimulation for PMS

A preliminary trial of photic stimulation (flashing light therapy) as a treatment for PMS was recently completed by Duncan Anderson and his associates at the Royal Postgraduate Medical School, Hammersmith Hospital, London (UK). It was an open study of 17 women, all of whom had confirmed, severe, and long-standing PMS.

The flashing light device is similar to the device previously used for treatment of migraine (Anderson, 1989). It consists of a mask that covers the eyes, shutting out all light. Mounted in the mask are red LED lamps, one over each eye, which flash alternately in left and right eyes. The device is portable and designed to be used by the patient at home. The brightness of the light and the frequency of flashing are controlled by the patient, with ranges of approximately 10 to 45 mcd and 0.5 to 50 Hz respectively (one frequency cycle consisting of light in the left eye for half the cycle and then light in the right eye for half the cycle). The patients were instructed to start at the brightest setting and at the flicker-fusion point (around 30 Hz) and then adjust the brightness and frequency for best comfort. The patients were asked to use the device for 15 minutes per day, every day throughout their menstrual cycle. The patients recorded their symptoms daily on diary cards for two menstrual cycles before treatment, three cycles during treatment, and one cycle after treatment was stopped.

At the end of treatment the median reduction in PMS symptoms for the 17 patients was 76%. Twelve of the 17 patients technically no longer had PMS. Although the results of an open trial are subject to placebo effects, the results were so large and persistent that it is unlikely that placebo can fully explain them. The complete results of this trial are published in detail elsewhere (Anderson, Legg, & Ridout, 1997).

Study of EEG during PMS

A study of EEG during PMS was recently completed by Istra Toner and her associates at the Royal Postgraduate Medical School in London (Toner et al., 1995). Six women with self-reported PMS had 21 channel QEEG recordings and P300 evoked potentials measured during mid-cycle and premenstrually. Ages ranged from 30 to 43 years and all were taking no treatment for PMS.

A significant increase in delta activity during PMS was observed (p=0.043) along with a suggestive, but not necessarily significant, decrease in beta activity. This is consistent with previous reports of increased slow activity and decreased fast activity during PMS (Harding & Thompson, 1976; Lamb, Ulett, Masters, & Robinson, 1953).

P300 evoked potential was elicited using an odd-tone procedure with a frequent tone (1000 Hz) and an odd tone (2000 Hz) presented in the ratio 4:1 at a rate of 1 per second. Using global field power averaging, a significant increase in P300 latency during PMS was observed (p=0.027).

Discussion and Interpretation

PMS is a "Slow Brainwave" Disorder

It is proposed that there is a group of disorders characterized by excessive low frequency EEG activity (see Table 1). Based on the EEG results in Table 1, PMS is seen to belong to this group of disorders.

Table 1
Disorders Characterized by Excessive Low Frequency EEG Activity

Disorder	Abbr.	Reported Brainwave Characteristic
Attention Deficit Hyperactivity	ADHD	Excess theta/beta ratio (Lubar, 1991)
Chronic Fatigue Syndrome	CFS	Slow alpha, excess theta (Lindenfeld, Budzynski Andrasik, 1996)
Minor Head Injury	MHI	Diffuse slow activity (Ayers, 1987, Duffy Iyer & Surwillo, 1989, both also quoted in Byers, 1995)
Toxic Trauma	TT	Excess slow activity (Heuser, Mana & Alamos, 1994)
Premenstrual Syndrome	PMS	Excess delta, slow P300 (Toner, et. al., 1995)

Treatment of Slow Brainwave Disorders with Photic Stimulation

The preliminary trial reported above shows the efficacy of photic stimulation as a treatment for PMS. The treatment of ADHD with photic stimulation has been developed extensively by Harold Russell and his associates, using frequencies of 18 Hz and 10 Hz alternating for two minute periods, with demonstrable improvements in IQ scores and behavior (Russell & Carter, 1993). Many clinicians appear to be using photic stimulation informally for ADHD and the other slow brainwave disorders, with anecdotal reports of successful treatment but with very few published result

Treatment of Slow Brainwave Disorders with Neurofeedback

Many neurofeedback (EEG biofeedback) practitioners report successful treatment of some or all of these slow brainwave disorders. For example, the Lubars have for many years worked with children with ADHD, training them with beta frequency biofeedback, with excellent results (Lubar 1989; 1991), the Othmers have a long history of success with beta frequency biofeedback with patients with all of the disorders in this group (Othmer, 1994), and there are many other practitioners using this approach. Generally the feedback protocol involves positive reinforcement of beta frequencies and negative reinforcement of theta frequencies, though various other protocols are also used successfully.

The Brainwave Frequency Hypothesis

A reasonable explanation that is commonly proposed for the above experimental and clinical results is that the key to treating these disorders (all characterized by excessive slow brainwave activity) is to speed up the brainwave frequency. It is proposed that this can be accomplished either by training the patients to speed up their own brainwaves (beta-training neurofeedback) or by entraining the patients' brainwaves with a photic stimulation device flashing at beta frequencies.

Problems with the Brainwave Frequency Hypothesis

Unfortunately there is evidence, both from photic stimulation research and from neurofeedback training, that undermines this brainwave frequency hypothesis.

In the trial of PMS and photic stimulation reported above, the patients were free to adjust the frequency of the flashing light at will, between 0.5 Hz and 50 Hz. A frequency of around 30 Hz (high beta) was suggested, based on previous clinical results, but the patients were free to change this at any time in any session. Of those patients who achieved a greater than 50% reduction in symptoms, about half chose to operate the flashing light in the range of 5 to 10 Hz, i.e., theta-alpha frequency, not beta frequency.

Furthermore, some neurofeedback clinicians report equally good results when treating slow brainwave disorders with frequency protocols quite different from the beta enhancement/theta reduction protocol discussed above. In fact, Hoffman, Stockdale, Hicks, and Schwaninger (1995) list six different neurofeedback protocols (including alpha training) that have been used successfully for minor head injury.

Apparently "speeding up" the brainwaves with photic stimulation or neurofeedback at beta frequencies is not an adequate explanation for the successful treatment of these disorders.

The Cerebral Blood Flow Hypothesis

Many studies have shown that excessive slow brainwave activity is closely associated with hypoperfusion, i.e., insufficient cerebral blood flow. These studies have been collected and summarized by Toomin (1994). Looking at the individual "slow brainwave" disorders we see that

in each case there is some evidence for hypoperfusion (see Table 2).

Table 2
Excessive Slow Brainwave Activity and Hypoperfusion

Disorder	Evidence for Insufficient Cerebral Blood Flow
ADHD	Localized hypoperfusion demonstrated by Zametkin, et.al. (1990)
CFS	Hypoperfusion caused by hypotension, Bou-Holaigah, Rowe, Kan, and Calkins (1995)
MHI	Hypoperfusion demonstrated by Ichise, et. al. (1994)
TT	Localized hypoperfusion demonstrated by Heuser, et. al. (1994)
PMS	Preliminary SPECT tests show localized cerebral hypoperfusion, Amen (personal communication, 1996)

The causal relationship between slow brainwave activity and hypoperfusion is unclear. It is possible that reduced neuronal activity demands less blood flow or that reduced blood flow causes reduced neuronal activity or even that there is a "vicious circle" with neither component being able to initiate recovery.

However, it is known that cerebral blood flow is increased by photic stimulation (for example, Fox, Raichle, Mintum, & Dence' 1988; Sappey-Marinier et al., 1992). It is possible that this is the mechanism by which photic stimulation relieves PMS and other slow brainwave disorders.

The Role of Frequency

This is not to suggest that frequency is without significance. The training frequency in neurofeedback and the flash frequency in photic stimulation have been shown to encourage or entrain brainwaves of that frequency and this may have therapeutic value independent of blood flow considerations, by training the patient's brainwaves to operate at beneficial frequencies. And in some cases, for example alpha-theta neurofeedback, frequency is obviously critical, enabling the patient to access early emotional material of great therapeutic importance.

However, Othmer (personal communication, 1996) has suggested that a major component of neurofeedback training is the exercising and training of the mechanisms of arousal and attention, regardless of the frequency which is being trained. Exercising these mechanisms might be expected to result in an increase in neuronal activity and associated cerebral blood flow.

Summary and Conclusions

PMS and EEG

An EEG study of six women with PMS demonstrated that, when they were premenstrual, their EEGs showed more slow (delta) activity and slower P300 evoked response than when they were mid-cycle. It is concluded that PMS belongs to a group of disorders characterized by excessive

slow brainwave activity.

PMS and Photic Stimulation

In a preliminary trial of photic stimulation as a treatment for PMS, seventeen women with PMS treated themselves with a take-home flashing light device for 15 to 20 minutes per day throughout their cycle. Thirteen of the seventeen experienced a greater than 50% reduction in their symptoms. It is concluded that photic stimulation is an effective treatment for PMS.

Brainwave Frequency vs. Cerebral Blood Flow

Some of the other "slow brainwave" disorders are also being treated effectively with photic stimulation and all of the disorders are being successfully treated with beta frequency neurofeedback. This has led to the common hypothesis that these treatments are effective because they "speed up" the brainwaves, but in fact, at least with these "slow brainwave" disorders, the frequency used in the treatment, whether photic stimulation or neurofeedback, seems to be of secondary importance. It is suggested that increases in cerebral blood flow and associated increases in neuronal activity may be of equal or greater significance.

Photic Stimulation vs. Neurofeedback

If both neurofeedback and photic stimulation are effective in the treatment of these "slow brainwave" disorders, perhaps the best treatment may often be a combination of the two. Photic stimulation has the advantages of low cost and portability; it can be given to patients as "homework" between sessions and as pre-training for neurofeedback, to "teach" the brain the frequency that is to be trained. Neurofeedback develops the patient's sense of self-control and also has the unique advantage of localization, the ability to affect neuronal activity and brain blood flow specifically at a training site chosen for its relevance to the disorder, rather than just in the cortex in general. The combination of neurofeedback and photic stimulation seems particularly appropriate for ADHD, where the patient may initially have motivational difficulties with the neurofeedback training and need assistance from any other modality available.

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