Vulvar Vestibulitis Syndrome with Electromyographic Biofeedback of Pelvic Floor Musculature

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Thirty-three women diagnosed as suffering from vulvar vestibulitis syndrome, marked by a significant history of long-term moderate to severe chronic introital dyspareunia and tenderness of the vulvar vestibule, were selected for treatment.

Abstract

Thirty-three women diagnosed as suffering from vulvar vestibulitis syndrome, marked by a significant history of long-term moderate to severe chronic introital dyspareunia and tenderness of the vulvar vestibule, were selected for treatment. Patients were given a computerized electromyographic evaluation of the pelvic floor muscles and were then provided with portable electromyographic biofeedback instrumentation and instructions on the conduct of daily, at-home, biofeedback-assisted pelvic floor muscle rehabilitation exercises. They received intermittent evaluations of pelvic floor muscles to ensure compliance and monitor their progress and symptom changes. The results show that after an average of 16 weeks of practice, pelvic floor muscle contractions increased 95.4%, resting tension levels decreased 68%, and the instability of the muscle at rest decreased by 62%. Subjective reports of pain decreased an average of 83%. Twenty-eight patients had abstained from intercourse for an average of 13 months. Twenty-two of these 28 patients resumed intercourse by the end of the treatment period. Six month follow-up indicated maintenance of therapeutic benefits. (J Reprod Med 1995;40:283-290)

Keywords: vulvar diseases, electromyography, vulvar vestibulitis syndrome.

Notes

Introduction

Patients with vulvar vestibulitis syndrome constitute an identifiable subset of patients with vulvodynia. Aside from "focal vestibulitis vulvae," 1 this condition has also been referred to as "erythematous vulvitis en plaque," 2 "hyperesthesia of the vulva," 3 "minor vestibular adenitis," 4 "burning vulvar syndrome," 5 "focal vulvitis" 6 and "vulvar vestibulitis syndrome." 7 A task force set up by the Tenth World Congress of the International Society for the Study of Vulvar Disease 8 replaced the term burning vulvar syndrome with the term vulvodynia and defined the condition clinically. Vulvar vestibulitis is typically marked by a history of intermittent, then continuous discomfort in the vulva and introital dyspareunia. Examination reveals significant focal tenderness of the vestibule on contact or pressure in the absence of vulvar lesions or identifiable vulvovaginal infection. Macular erythematous patches may be noted along the hymeneal sulcus, at the openings of the major and minor vestibular glands. The paraurethral glands are also frequently involved. The erythema may vary in intensity and may not be apparent in all cases or at all times. Histology may reveal a chronic inflammatory cell reaction surrounding the vestibular glands.

The typical patient is white, upper middle class, sexually experienced and in her 20s or 30s. The etiology is unknown, and although attempts have been made to explain the mechanism of pain, 9 all therapeutic approaches utilized to date have been empirical. Most are based on their success in treating other conditions presumed to be of similar origin and are aimed at reducing the secondary symptomatology, with varying degrees of success. Studies have not been able to demonstrate etiologic involvement of any of the more common infections, but patient reports indicate that the onset is frequently associated with vulvovaginal infection or trauma and exacerbated by application of topical medications. Tovell and Young l suggested that the high incidence of reports of vaginitis preceding the onset of this condition (8510) may indicate misdiagnosis; however, the possibility of
misdiagnosis is difficult to differentiate from infection as the etiologic agent or from the possibility that the topical agents used in treatment for these conditions may have elicited a hypersensitivity response. Vulvar vestibulitis may well be multifactorial. Local inflammation or infection with bacteria, Candida and human papillomavirus (HPV) have been implicated in some cases. However, a study attempting to associate vulvodynia with HPV proved to be unproductive. Association versus causation is difficult, if not impossible, to prove in retrospect. In the acute setting, the major differential diagnoses to be ruled out include herpes simplex virus (HSV), HPV, squamous papillomatosis, vulvovaginitis, traumatic injury, and vulvar dystrophy or dysplasia, all of which have been associated with burning pain and/or acute tenderness in this area.

Spontaneous remission may occur but is uncommon, and generally those cases of sufficient duration to result in referral to our service are past the usual duration for spontaneous resolution. Although surgical resection of the affected tender vestibular area has been the "gold standard" of therapy, it is not uniformly successful, and success is variously defined as degree of improvement and postoperative function. Patients with an underlying component of burning pain or those with associated urinary symptoms have had even less success with surgical therapy since it does not address these symptoms. Medical therapies include antihistamines, tricyclic antidepressants, compresses, topical anesthetics and lubricants. Topical remedies do not cure the condition, but soaks of Burow's, Aveeno-oilated or aqueous lidocaine solution have been reported to produce temporary and partial relief of symptoms in some patients. Steroids have been tried for their anti-inflammatory effect. Peckham reported that neither topical nor systemic steroids were effectual. Laser surgery is not recommended due to prolonged and painful healing time. Local injections of alpha interferon (r-alpha IFN) in selected patients have not proven entirely successful, but immunotherapy may be beneficial in some cases. Dietary restrictions and reduction of irritants in the urine or in topical agents are reported as being successful in some cases.

No reports are found in the literature regarding any relationship between pelvic floor muscles and focal vestibulitis vulvae. Our rationale for studying pelvic floor muscles is that patients with focal vestibulitis often show hyperirritability of pelvic floor muscles, which, when compressed are locally tender. Pelvic floor musculature is not responsive to general states of muscle tension; standard progressive muscle relaxation techniques are not effective. Travell reported that this myofascial hyperirritability can be activated by localized tissue disturbance. In the case of vulvar vestibulitis syndrome, we believe that cutaneous vulvar disturbances destabilize pelvic floor muscles. (Note that the principal motor and sensory nerve fibers to both the vulva and the pubococcygeus are branches of the pudendal nerve plexus containing fibers from S3 and 4.) Travell and Simons reported that such muscle disturbances, reflected in discoordination of the electromyography (EMG), are prone to develop in muscles that lie within the pain reference zone of disturbed tissue and in turn reflex back through a dorsal root spinal cord mechanism to perpetuate tissue disturbance via its effects on local autonomically (sympathetic) mediated activity, including vascular changes and histamine release. To the extent that destabilization of pelvic floor muscles is a factor in perpetuating the vulvar skin disturbance and accompanying pain described above, restabilization of pelvic floor muscles should allow this condition to resolve. Since Travell and Simons reported that this type of muscle disturbance is reflected in EMG records, EMGs were used to objectively measure that status of the pelvic floor muscles in the present study.

Patient Selection

Patients for this study were drawn from the Cutaneous-Vulvar Service of St. Luke's Roosevelt Hospital Center, which currently manages vulvar vestibulitis as follows. The patient is examined in detail to exclude any contributing bacterial vaginosis or vaginitis or concurrent dermatologic problem. A gynecologic examination and inspection of the glabrous skin are both necessary. Initial therapy, depending upon the predominant symptomatology, commences with antihistamines: terfenadine, 60 mg twice daily, or hydroxyzine, 25 mg, h.s.; or tricyclic antidepressants, amitriptyline or nortryptaline, 10-25 mg, h.s. Dosages are evaluated in two to four weeks and are adjusted accordingly. The patient is also prescribed a colloidal oatmeal (Aveeno-oilated) solution to be used in dilution as a cold compress two or three times a day for 10 minute periods. Patient education and psychological support are also provided. Even with successful therapy, the symptoms are slow to resolve, averaging six weeks to several months until relief. Patients whose response to the above treatment regimen was partial or suboptimal were advised to participate in the present study.
The patients were 33 Caucasian females, 19 married, 14 single, 29 nulliparous and 4 multiparous, ranging in age from 21 to 45 years, with a mean of 31.5. Duration of experiencing vestibulitis symptoms ranged from 2 to 6 years, with a mean of 3.4. The self-reported symptoms varied from only introital dyspareunia to chronic, intense pain without any provocation. Twenty-eight of the 33 patients were abstinent from intercourse at the time of evaluation, and for them the average duration of abstinence was 13 months. The diagnosis of vulvar vestibulitis syndrome was confirmed by physical examination; in addition, tension and/or sensitivity to palpation of pelvic floor muscles was present. Other coexisting vulvovaginal conditions were excluded; if intercurrent vaginitis was present, it was specifically diagnosed and treated, and reevaluation of the diagnosis was performed prior to proceeding with the protocol. None of the patients selected for this study had prior surgery for the condition. All reported the previous use of topical treatments, and 27 took amitriptyline (range, of 5-85 mg) during the course of the treatment.

**Procedures and Equipment**

Once referred for pelvic floor EMG biofeedback, the patient underwent a brief history review, the initial pelvic floor muscle EMG assessment and training in the use of the portable EMG home trainer.

*The instrumentation employed in this study consisted of:*

1. A surface EMG single-user vaginal sensor (model SUP-V, PerryMeter Systems, Division of Biotechnologies, Inc., Dallas, Texas). This single-user sensor eliminates concerns about adequate sterilization; is easily inserted by the patient in private, with minimal instruction and without the need to completely disrobe; minimizes discomfort; and produces EMG data that correlate 99% with data produced by invasive fine wire stainless steel electrodes.17

2. The portable EMG biofeedback instrument was used by the patients for daily, in-home practice. The sensor described above can be used with any brand of EMG instrument, though some require a simple plug adapter. This study used a portable EMG biofeedback instrument specifically developed for use with the above sensor for rehabilitation of pelvic floor muscles (Personal Perineometer, model PP-200, Farrall Instruments, Inc., Grand Island, Nebraska, for Biotechnologies). This instrument provides visual feedback in the form of a circle of 20 lights; each light representing 0.25, 0.5 or 1.0 microVolt, according to the amplification setting selected. Two lights in the center of the circle alternate illumination for 10 seconds each, instructing the patient to alternately contract and relax the pelvic floor muscles.

3. Computerized EMG data acquisition equipment was used in the office for initial and periodic follow-up assessments of pelvic floor musculature. This study used the I-330 hardware interface with the Personal Computer Physiological Monitoring System (J & J Engineering, Inc., Poulsbo, Washington) and an Epson Equity LT 286 lap top computer (Torrance, California). EMG amplifier module model M-501 with a narrow filter bandpass (100-200 Hz, common mode rejection ratio 120 db), sensitivity setting of 0-100 microVolt root mean square, input noise 0.25 microVolt and 60-Hz notch filter, was used. The software used for data collection was the Universal Sensing Environment, version 1.20, system (J & J Engineering) and the Pelvic Muscle Rehabilitation System Application Program (version .90), (Biotechnologies).

A brief history was taken, including patient demographic data and information concerning the current condition, including onset, prior treatment, sexual activity and related symptoms, such as urinary frequency and urgency, low back pain and gastrointestinal symptoms. After the patient had privately inserted the sensor, she was seated, fully clothed, in a recliner. The sensor was then connected to the computerized EMG system, and the clinician, along with the patient, viewed the EMG signal on the computer screen. Information was gathered for the resting EMG levels of the pelvic floor muscles over a 1- to 5-minute period. The patient was then asked to tighten the pelvic muscles and to hold the contraction for 10 seconds to make sure that she was able to voluntarily contract the correct isolated muscle group. In many cases the patient had difficulty contracting the muscle, sensing the contraction or isolating the contraction to just the pelvic floor, without involvement of accessory muscles of the legs, abdomen and buttocks. In these cases the patients were given the necessary training with the aid of additional EMG information from accessory muscles and a videotape demonstrating pelvic floor muscle contractions until she was able to selectively contract the pelvic floor muscles voluntarily on command. In the first session and each subsequent session the patients were asked to provide subjective pain rating on a scale of O (least) to 10 (most).
Patients were given the anchor points on this scale by telling them to consider 0 as no pain at all, at any time, and 10 as the worst vestibular pain they had ever experienced.

The automated data collection protocol was then started. Consistent with the functions of the pelvic floor musculature (sexual, sphincteric and supportive), and in order to look at recruitment and endurance, there were three phases to the assessment, contracting the muscles for different durations (1, 10 and 60 seconds). At each contraction and intervening relaxation, maximal amplitude, minimal amplitude, mean amplitude and signal standard deviation were recorded.

**The automated data collection protocol proceeded as follows:**

1. Three 5-second baseline periods.

2. Six 5-second periods; in the middle of each period the patient was asked to create one strong, brief contraction.

3. Three 5-second baseline periods.

4. Ten 10-second periods alternating between rest and contract, starting with a rest-10-second rest/10-second contract repeated five times.

5. Ten seconds of rest.

6. One 60-second contraction.

Patients were then instructed in the operation of the portable EMG for home use. The standardized instructions used for all patients were to conduct 60 repetitions of the 10-second rest/10-second contract cycle (20 minutes), twice a day, with several hours between the two practice sessions. In addition, patients were instructed that as the number of lights "on" went down during a contraction (fatigue), they were to attempt to strengthen the contraction. Instructions were also given to increase sensory awareness by concentrating on the difference in the feelings of the muscle during contraction and relaxation.

Patients were given in-office reevaluations intermittently in order to ensure continued compliance with the home exercise regimen, to correct any negative or counterproductive practice habits, to collect data on pain and sexual activity and to reexamine the pelvic floor musculature with the above protocol. Patients were reexamined intermittently for a total of six evaluations. At the end of those evaluations the patients were instructed to continue the home exercises as described above, without the use of biofeedback, for a minimum of three months. Patients were contacted by telephone six months after their sixth evaluation to collect data on pain levels and sexual activity.

**Results**

In Table I, "pain" represents subjective ratings from the patients on a scale of 0 (least) to 10 (most). The "intercourse" data represent the number of patients reporting at least one episode of intercourse in the preceding 30-day period. The "contract" data represent the average EMG amplitude in microVolts over the five 10-second contraction periods alternated with 10-second rest periods. The "relax" data represent the average EMG amplitude in microVolts over the five 10-second rest periods. The "relax SD" data represent the standard deviation of the EMG signal during the five 10-second rest periods, a measure of the stability of the muscle at rest. "Number of days" represents the average number of days between evaluations. Figure 1 graphically represents the normalized (z score) measures of pain, intercourse, contraction, relaxation and relaxation standard deviation over the six evaluation sessions. Initial EMG evaluation of pelvic floor muscles in this patient group showed pretreatment mean resting levels of 2.5 microVolt RMS and pretreatment mean 10 second contractions 6.5 microVolt RMS. Post treatment mean resting levels were 0.8 microVolt RMS and l0-second contraction readings averaged 12.7 microVolt RMS. No population norms exist for these measures, so it is not possible to state that these readings differed significantly from the population norm or a comparable asymptomatic group. Perry, Hultett and Bollinger18 reported that patients with conditions associated with chronic pelvic tension show average pretreatment resting levels of 3.5 microVolt RMS and average pretreatment 10-second contractions of 6.9 microVolt RMS. Perry and Whipple 19 reported that their asymptomatic research subjects...
attain a 10-second contraction average of 8.77 microVolt RMS. Perry 20 stated that in their clinical experience, resting EMG levels in patients with chronic pelvic tension register in the 3-5 microVolt RMS range, while normal persons register 1-2 microVolt RMS; 12-15 microVolt RMS IO-second contraction average should be considered minimally healthy. With respect to pelvic floor muscle instability, our findings are that the standard deviation of the relaxation EMG signal is .42 microVolt RMS pretreatment and .16 microVolt RMS post treatment. No data on pelvic floor muscle stability could be found anywhere in the literature to serve as a comparison. Results indicated that after an average of 16 weeks of practice, pelvic floor muscle contractions increased 95.4%, resting tension levels decreased 68%, and the instability of the muscle at rest decreased by 62%. Subjective reports of pain decreased an average of 83%. Twenty-eight patients had abstained from intercourse for an average of 13 months, and 22 of these 28 patients (79%) resumed intercourse by the end of the treatment and follow-up period. Seventeen of the 33 patients reported pain-free intercourse at the six-month follow-up. A repeated measures analysis of variance indicated that over the six evaluation sessions and six-month follow-up of this study, subjective pain reports showed a significant decrease (F(6,192)= 162.86, P<.0001), and patients having intercourse showed a significant increase (F(6,192)= 24.41, P<.0001). Over the six evaluation sessions EMG amplitude during contractions showed a significant increase in muscle strength (.16(x)=20.51, L’.0001), and during relaxation periods, both resting EMG amplitude and the standard deviation of the EMG amplitude showed significant decreases (F(5,160)=26.54, P<.0001 and F(5,160)=14.11, P<.0001, respectively), indicating greater relaxation and stability in the pelvic floor musculature over the six evaluations.

Univariate F-tests comparing each evaluation with the previous evaluation for each measure yield significance in every case except the initial evaluation as compared to the second evaluation for both the relax standard deviation and the intercourse measures and for the second evaluation as compared to the third evaluation on the standard deviation measure (Table II). Thus, at each evaluation, pain decreased from the previous evaluation, the number of patients having intercourse increased from the previous evaluation after the second evaluation, strength increased from the previous evaluation, resting tension decreased from the previous evaluation, and muscle stability showed significant increases between each evaluation after the third evaluation.

In order to determine changes in characteristics of muscle activity that predict changes in pain or intercourse status, change scores were calculated by subtracting the sixth evaluation value from the first evaluation value for each measure. Descriptive statistics on the distributions of change scores for each measure reveal that they meet the assumption of normality of distribution required to conduct parametric statistical analyses. Zero order correlations between all possible pairings of these variables yielded no significant findings. Multiple regression analyses were conducted using the pain and intercourse status change measures as the dependent variables and all other measures and possible combinations of measures as predictor variables. Only the change in standard deviation of the relaxation significantly predicted change in pain (t=-2.036, P<.05). Thus, the more the muscle stabilized, the more subjective pain decreased. Change in intercourse status was predicted only by change in pain (t= - 2.243, P < .03). Thus, as pain decreased, the patient was more likely to engage in intercourse. When change scores for the pain and intercourse status were calculated by subtracting the value for the six-month follow-up from that of the first evaluation, no change in other measures predicted change in pain or intercourse status.

Analyses were also conducted to determine if initial characteristics of muscle activity were predictors of pain or intercourse status change. Descriptive statistics for each initial measure of muscle activity revealed that they meet the assumption of normality of distribution required to conduct parametric statistical analyses. Multiple regression analyses were conducted using the pain and intercourse status change measures from the initial evaluation to six-month follow-up as the dependent variable and all the initial muscle measures and possible combinations of measures as predictor variables. The results indicate that only the initial pain level significantly predicts pain change (Table III). Thus, the higher the initial level of pain, the greater the pain reduction. The high level of significance of this statistic clearly indicates that this is not merely regression toward the mean. The results indicate that intercourse status change is predicted by initial intercourse status and initial level of pain (Table IV). That is, women who were initially high in pain and not engaging in intercourse were more likely to show a change in intercourse status.

Discussion
Thirty-three women suffering from vulvar vestibulitis syndrome were shown to have elevated resting tension and contractile weakness of pelvic floor musculature. Rehabilitation of pelvic floor musculature with twice-daily exercises resulted in stronger, more relaxed and more stable pelvic muscles accompanied by a decrease in subjective pain reports and an increase in intercourse. While pelvic muscle stabilization measures lag behind measures of muscle strengthening and relaxation, only changes in pelvic muscle stability predict a reduction in self-report pain measures. The finding that pelvic floor muscle instability is a critical factor in vulvar vestibulitis syndrome is consistent with a recent study that urethral pressure variability is significantly greater in patients with vestibulitis than in either chronic pelvic pain patients or asymptomatic controls. The lag in symptomatic response is consistent with response to other successful therapies and is additional evidence that this may be an autonomically maintained pain syndrome. None of the muscle characteristic measures on initial evaluation predict pain or intercourse outcomes, so at this point it is impossible to select patients who are better candidates for this procedure. However, since all patients' terminal, self-reported pain was in the lower third of the pain rating scale, no matter where they started, the results show that those women who started with more pain showed a greater reduction in pain. Also, not surprisingly, as pain decreases, the likelihood of having intercourse increases.

In conclusion, pelvic floor muscle instability is a critical factor in pain associated with vulvar vestibulitis syndrome. A biofeedback-assisted exercise program that stabilizes the pelvic floor muscles significantly reduces and, in some cases, eliminates symptoms of vulvar vestibulitis syndrome. Further research is being conducted using additional measures of muscle activity, such as EMG spectral analysis and recruitment and recovery latencies, and studying the response of patients with vulvar vestibulitis syndrome as compared to those with other pelvic pain syndromes, such as interstitial cystitis, to pelvic floor muscle rehabilitation. The response to this therapy suggests that whatever the initial insult or etiologic factor, vulvar vestibulitis syndrome may be a result of autonomically mediared pain. This mechanism as a final common pathway for multiple etiologies may explain the lack of consensus on a single antecedent despite consistency in symptomatology of the syndrome.

Ultimately, successful therapy for this challenging clinical problem may involve intervention at multiple points of the cycle of autonomically maintained pain and its initial and intercurrent exacerbations by irritative, infectious and traumatic factors.

Discussion

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Notes

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Table I
Mean Pain Ratings, Intercourse Status, Muscle Contractile Strength, Muscle Relaxation Level, Muscle Relaxation Standard Deviation Average Number of Days Between Each of the Six Evaluations and Pain Rating at the Six-Month Follow-up

<table>
<thead>
<tr>
<th>Eval no.</th>
<th>Pain</th>
<th>Intercourse</th>
<th>Contract</th>
<th>Relax</th>
<th>Relax SD</th>
<th>No. of days</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>6.9</td>
<td>5</td>
<td>6.5</td>
<td>2.5</td>
<td>0.42</td>
<td>14</td>
</tr>
<tr>
<td>2</td>
<td>6.3</td>
<td>8</td>
<td>9.5</td>
<td>1.8</td>
<td>0.38</td>
<td>16</td>
</tr>
<tr>
<td>3</td>
<td>5.4</td>
<td>12</td>
<td>11.3</td>
<td>1.7</td>
<td>0.36</td>
<td>20</td>
</tr>
<tr>
<td>4</td>
<td>3.9</td>
<td>15</td>
<td>11.2</td>
<td>1.2</td>
<td>0.27</td>
<td>24</td>
</tr>
<tr>
<td>5</td>
<td>2.8</td>
<td>19</td>
<td>11.6</td>
<td>1.0</td>
<td>0.22</td>
<td>28</td>
</tr>
<tr>
<td>6</td>
<td>1.2</td>
<td>27</td>
<td>12.7</td>
<td>0.8</td>
<td>0.16</td>
<td>180</td>
</tr>
<tr>
<td>Follow up</td>
<td>0.9</td>
<td>27</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Table II
Univariate F-Tests Comparing Each Evaluation Level with the Previous Evaluation Level for Measures of Pain Ratings, Intercourse Status, Muscle Contractile Strength, Muscle Relaxation Level and Muscle Relaxation Standard Deviation

<table>
<thead>
<tr>
<th>Eval no.</th>
<th>Pain (F(1,32) Sig)</th>
<th>Intercourse (F(1,32) Sig)</th>
<th>Contract (F(1,32) Sig)</th>
<th>Relax (F(1,32) Sig)</th>
<th>Relax SD (F(1,32) Sig)</th>
<th>Intercourse (F(1,32) Sig)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td></td>
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<td></td>
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<td>4</td>
<td></td>
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</tr>
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<td>5</td>
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<td>6</td>
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<tr>
<td>Follow up</td>
<td></td>
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</tr>
</tbody>
</table>
Table III

Multiple Regression Analysis Using Initial Muscle States and Intercourse and Pain Status as Predictors of Pain Change from the First Evaluation to the Six Month Follow up.

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>SE B</th>
<th>Beta</th>
<th>T</th>
<th>Sig of T</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial pain rating</td>
<td>-0.753637</td>
<td>0.116927</td>
<td>-0.795231</td>
<td>6.445</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Initial intercourse status</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Initial contractile strength</td>
<td>-0.068386</td>
<td>0.078082</td>
<td>-0.109371</td>
<td>0.876</td>
<td>0.3888</td>
</tr>
<tr>
<td>Initial relaxation level</td>
<td>0.013475</td>
<td>0.171554</td>
<td>0.010066</td>
<td>0.079</td>
<td>0.9380</td>
</tr>
<tr>
<td>SD of initial relaxation level</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table IV

Multiple Regression Analysis Using Initial Muscle States and Intercourse and Pain Status as Predictors of Intercourse Status Change from the First Evaluation to the Six-Month Follow-up

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>SE B</th>
<th>Beta</th>
<th>T</th>
<th>Sig of T</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial pain rating</td>
<td>-0.718842</td>
<td>0.181985</td>
<td>-0.546751</td>
<td>-3.950</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>Initial intercourse status</td>
<td>0.100222</td>
<td>0.038666</td>
<td>0.376541</td>
<td>2.592</td>
<td>0.0152</td>
</tr>
<tr>
<td>Initial contractile strength</td>
<td>0.003529</td>
<td>0.025821</td>
<td>0.020097</td>
<td>0.137</td>
<td>0.8923</td>
</tr>
<tr>
<td>Initial relaxation level</td>
<td>-0.047260</td>
<td>0.056731</td>
<td>-0.125695</td>
<td>-0.833</td>
<td>0.4121</td>
</tr>
<tr>
<td>SD of initial relaxation level</td>
<td>0.416176</td>
<td>0.397756</td>
<td>0.150929</td>
<td>1.046</td>
<td>0.3047</td>
</tr>
</tbody>
</table>

Figure 1
Normalized (z score) measures of pain ratings, intercourse status, muscle contractile strength, muscle relaxation level and muscle relaxation standard deviation for each of the six evaluations.

References:
References

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