# EEG Biofeedback Training for PMS

*by Susan F. Othmer and Siegfried Othmer, Ph.D.*

**Introduction**

PMS, or Pre-Menstrual Syndrome, exists. However, it is not a unitary condition. The symptomatology is highly variable among individuals. In fact, PMS is not a recognized disorder within the DSM-III-R. It can perhaps best be looked at as a condition of disregulation, one to which biofeedback should be highly applicable. Cyclic hormonal variations provide the stressor, to which a particular individual, with specific systemic vulnerabilities, succumbs in a variety of ways. It is not to be expected that the condition should manifest with a high degree of homogeneity.

PMS symptoms include a variety of physical and emotional symptoms associated with a specific phase of the menstrual cycle. Premenstrual symptoms are reported by at least 75% of menstruating women, but they may not be severe or debilitating. In an effort to clinically define the PMS population with significant clinical symptoms and behavioral impairment, the DSM-III-R established criteria for Late Luteal Phase Dysphoric Disorder (LLPDD). The criteria include emotional symptoms (irritability, mood swings, anxiety, and depression), less interest in the usual activities, fatigue, trouble concentrating, change in sleep or appetite, and various physical symptoms. These symptoms must be correlated with the premenstrual phase only and result in serious impairment of relationships or interference with activities.

Many of the PMS symptoms are characteristic of depression as well, and indeed PMS may be seen as a depressive syndrome. Antidepressant and antianxiety medications often provide relief from some emotional PMS symptoms. Medical management must be maintained continuously, and generally involves some undesirable side effects. The lack of successful medical management again augurs well for a biofeedback intervention. The fundamental issue is "disregulation", for which the remedy is "re-regulation", rather than the more unilateral intervention implied by anti-depressants or anxiolytics.

Intervention with EEG biofeedback has been found clinically to be very helpful to individuals suffering from both physical and emotional PMS symptoms. Most of these individuals were referred for specific symptoms which troubled them, rather than for "PMS". However, these symptoms were related to the menstrual cycle in the classical temporal PMS pattern. I.e., they appeared in the late luteal phase of the ovulation cycle. Others reported PMS symptoms unrelated

to their referral. Regardless of whether a person was referred for PMS or for specific symptoms, the training of the person revolved to a large degree around the constellation of symptoms associated with PMS. Our own criteria of training outcome usually included assessment of remediation of this constellation of symptoms.

At its best, biofeedback training addresses itself to the whole individual, not to a particular symptom. And also at its best, biofeedback addresses itself to the condition of "disregulation" globally, rather than with respect to a particular symptom. The fact that EEG biofeedback simultaneously addresses a broad spectrum of symptoms successfully is powerful support for the view that training the brain directly addresses conditions of disregulation in their largest generality. Conversely, assessment of progress with EEG training requires survey of all of the client's relevant symptomatology. Under conditions of cortical "disregulation" in our client population, almost irrespective of etiology, PMS symptoms were usually reported at intake. Conversely, when such "disregulation" was successfully remediated, this would usually manifest in the PMS symptoms as well. The premenstrual syndrome can almost be taken as a paradigm for "cortical disregulation" in menstruating women.

From this global perspective on PMS, it follows that the appropriate training protocol relates to the underlying deficiency in control mechanisms rather than to the specific symptoms which manifest. Hence, the constellation of symptoms, as well as the results of testing, are used to elicit whether the person tends toward under-or over-arousal, and to the presence of various instabilities, or volatilities in behavior. Then a priority is placed on which protocol is to be used first. This priority certainly takes into account the predominant symptoms which brought the client to us in the first place. However, that is by no means the only, or even the primary, concern.

We have stated above that PMS symptoms are to be seen in the context of underarousal. Nevertheless, symptoms we normally regard as related to overarousal, such as migraine headaches or anxiety, may be observed as well. This is true not only for PMS, but for underarousal in general. Both hyperactivity and hypoactivity arise out of the ground of underarousal. Anxiety is frequently seen in the context of depression. By increasing cortical activation via EEG training, we increase cortical control and physiological self-regulation which also addresses overarousal conditions.

**A Discussion of Mechanisms**

To date, biofeedback in general has been largely oriented toward conditions of over-arousal, i.e. anxiety, hypervigilance, adverse stress response, etc. Biofeedback has had little to offer for conditions of underarousal such as depression. EEG biofeedback is the singular exception. EEG biofeedback has been used most extensively to date with epilepsy and with Attention Deficit Disorder. The latter is fundamentally a condition of underarousal, and remediation with EEG biofeedback is understood to be facilitated by restoration of autonomous regulation of arousal. With epilepsy the matter is less immediately clear. However, epilepsy can be subsumed in the larger category of traumatic brain injury, inasmuch as at least forty percent of epilepsy cases are traceable to known instances of traumatic brain injury. And depression is an almost universal concomitant of traumatic brain injury. It may also be noted that behavioral management techniques for epilepsy revolve almost completely around overt management of arousal level by the individual. Hence, efficacy of the EEG training for epilepsy may also be seen in terms of "re-regulation of arousal", as well as increasing the stability of a hyperexcitable cortex.

It is likely that both elements are present in the remediation of PMS by EEG biofeedback: normalization of control of arousal, and broadening the range of cortical stability. The former is applicable to the emotional symptoms and to pain. The latter is applicable specifically to such symptoms as migraine headaches, as well as perhaps to the more extreme behaviors sometimes observed with sudden onset, such as rages and paranoias.

**EEG Training Protocols**

EEG biofeedback training fits a learning model. The brain is challenged in a particular way, and over time, it acquires the skill to meet the challenge. As it does so, the threshold of success is adjusted to maintain the challenge. The process is continued until symptomatic relief is obtained. The training goals relate to narrow frequency bands within the lower end of the beta frequency range, 12-18 Hz. Most commonly, the frequency bands trained are 12-15 Hz (SMR band), or the 15-18 Hz band (which we call "beta"). For some cases, a filter intermediate to these is found to be appropriate: 13.5-16.5 Hz (which we call "15 Hz"). Accompanying the training of activity in these narrow bands is an inhibit function which is used to discourage large amplitude activity in the low frequency bands ("theta," or 4-7 Hz), and in the higher beta range (22-30 Hz, in our implementation). The lower frequency activity is a concomitant of underarousal or a deficiency in cortical stability. Excessive high-frequency activity is a concomitant of anxiety, alcoholism, drug addiction, hypoglycemia, or mania. Excessive EMG activity also is observable in this band.

The training in the low beta range is deemed to be successful because the mechanisms by which activation is modulated, and by which the intrinsically excitable cortex is stabilized, is operative in this frequency range. This thalamocortical regulatory mechanism, which also governs the sleep/wake cycle, has recently been elucidated by M. Steriade. Confirmation of this mechanism is found from the fact that training slightly different frequency bands within the low beta region achieves quite different results in terms of physiological arousal.

The intake interview, baseline EEG, family history questionnaire, and results of testing are all used to specify the appropriate EEG training protocol at the outset. Testing in connection with PMS consists of the T.O.V.A., a computerized continuous performance test. This test helps to clarify where the individual is functioning with respect to the variable of arousal (underarousal/overarousal). This information is then used to specify the appropriate training frequency band. The clinical choices to be made consist not only of selection of the appropriate reward frequency band, but also the selection of the appropriate cortical site.

Nearly all of the training for PMS with the SMR/beta protocols is performed at the standard sites C3, Cz, or C4. In fact, the vast majority is done with beta training (15-18 Hz) at C3. The results of the training observed from session to session will then confirm the initial site and band-pass filter selection, or indicate the need for a change to a different training configuration. Since the training is not necessarily being conducted during a symptomatic period, the cues may be subtle. The client is questioned in detail about changes in sleep patterns, pain symptoms, mental functioning, etc. Changes in PMS symptoms are usually seen with the very next period after onset of the training. Any training sessions conducted during the symptomatic period are particularly illuminating with respect to the clinical choice of protocol.

**Clinical Outcomes**

The very nature of biofeedback makes it difficult to establish good criteria for clinical outcomes by which results can be compared among practitioners and among a variety of approaches. First of all, the criteria of success should only be applied to those who have in fact completed a significant number of training sessions. In our experience, the number of sessions required ranges from twenty to forty. On the other hand, those who are not progressing with the training for one reason or another are not usually encouraged to continue to that point. Hence, the group to which criteria of success may be properly applied has already become "selected for success" to some degree. Given that caveat, it is our experience that on the order of 90% of those who stay committed to

the program do in fact benefit significantly in terms of symptom relief, and that these benefits are retained for the long term (months to years).

If one asks the question about what fraction of those who enter the training program achieve a satisfactory outcome, the success rate is lower. We observe that adults are much more difficult to retain in the program than children, and this is true regardless of the diagnosis, be it epilepsy, traumatic brain injury, depression, alcoholism, or PMS. Adults are much more likely to see to the needs of their children than to their own needs. We observe this in families who are also bringing their children. Although a parent may bring her child faithfully, she will be less likely to follow through on training for herself. We observe that this has little to do with the intrinsic efficacy of biofeedback. In any case, the success rate by this criterion is approximately 70 percent.

The symptom relief experienced by those who successfully complete the program includes all of the emotional symptoms associated with PMS, and the physical symptoms, including migraines. Some reach the point where they are essentially symptom-free, and do not experience the usual prior notice of the onset of menses. Favorable experience has also been observed with dysmenorrhea, and with such symptoms as excessive bleeding. The data on such phenomena are isolated, and not yet subject to statistical treatment.

## References

Diagnostic and Statistical Manual of Mental Disorders (DSM III-R). Published by the American Psychiatric Association, 1987, pp.367-369.

Severino, S.K., & Molina, M.L. (1989). Premenstrual Syndrome, A Clinician's Guide. New York: The Guilford Press.

Rubinow, D.R. (1992). The Premenstrual Syndrome: New Views. *The Journal of the American Medical Association*, 268, 1908-1912.

Steriade, M., et al. (1993). Thalamocortical Oscillations in the Sleeping and Aroused Brain. Science, 262, 679-685.