EFFECT OF SAHAJA YOGA PRACTICE ON STRESS MANAGEMENT IN PATIENTS OF EPILEPSY* 


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Abstract: An attempt was made to evaluate the effect of Sahaja yoga meditation in stress management in patients of epilepsy. The study was carried out on 32 patients of epilepsy who were randomly divided into 3 groups: group I subjects practised Sahaja yoga meditation for 6 months, group II subjects practised postural exercises mimicking Sahaja yoga and group III served as the epileptic control group. Galvanic skin resistance (GSR), blood lactate and urinary vinyl mandelic acid (U-YMA) were recorded at 0, 3 and 6 months. There were significant changes at 3 & 6 months as compared to 0 month values in GSR, blood lactate and U-VMA levels in group I subjects, but not in group II and group III subjects. The results indicate that reduction in stress following Sahaja yoga practice may be responsible for clinical improvement which had been earlier reported in patients who practised Sahaja yoga.

Key words: epilepsy management stress yoga

INTRODUCTION

Alternate methods of seizure control have recently evoked considerable interest as epilepsy is now being looked upon as a neurophysiological or a behavioural neurological problem rather than purely a neurological one. Secondly drug therapy often does not provide complete seizure control. Techniques like biofeedback (1) relaxation (2) have been employed for seizure control but well planned studies are few. Some studies have included small number of subjects (3) or are single subject designs (4) making the validity of statistical analysis questionable.

Some of the behavioural techniques do give encouraging results but the mechanisms by which they benefit are not clear. It is suggested that reduction in stress may be an important factor in seizure reduction (5). Stress may precipitate seizures in a predisposed person; daily stress is reported to be a significant predictor of seizure activity (6).

Yoga an ancient Indian culture and way of life, which gives the practitioner a healthy body and sound mind is known to alleviate stress and produce relaxation. Many branches of yoga have been described (7), such as 'Hatha' yoga, 'Karma' yoga, 'Bhakti' yoga and others. Yoga includes postural exercises ('asanas'), breath control ('Pranayama') and at a higher state aims to 'yoke' or 'join' the individual soul to the universal soul. Sahaja yoga is reported to help in the prevention of stress disorders (8). The technique is simple, it does not involve adoption of any complicated postures and can be easily practised by any person.

We have found Sahaja yoga practice reduced seizure frequency and seizure duration (9). In the present study, we investigated the effect of Sahaja yoga practice on Galvanic Skin Resistance (GSR). Blood lactate of levels, parameters of levels of tension or relaxation in order to study whether reduction in the level of stress is responsible for clinical improvement in epileptics following Sahaja yoga practice.

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METHODS

Subjects:

Thirty female and 2 male epileptics in the age group of 15-35 years, mean (SE) age 22.5 (1.18) years were studied. Criteria for inclusion in the study were (i) patients clinically diagnosed as having primary idiopathic epilepsy (clinical seizure; CT scans or MRI did not reveal any abnormality), (ii) greater than four seizures in 3 months and (iii) awareness of every seizure episode by self or family member. The subjects were epileptics attending the Neurology Clinic, Lady Hardinge Medical College and Smt. S.K. Hospital. An informed, written consent was obtained from each subject prior to enrolment in the study. The study was approved by the Ethical Committee of the Institution. The subjects were categorized in 3 groups: Group I, n=10, consisted of epileptics who practised Sahaja yoga for 6 months; Group II, n=10, consisted of epileptics who practised mimicking exercises for 6 months; Group III, n=12, were epileptics who did not practise Sahaja yoga or mimicking but served as controls.

The subject characteristics and their relevant clinical data are shown in Table I. The subjects in group I, II and III were comparable for age and duration of illness. Statistical analysis using one-way analysis of variance (ANOVA) revealed no statistically significant differences between the three groups of subjects at the commencement of the study.

Practice programmes:

The patients in group I practised Sahaja yoga twice daily for 20-30 minutes under the guidance of a trained 'Sahaja Yogi' or instructor. They reported to the Department of Physiology on all working days for the first month and thereafter twice a week for the remaining period. On the day of the weekly Neurology Clinic, the patients practised Sahaja yoga in a room in the Medical Out Patient Department.

The subjects practised meditation in a quiet, well illuminated room sitting in a comfortable posture. The technique used for Sahaja yoga practice was as described (10). A typical session of meditation consisted of questions and assertions by the subject. Thereafter the subject practised silent meditation. If a thought came to the mind, she/he was instructed to simply witness the thought but not to flow deeper into it.

Gradually, with practice the subjects reported to be in a state of "thoughtless awareness". Sahaja yoga was practised at bedtime by sitting in silent meditation with the feet dipped in warm saline water.

Group II subjects who practised mimicking exercises were provided with the same environment and attention as group I subjects. However, actual meditation was not practised by these subjects. The subjects were instructed to simply place their hand at different positions as during Sahaja yoga practice and thereafter sit quietly with their eyes closed.

TABLE I: Clinical characteristics of subjects.

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>Age (yrs) (Mean ± SE)</th>
<th>Gender</th>
<th>Level of Education</th>
<th>Type of Seizures</th>
<th>Duration of illness (yrs) (Mean ± SE)</th>
<th>AEDs</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>10</td>
<td>24.6 ± 2.1</td>
<td>Females</td>
<td>Primary School</td>
<td>GTC</td>
<td>7.3 ± 1.1</td>
<td>PHT + PB 5</td>
</tr>
<tr>
<td>II</td>
<td>10</td>
<td>23.7 ± 2.5</td>
<td>Males</td>
<td>Secondary School</td>
<td>CPS</td>
<td>5.6 ± 0.9</td>
<td>PHT + PB 3</td>
</tr>
<tr>
<td>III</td>
<td>12</td>
<td>19.7 ± 1.4</td>
<td>Males</td>
<td>Senior School</td>
<td>SPS/GTC</td>
<td>4.2 ± 0.7</td>
<td>CBZ + PB 2</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Graduate</td>
<td></td>
<td></td>
<td>Primadone 1</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>+CBZ + PB</td>
</tr>
</tbody>
</table>

GTS - Generalised tonic seizures, SPS - Simple partial seizures.
CPS - Complex partial seizures
AEDs - Anti-epileptic drugs, PHT - Phenytoin, PB - Phenobarbital, CBZ - Carbamazepine
VPA - Valproate
GSR

GSR was recorded using a Biotrainer (model GBF-102R Japan). The subjects were seated in a comfortable posture. The palmar surface of the index and middle fingers were thoroughly cleaned with spirit and the electrodes were fixed on them with electrolyte paste. The subject was connected to the Biotrainer. The gain of the instrument was adjusted for the degrees of variation of colour and sound. Absolute values of GSR are noted when there is orange light on the screen or when the indicator on the recorder is pointing to '0'(%) (unit 100 kohms). The subjects were allowed 10-15 minutes for getting used to the instrument. Thereafter GSR readings were noted at 1 min intervals for 5 minutes.

Blood lactate:

Blood lactate was estimated using YSI model 23-L Lactate Analyser. The L-lactate in the injected sample is oxidized by the catalytic action of L-lactate oxidase and flavin adenine dinucleotide (FAD) producing hydrogen peroxide and pyruvate.

\[ \text{L-lactate} + \text{O}_2 \rightarrow \text{H}_2\text{O}_2 + \text{Pyruvate} \]  

The \( \text{H}_2\text{O}_2 \) produced in reaction 1 comes in contact with a platinum anode held at a potential of +70 volts with respect to a silver reference cathode. Reaction 2 takes place at the platinum anode, yielding a current which is linearly proportional to the concentration of lactate in the sample.

\[ \text{H}_2\text{O}_2 \rightarrow 2\text{H} + \text{O}_2 + 2\text{e}^- \]  

The circuit is completed by the silver reference cathode as shown in reaction 3

\[ 2\text{AgCl} + 2\text{e}^- \rightarrow 2\text{AgCl}^- \]  

At constant chloride concentration the potential of this reaction is practically independent of current.

Samples were drawn from the fingertip after discarding the first 2-3 drops. The instrument was set to run, rinsed with water, injected with 5 mmol/L standard solution and calibration was adjusted. Thereafter the sample 100-200 uL was injected using a syringe and the reading was noted.

U-VMA

U-VMA was estimated by the method of Pisano et al (11). The method involves the conversion of VMA to vanillin which is estimated spectrophotometrically, VMA and other phenolic acids are extracted from acidified urine with ethyl acetate and reextracted into aqueous potassium carbonate solution. VMA is oxidized to vanillin by addition of sodium metaperiodate to the carbonate extract. Vanillin is then separated from contaminating phenolic acids by selective extraction to toluene and back extracted to carbonate. The vanillin concentration is determined at 360 um.

The subjects were instructed to collect their 24 hour urine in a dark brown bottle containing 10 ml HCl, 6 mol/L. The addition to HCl kept the pH of the urine at or below 2. The total volume of the urine was measured. Aliquots were stored at or below 4°C in a refrigerator. For further details the reader is referred to Chattoraji and watts (2).

Statistical analysis:

Comparisons of 0, 3 and 6 months values in each group were made by 2 way Analysis of variance. Significant F values were further evaluated with calculation of least significant difference (lsd) in order to check whether the differences were significant at 3 and/or 6 months. Statistical significance was taken at the level of probability (p) less than 0.05.

RESULTS

Table II shows the GSR values in group I, II and III subjects. GSR was significantly increased at 3 months (\( P<0.01, \text{lsd } 0.01 = 215 \)) and at 6 months (\( P<0.001, \text{lsd } 0.001 = 29 \)) as compared to 0 month in group I subjects. There were no significant changes at 3 and 6 months in group II and III subjects.

| TABLE II : Galvanic Skin Resistance in group I, II and III subjects. |
|--------------------------|--------------------------|--------------------------|
|                          | Group I (n=10) | Group II (n=10) | Group III (n=12) |
| GSR (x 100 Kohms)        | Mean           | SE            | Mean           |
| 0 mo                     | 3.9            | 6.3           | 6.4            |
| 3 mo                     | 6.1**          | 8.0           | 4.7            |
| 6 mo                     | 7.2***         | 7.8           | 5.1            |

\[ F=9.9, \text{df}=2.18 \]

\( **P<0.01, ***P<0.001 \) vs 0 mo values.
Table III shows the blood lactate levels in group I, II and III subjects. Blood lactate levels decreased significantly in group I subjects at 3 months (P<0.05, lsd \(0.05=0.07\)) and at 6 months (P<0.01, lsd \(0.01=0.10\)) as compared to the 0 months value. There were no significant changes at 3 and 6 months in group II and III subjects.

**TABLE III: Blood lactate levels in group I, II and III subjects.**

<table>
<thead>
<tr>
<th></th>
<th>Group I (n=7)</th>
<th>Group II (n=6)</th>
<th>Group III (n=7)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>0 mo</strong></td>
<td>Mean 0.697</td>
<td>0.703</td>
<td>0.660</td>
</tr>
<tr>
<td></td>
<td>SE 0.032</td>
<td>0.034</td>
<td>0.032</td>
</tr>
<tr>
<td><strong>3 mo</strong></td>
<td>Mean 0.609*</td>
<td>0.647</td>
<td>0.676</td>
</tr>
<tr>
<td></td>
<td>SE 0.041</td>
<td>0.025</td>
<td>0.056</td>
</tr>
<tr>
<td><strong>6 mo</strong></td>
<td>Mean 0.571**</td>
<td>0.630</td>
<td>0.676</td>
</tr>
<tr>
<td></td>
<td>SE 0.029</td>
<td>0.036</td>
<td>0.024</td>
</tr>
</tbody>
</table>

*P<0.05 **P<0.01 vs 0 mo values.

Table IV shows the urinary VMA levels in the 3 groups. Group I had significantly lower levels at 3 and 6 months (P<0.01, lsd \(0.01=0.53\)). No significant changes were obtained in group II and III subjects.

**TABLE IV: Urinary vinyl mandelic acid (VMA) levels in group I, II and III subjects.**

<table>
<thead>
<tr>
<th></th>
<th>Group I (n=7)</th>
<th>Group II (n=6)</th>
<th>Group III (n=6)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>0 mo</strong></td>
<td>Mean 5.83</td>
<td>5.83</td>
<td>5.50</td>
</tr>
<tr>
<td></td>
<td>SE 0.32</td>
<td>0.30</td>
<td>0.25</td>
</tr>
<tr>
<td><strong>3 mo</strong></td>
<td>Mean 5.12**</td>
<td>5.84</td>
<td>5.28</td>
</tr>
<tr>
<td></td>
<td>SE 0.30</td>
<td>0.54</td>
<td>0.24</td>
</tr>
<tr>
<td><strong>6 mo</strong></td>
<td>Mean 5.08**</td>
<td>5.34</td>
<td>5.57</td>
</tr>
<tr>
<td></td>
<td>SE 0.24</td>
<td>0.51</td>
<td>0.38</td>
</tr>
</tbody>
</table>

**P<0.01 vs 0 mo values.

**DISCUSSION**

The reduction in stress appears to contribute significantly to seizure reduction following Sahaja Yoga practice. In order to assess the ‘placebo’ effect, the study design included an additional control group viz. subjects practising mimicking exercises. Since no significant changes were obtained in this group, the placebo factor could be ruled out. The sample size (n=32) is also the largest reported so far. It is important that the anti epileptic medication remain unaltered for the study duration we attempted. The same two patients in group I had reduced medication and 1 patient in group II increased medication and 2 patients in group III also increased medication.

Sahaja Yoga practice led to significant changes in all the parameters indicating reduction in the level of stress. The increase in GSR indicates decreased sympathetic activity. The change in GSR was 55.5% at 3 months and 83.4% at 6 months compared to 0 month in group I subjects. Group II subjects also showed a rise of 26.9% at 3 months and 23.1% at 6 months, which was not statistically significant. No trend was seen in group III subjects. Values of GSR obtained in the present study are comparable to those reported in literature (13). The finding of an increased GSR is in conformity with that following Shaja yoga practice (8) and following TM practice (14).

Changes in skin resistance are a measure of sweat gland activity and are related to the sympathetic nervous system. Normally sweat glands offer a low resistance to the current passing through the electrodes. When glands are inactive its membrane has higher resistance than when excited. Sweat glands being modulated by the hypothalamus, reticular formation and cerebral cortex reflect the activity of different areas of the central nervous system related to the individual's arousal and awareness. Hence the changes in activity of the sweat glands are closely related to one's level of tension or relaxation (15).

Blood lactate levels decreased by 12.6% at 3 months and 18.1% at 6 months as compared to 0 month values in group I subjects. No significant changes were seen in group II and III subjects. Urinary VMA levels were also significantly reduced following Sahaja yoga practice. The decrease was 12.2% at 3 months and 12.9% at 6 months. The other 2 groups did not show any trend. These results are in agreement with previous studies on Yoga asanas and TM (16, 17).

The decrease in blood lactate and urinary VMA...
levels indicate a more relaxed state. It is reported that blood lactate levels increase in anxiety states and that infusion of lactate ions into patients of anxiety neurosis produces symptoms of anxiety (18). Catecholamine excretion is reported to be increased during distress and elevated levels in the urine reflect a corresponding enhanced release of catecholamines from the sympathoadrenomedullary system (19). The metabolism of catecholamines is influenced by social stress (19). The metabolism of catecholamines is influenced by social stress (20), novelty (21) and emotional stress (22, 23).

The role of stress in epileptics is complex. A moderate degree of anxiety may keep the patient alert and reduce the likelihood of seizure (24). Stress disorders are common in epileptics and more common than in the general population (25). Epilepsy especially when starting early in life impairs the learning of coping responses to stress. Secondly anti epileptic medication also impairs learning and interferes with normal responses. Hence, an epileptic suffers not only from normal stresses that a chronic illness imposes but is also handicapped in terms of responding to those stresses by the illness itself and by its treatment (26).

It is important to consider which type of patients benefit more from such treatments. We found that patients with psychosocial problems or those who were more anxious were more responsive to Sahaja yoga practice. Subsequently these patients were much better adjusted in their family and society. One patient who frequently suffered from headaches earlier improved. Another subject earlier unemployed was gainfully self-employed.

The mechanism by which Sahaja yoga benefits cannot be deciphered from the present study. It is possible that 'conditioning' of the limbic system leads to clinical improvement. It is well known that the limbic system regulates the homeostatic mechanisms through the autonomic nervous system outflows and regulates endocrine secretions. Emotional or mental stress acting via these mechanism may disturb the homeostasis, which when restored may lead to overall clinical improvement.

ACKNOWLEDGEMENTS

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REFERENCES


