N National
V Vulvodynia
A Association

NVA News

Volume III, Issue III Fall 1997

# Biofeedback and Vulvovaginal Pain

## *Summary of a Lecture by Howard I. Glazer, Ph.D.*

*Howard I. Glazer, Ph.D., is a clinical associate professor of psychology in Obstetrics and Gynecology at Cornell University Medical College, and an associate attending psychologist at New York Hospital. He specializes in the use of surface electromyographic biofeedback in the treatment of vulvovaginal pain.*

Biofeedback, as the name suggests, involves the electronically-assisted measurement of physiologic processes such as heart rate, blood flow, and muscle contraction. Through the use of highly specialized computers, a specific physiologic process is translated into an auditory or visual signal which the patient learns to control by modifying the physiologic response. For example, a light turns off when the patient relaxes a particular muscle. After the initial training sessions in a biofeedback practitioner's office, practice typically takes place at home using portable equipment. Over time, the patient achieves some control of the underlying process, e.g., increasing blood flow to the extremities or relaxing neck muscles. This article focuses on the use of a specific type of biofeedback, surface electromyography (sEMG), in the treatment of vulvodynia and vulvar vestibulitis.

**History of Biofeedback**

Biofeedback had its origins in principles of learning, an area of experimental psychology. It was developed in the late 1960s in the laboratories of Dr. Neal E. Miller, a psychologist at Rockefeller University. (Glazer was his post-doctoral student.) In Dr. Miller's laboratory, biofeedback was used to demonstrate the capacity of animals and humans to self-regulate certain physiologic systems which were not previously thought to be under voluntary control. At the time, for example, it was widely held that gastrointestinal and cardiac responses were completely involuntary. Dr. Miller hypothesized that voluntary control of such responses offered great potential in the treatment of many disorders. For example, he proposed that learning to exert direct control over vascular constriction (narrowing of blood vessels) could reduce or eliminate migraine headaches. The ultimate success of biofeedback in teaching patients to regulate physiologic processes has led to applications in the treatment of many disorders, including irritable bowel syndrome, heart disease, and chronic pain.

*See BIOFEEDBACK, page 8*

# Medications for Chronic Pain

Vulvodynia is a chronic condition that exhibits many characteristics of neuropathic pain, i.e., pain resulting from nerve damage. Neuropathic pain is characterized by burning and, in some cases, stabbing sensations. Unlike vulvar vestibulitis patients who feel pain primarily upon touch, women with vulvodynia live with constant pain.

To alleviate the burning component, many doctors prescribe medications that have a successful track record in the treatment of other neuropathic pain conditions. This class of drugs is not intended to "cure" the disorder, but to provide symptomatic relief. This article will focus on a few of the most widely used medications in the treatment of vulvar pain.

*See MEDICATION, page 3*

## INSIDE

## LETTER FROM THE EXECUTIVE DIRECTOR

Dear Friend of the NVA:

As a result of the continuous publicity that vulvodynia has received in popular magazines this year, thousands of women have contacted the NVA in the past six months. In 1997, vulvodynia was discussed in health articles in New Woman, Ladies Home Journal, Good Housekeeping, Redbook, and American Health, among others. We continue to work hard to obtain this media exposure, and our next coverage will be in the December issue of Self magazine. NVA medical advisory board member Stanley Marinoff, M.D., and biofeedback expert Howard Glazer, Ph.D., were both interviewed for this upcoming article.

Everyone at the NVA is gratified by this explosion of publicity. After we started this organization in 1994, we spent two years writing press releases and knocking on doors, to no avail. Finally, now that our first vulvodynia conference was held at the National Institutes of Health, reporters and editors are knocking on our door. This means we're on our way to achieving recognition for this disorder.

At the same time, more and more doctors are finding out about vulvodynia from their patients, medical conferences, and the NVA newsletter. Although many of these doctors are not familiar with the treatment of vulvodynia, they are beginning to acknowledge that patients with unexplained vulvar pain need to see a specialist in vulvovaginal disorders, not a psychiatrist. Every time I receive a phone call from a health care professional interested in learning more about vulvodynia, I feel a sense of accomplishment. I am, however, keenly aware that we still have a long way to go in creating awareness of this disorder.

In this issue, the NVA is proud to announce the recipient of its first research award. The funds for this grant were provided by a longtime NVA supporter who asked that her donation be used specifically for vulvodynia research. It is our hope that we will receive future donations that will enable us to support other important research projects. Studies are needed to establish the prevalence of vulvodynia, to determine what causes it, and to measure the success rate of different treatments. Without this research, the treatment of vulvodynia will continue to be based on anecdotal evidence instead of scientific proof—and too many women will continue to suffer.

Very truly yours,

Phyllis Mate

# Medication

*(from page 1)*

## TRICYCLIC ANTIDEPRESSANTS

The most commonly prescribed group of medications for neuropathic pain are the tricyclic antidepressants such as amitriptyline, desipramine, and nortriptyline. The first tricyclics were approved by the FDA in the early 1960s. These drugs alter the transmission of pain impulses by modifying the levels of natural chemical messengers known as neurotransmitters. More specifically, the tricyclics alter the transmission, to varying degrees, of norepinephrine, acetylcholine, and serotonin at the neuronal junction. The end result is that the brain does not receive a pain message.

### Dosage

Generally, tricyclic dosage for the treatment of neuropathic pain is lower than the amount required to treat depression. Treatment begins at a very low dosage to enable the body to adjust to the drug. Consequently, it can take as long as two or three months to attain a dosage at which the patient experiences pain relief. If the patient becomes symptom-free on the medication, an attempt is usually made to reduce the dosage after some months have passed. The goal is for the patient to take the least amount of medication necessary to control pain. To avoid an adverse reaction, the medication is reduced slowly.

There are a few general guidelines for taking tricyclic antidepressants. As with most medications, they may be taken with food to minimize gastric distress. Ideally, tricyclics should not be taken at the same time of day as other drugs, but one to two hours afterward. Alcohol should be avoided because the tricyclic will increase its effect. Extreme drowsiness may result if tricyclics are combined with alcohol or other depressants such as narcotics, painkillers, sleeping medications, and tranquilizers. It is also especially important to check with your doctor or pharmacist before combining tricyclics with certain other drugs including Tagamet, Prozac, thyroid medications, and drugs that improve breathing, e.g., Ventolin.

### Side Effects

Tricyclic antidepressants may produce numerous side effects. The four most common groups of adverse side effects are sedative, cardiac, hypotensive (blood pressure-lowering), and anticholinergic (blocking the passage of impulses through the parasympa-

*See MEDICATION, page 4*

***Treatment begins at a very low dosage to enable the body to adjust to the drug.***

***NVA News***
**National Vulvodynia Association**
**P.O. Box 4491**
**Silver Spring, Md. 20914-4491**
**(301) 299-0775**
**FAX: (301) 299-3999**

The *NVA News* is published three times per year.

**Editor:**
Phyllis Mate

**Layout:**
Andrea Hall

**Contributors:**
Robin Masheb
Harriet O'Connor

## Medication

*(from page 3)*

thetic nerves). The most obvious anticholinergic effects are dry mouth and constipation. Anticholinergic effects on cognitive function such as confusion, short-term memory problems, and impaired attention also are reported, especially in the elderly. Some side effects, such as sedation and heart rate acceleration, tend to dissipate as the body adjusts to the drug, but the anticholinergic effects typically persist. Additional common side effects include nervousness, weight gain due to increased appetite, and an allergic-type skin reaction to the sun.

There are actions that can be taken to minimize some of the side effects of tricyclics. To protect against sun sensitivity, wear sunscreen (minimum SPF 30) and a wide-brimmed hat. If your doctor has prescribed the drug once a day at bedtime and early morning drowsiness is a problem, take the tricyclic earlier in the evening. Alternately, the doctor can reduce the initial dosage or have you switch to a less-sedating tricyclic. In order to counteract dry mouth, many people find that sucking on hard candy or sipping beverages is helpful. Increased fluid and fiber intake on a daily basis usually resolve the problem of constipation. Good sources of fiber include whole grains, and raw fruits and vegetables. If it is not possible to eat a high fiber diet, Metamucil or similar natural bulking agents are recommended. In order to prevent constipation, it is also important to drink 64-80 ounces of fluids (water, juice, decaffeinated coffee) daily.

**Amitriptyline and desipramine**

The most widely prescribed tricyclic is amitriptyline (Elavil), probably because it is the antidepressant with which many doctors are most familiar. The majority of vulvodynia patients on this medication take between 20 and 50

*See MEDICATION, page 5*

### Antidepressant Side Effects

| Generic/ Brand Names | Anticholinergic | Sedative | Heart Rate/Rhythm | Hypotensive |
|---|---|---|---|---|
| desipramine/ Norpramin | mild | mild | mild | mild |
| nortriptyline/ Aventyl, Pamelor | moderate | mild | mild | mild |
| trazodone/Desyrel | mild | moderate | moderate | moderate |
| imipramine/ Tofranil | moderate | moderate | moderate | moderate |
| doxepin/Adapin, Sinequan | moderate | strong | moderate | moderate |
| amitriptyline/ Elavil | strong | strong | strong | moderate |

Source: excerpted from Wolfe, Fugate, Hulstrand, Kamimoto, Public Citizen Health Research Group, Worst Pills, Best Pills (1993).

## Medication

*(from page 4)*

mg./day. Doctors usually prescribe an initial dosage of 10-20 mg. at bedtime and increase it by 10-20 mg. every week to ten days. Some doctors do not raise the dosage higher than 50 mg.daily, largely because the early medical literature only specified the use of "low dose" antidepressants for neuropathic pain. In recent years, however, other doctors have found that higher doses of tricyclic antidepressants are required for pain management. If symptom relief is not attained at 50 mg./day, they raise the dosage until it provides pain relief or reaches 150-300 mg./day.

Many patients stop taking amitriptyline for one of two reasons: they do not experience pain relief at their present dosage or they cannot tolerate the side effects. Patients on amitriptyline who are not experiencing pain relief should first inform their doctor. It is possible that not enough time has passed for the medication to have reached a therapeutic level or that the dosage needs to be adjusted. Similarly, if the side effects of amitriptyline cause considerable distress, patients should consult their doctor about the alternatives, such as switching to a different medication.

It is possible to experience unpleasant side effects on one tricyclic, yet tolerate another quite well. For example, many women cannot take amitriptyline because it makes them very drowsy, increases their heart rate, and/or impairs their mental functioning. This is especially true of the el-

*See MEDICATION, page 6*

# NVA Awards First Research Grant

After reviewing several outstanding research proposals, the NVA medical/executive board is pleased to announce that its $15,000 research grant has been awarded to Ursula Wesselmann, M.D., Assistant Professor of Neurology and Neurosurgery at The Johns Hopkins University School of Medicine. During her past three years at Johns Hopkins Hospital, Dr. Wesselmann has collaborated with gynecologist Dr. David Foster in the treatment of vulvodynia, and established a successful pain clinic focusing primarily on the treatment of chronic pain syndromes in women. Complementary to her clinical practice, she has built up a basic science laboratory devoted to the study of pain mechanisms.

The long-range goal of Dr. Wesselmann's current research is to advance our knowledge of the neural mechanisms underlying vulvodynia. She believes that one must understand the pathophysiology of vulvodynia in order to design specific treatment modalities directed against the pain mechanisms. This has been shown to be the most successful strategy for other chronic pain syndromes such as migraine headache, for which new medications were recently developed. Dr. Wesselmann and the NVA are hopeful that her basic science studies will result in improved treatment strategies for women who suffer from vulvodynia. ■

*(**Note**: Dr. Wesselmann was featured in the Summer 1996 issue of the NVA News.)*

### Letter to the Editor

Dear Newsletter Editor:

I recently had a miscarriage and am trying to get pregnant again. I would like to be contacted by anyone with vulvodynia who is currently pregnant, trying to get pregnant, or has already given birth. I can be reached by telephone or mail.
Also, I would like to know if any of the newsletters will cover pregnancy and vulvodynia. Thanks!

Amy Livingston
1857 E. Nevada Ave.
St. Paul, MN 55119
Home phone: (612) 771-4878

***Editor's Note**: There was a short article on vulvodynia and pregnancy in the Summer 1996 issue of the NVA News. I would like to encourage other readers interested in this topic to share their experiences by sending a letter to: NVA News Editor, P.O. Box 4491, Silver Spring, MD 20914.*

# Medication

*(from page 5)*

derly. In these situations, a doctor may choose to prescribe desipramine (Norpramin), a tricyclic antidepressant with considerably milder side effects (see chart on page 4).

David Foster, associate professor of Obstetrics/Gynecology, University of Rochester Medical School, prefers to prescribe desipramine for vulvodynia patients because of its potency for chronic pain relief and favorable side effect profile. Dr. Foster has recently been studying the effect of desipramine on vulvar pain. Although some of his patients respond at 50 mg./day, the majority require 150 to 200 mg. to experience noticeable pain relief. To date, symptoms have improved significantly in 68 percent of his patients using 150 to 200 mg. desipramine and 5 percent topical lidocaine.

### SSRIs — THE NEWER ANTIDEPRESSANTS

The selective serotonin reuptake inhibitor (SSRI) antidepressants, such as Prozac, Zoloft, and Paxil, have not yet been used extensively with vulvodynia patients. Today, many psychiatrists prefer to use them in treating depression because the SSRIs appear to have fewer side effects than the tricyclics. There is some research data and clinical experience to support their use in the treatment of chronic pain, but at this time their efficacy in the treatment of vulvodynia is unknown.

### ANTICONVULSANTS

Another group of medications frequently used to treat neuropathic pain are the anticonvulsants, commonly used to prevent seizures. These medications are typically prescribed and monitored by a neurologist who treats chronic pain patients. For the treatment of vulvodynia and other types of neuropathic pain, these medications are often prescribed at a lower dosage than is recommended for seizure control. Anticonvulsants tend to be given to vulvodynia patients when symptoms include a stabbing or lancinating (shooting) component; sometimes they are prescribed in conjunction with a tricyclic antidepressant.

### Tegretol (carbamazepine)

For the past 25 years, one of the most commonly used antinconvulsants in the treatment of neuropathic pain has been Tegretol. It has been widely used in the treatment of trigeminal neuralgia, a nerve condition causing episodes of extremely severe facial pain. Tegretol has also been used extensively in the treatment of peripheral neuropathy and pain syndromes following shingles, e.g., post-herpetic neuralgia.

### Dosage

Tegretol should be taken with food, preferably divided into three equal doses a day (or twice a day with the new extended release tablets). Similar to the regimen followed with antidepressants, the patient begins with a very low dose and may not reach a therapeutic level for four to eight weeks. By starting with a low dose and increasing it slowly, side effects of the drug may be minimized. For the treatment of pain, the initial adult dose is usually 100 mg./day, which is then increased by 100 mg. at weekly intervals. The recommended dosage for pain conditions is usually in the range of 400-800 mg. daily. Blood levels may be checked to ensure that dosing is adequate.

As with all newly prescribed medications, it is critical for the patient to tell her doctor what other medications she is currently taking. In particular, the use of erythromycin, cimetidine (Tagamet), propoxyphene (Darvon), isoniazid, or calcium channel blockers such as Calan may cause Tegretol to become toxic. It should also be noted that a combination of Lithium and Tegretol may cause harmful nervous system side effects.

### Side Effects

There are several side effects associated with the use of Tegretol, especially when dosing is first started. Dizziness, drowsiness,

*See MEDICATION, page 7*

## Moving?

Please send your change of address to the NVA:
P.O. Box 4491
Silver Spring, MD
20914

Newsletters are sent by bulk mail and are not forwarded by the U.S. Postal Service.

# Medication

*(from page 6)*

nausea, unsteadiness, and vomiting are the most common. These are likely to dissipate as the body adjusts to the medication. The most severe (and less common) side effects involve the blood, the skin, and the cardiovascular system. In addition to having regular medical examinations, it is important to report any noticeable drug reaction to your doctor. Because Tegretol may cause serious blood abnormalities, patients on this drug must undergo routine blood monitoring. Blood tests are conducted frequently at the beginning of treatment; follow-up blood tests should be performed every six to 12 months.

***It is possible to experience unpleasant side effects on one tricyclic, yet tolerate another quite well.***

As with other anticonvulsants, long-term use of Tegretol may contribute to certain physical disorders such as osteoporosis. As a preventative measure, it is advisable to take calcium citrate (plus Vitamin D) and magnesium supplements. Yearly opthamological examinations and regular dental check-ups also are recommended.

### Neurontin — The newest anticonvulsant

Over the past two years, many neurologists have been prescribing Neurontin (gabapentin), a new anticonvulsant drug, for the treatment of some chronic pain conditions. This medication was approved by the FDA in 1994. Unlike Tegretol, routine blood monitoring of Neurontin is not considered necessary for safe use. Since Neurontin appears to be effective and has far fewer side effects than other anticonvulsants, many neurologists are starting to use it in the treatment of neuropathic pain.

### Dosage

As an anticonvulsant, the recommended dosage of Neurontin is 900-1800 mg. daily, usually divided into three equal doses. For the treatment of pain, it is best to start with a small dosage, such as 300 mg./day, and increase the amount slowly, adding 300 mg. every five to seven days. Dosages up to 2700 mg. daily have been well-tolerated by patients in long-term clinical studies. Neurontin may be taken with or without food, but not within two hours of ingesting Maalox or other antacids, because the combination will decrease its effect. Even though it is not expected to be of clinical significance, it should also be noted that cimetidine (Tagamet) causes a small reduction in the excretion of Neurontin.

### Side Effects

One of the major advantages of Neurontin is that it is well-tolerated compared to the other anticonvulsants. However, it can cause some adverse side effects. The most common of these include sleepiness, ataxia (inability to coordinate voluntary bodily movements, such as walking), fatigue, dizziness, and nystagmus (involuntary rapid movement of the eye). In some patients, weight gain and/or fluid retention may occur.

### A Final Word

It is important to take tricyclic antidepressants and anticonvulsants only as they are prescribed by your doctor. These medications should not be abruptly discontinued. If you want to stop taking one of these medications, consult your doctor first.

(***Editorial Note**: Tricyclics and anticonvulsants must be kept away from young children, as overdosing may be lethal.*)■

### Back Issues Available to Subscribers

Eight back issues of the newsletter are available to anyone who is an NVA contributor. If you wish to purchase all the back issues, please send your name and address, along with a check for $30, to:

Newsletters
P.O. Box 4491
Silver Spring, MD 20914

# Biofeedback

*(from page 1)*

Some years later, Dr. Catherine Bergio first applied biofeedback technology to the pelvic floor muscles in the treatment of urologic conditions such as urinary urge incontinence and stress incontinence. Urge incontinence refers to the involuntary loss of urine resulting from abrupt urinary urge. Stress incontinence involves the loss of urine when laughing, coughing, sneezing, standing quickly, or experiencing other acute intra-abdominal pressure events; it is associated with a weakened external urethral sphincter. Both the external urethral and anal sphincters are the continuation of fibers from the pelvic floor muscle. Compromised or weakened pelvic floor muscles and sphincters may result from surgery, trauma, decrease in estrogen, normal muscle deterioration, or neurologic degeneration.

To correct both types of urinary incontinence, Dr. Bergio used a biofeedback combination. Activity of the urethra, pelvic floor musculature, and detrusor muscle (bladder muscle) were measured. The treatment worked as follows: sEMG readings of the external urethral sphincter enabled the patient to exert control over the contraction of the sphincter, turn off activity of the detrusor muscle, and thereby prevent involuntary urine loss associated with abrupt urge or acute intra-abdominal pressure.

One drawback of Dr. Bergio's incontinence treatment was the invasive nature of the procedure. Treatment required that tubes be inserted in the bladder and urethra, and external surface patches be applied to the perineal area. Another limitation was the need to conduct the treatment in a hospital setting. Subsequently, Dr. John Perry developed a less invasive office procedure, requiring the patient to insert a small tampon-like sensing device into the vagina to measure the electrical activity of the pelvic floor muscle. Dr. Perry's procedure is still in use today. The patient privately inserts the sensing device and then re-enters the office fully clothed, with the sensor wire pulled through the clothing. The wire is then plugged into a computer which reads and analyzes the electrical activity of the pelvic floor muscle.

***To date, vulvodynia and VVS have been diagnosed based on a description of symptoms, rather than physical findings or etiology.***

### Application to Vulvovaginal Pain

In 1991, Dr. Glazer was approached by a group of specialists working exclusively with patients suffering from vulvovaginal pain conditions such as vulvodynia and vulvar vestibulitis syndrome (VVS). This group had identified, upon digital vaginal examination of their patients, high levels of tension and instability in the pelvic floor muscle. This was not surprising since it was well-known that in any area of the body where soft tissue pain is experienced, the local muscle becomes tense as part of a natural guarding process intended to protect the area from pain. Dr. Glazer hypothesized that the muscle tension was a consequence of the vulvar pain. If this hypothesis were true, working with the pelvic floor muscle would not relieve the pain, because the underlying cause of the vulvodynia or VVS would not be addressed.

At the time, however, commonly used treatments for vulvodynia and VVS were not very effective. Topical palliatives (e.g., Aveeno solution), low dose tricyclics, and antihistamines provided minimal pain relief. Surgery, an option for some patients, was rarely pursued as a first line treatment. Given these limited alternatives, Dr. Glazer and the group of vulvar pain specialists decided to try biofeedback. Dr. Glazer treated approximately 50 patients with diagnoses ranging from pure VVS (i.e., sharp vaginal entry pain only) to combinations of subtypes of vulvodynia. Many patients also had associated conditions such as interstitial cystitis, irritable bowel syndrome, and fibromyalgia. Surprisingly, the

*See BIOFEEDBACK, page 9*

## Biofeedback

*(from page 8)*

strengthening, relaxation, and stabilization of the pelvic floor muscle in many of these patients resulted in pain relief. Unlike the rapid relief experienced by urologic patients, however, the vulvar pain patients needed many months of diligent daily practice to achieve symptomatic relief.

### Research Findings

The results of Dr. Glazer's first 35 patients were reported at the 1993 Congress of the International Society for the Study of Vulvovaginal Disease, and published in the April 1995 Journal of Reproductive Medicine. In summary, 83 percent reported overall improvement in vulvar pain symptoms. Slightly over 50 percent of the 35 patients were asymptomatic at the end of treatment, and remained pain-free at six month follow-up. Subsequent to publication of the study, Dr. Glazer continued to follow the progress of these asymptomatic patients, all of whom were still pain-free after two or more years. This study is currently being replicated by a Canadian research group, with Dr. Glazer's participation. Preliminary data appears consistent with the results of the initial study.

These findings have led to a change in some health care professionals' understanding of vulvovaginal pain. Originally, it was believed that muscle activity was a secondary process intended to protect the area from pain. The observation that decreasing pelvic floor muscle instability leads to symptomatic vulvar pain relief, however, suggests that muscle instability plays an integral part in the maintenance of the pain.

Clearly more research is needed, but one explanation for these findings may stem from a chronic pain condition known as reflex sympathetic dystrophy (RSD). The typical response to injury or trauma is as follows: histamine is released and causes swelling; vasoconstriction occurs and leads to proliferation of blood vessels and redness; and guarding of the tissue takes place and results in muscle tension. These natural responses usually protect and help resolve the injury. As the tissue heals, these responses dissipate. When RSD occurs, however, these protective responses do not dissipate. Instead, the mechanisms which are intended to promote healing persist, and the pain condition is maintained rather than resolved. In other words, swelling, redness, and guarding are only helpful in the early stages of the healing process following an injury or trauma. Prolonged swelling, redness, and guarding actually maintain the pain condition.

In the April 1995 issue of the Journal of Reproductive Medicine, Dr. Glazer and his colleagues, Gae Rodke, M.D., Charles Swencionis, Ph.D., Alexander Young, M.D., and Ronny Hertz, M.D., hypothesized that instability of the pelvic floor muscle maintained VVS. The authors speculated that a hyperactive and unstable pelvic floor muscle might cause a reflex, or signal, from the nerves of the pelvic floor to the nerves of the local spinal cord. Thus, VVS might be a variation of RSD.

There is no strong scientific evidence that VVS is a variation of RSD, but the theory does provide a descriptive understanding of what may be happening in VVS. According to this theory, the nerves of the local spinal cord activate the sympathetic nervous system, which in turn causes the release of histamine and vasoconstriction. When inflammation and increased blood vessel growth occur in an area of densely packed nerve endings (as is the case in the soft tissue area of the vulva), pressure applied to the soft tissue produces a burning sensation as experienced in vulvodynia, and sharp pain and tissue tenderness as reported in VVS.

Another finding with important treatment implications for vulvovaginal pain conditions emerged from the 1995 study. The researchers were interested in determining which characteristic(s) of the pelvic floor

*See BIOFEEDBACK, page 10*

**Web Site Change**

The NVA's Web Site has a new address:

www.nva.org

## Biofeedback

*(from page 9)*

muscle were associated with pain relief. They discovered that the stability of the muscle at rest was the only characteristic that predicted pain relief. Whereas the goal for incontinent patients was to increase control by strengthening the pelvic floor muscle, simply strengthening the muscle did not relieve pain in VVS patients. Rather, the results of this study indicated that the stabilization or reduction of variability of the pelvic floor muscle at rest was the key factor in alleviating vulvar pain.

It should be noted that biofeedback equipment and protocols for the pelvic floor muscle were originally designed for the treatment of incontinence. To treat vulvovaginal pain conditions, it is essential to have software that also measures pelvic floor muscle stability. In addition to this requirement, the other important component of treatment is the protocol, i.e., the method of scientific measurement in which the same procedures are followed at each session, so that changes in the muscle can be monitored over time.

A second study at Cornell University is currently in the process of completion and publication. The purpose of this study, by Romanzi, Polenewski, and Glazer, is to investigate the accuracy and reliability with which physicians diagnose patients with pelvic floor muscle tension. Currently, physicians identify pelvic floor muscle tension by palpation. This research is important to determine whether physicians are correctly identifying and referring vulvovaginal pain patients for sEMG biofeedback treatment.

In this study, pelvic floor muscle tension was measured in two ways: by physicians' examinations and with sEMG biofeedback. Findings indicated that physicians' measurements were not consistent with sEMG biofeedback readings. In addition, the consistency with which physicians accurately identified muscle tension varied greatly among practitioners. Generally, physicians' accuracy improved when identifying muscle tension within the same patient, in repeated sessions over time. Overall, these findings suggested that physicians do not assess pelvic floor muscle tension with sufficient reliability, and that an initial sEMG biofeedback assessment is a more reliable diagnostic tool.

Another biofeedback study by White, Jantos, and Glazer was published in the Journal of Reproductive Medicine in April 1997. The purpose of this study was to differentiate subtypes of vulvodynia patients from normals, based on readings of pelvic floor muscle stability. Consistent with the results of the 1995 study, the stability of the muscle at rest proved to be 92 percent accurate in identifying vulvodynia patients. Additional characteristics that reliably differentiated vulvodynia patients from normals included: the speed of onset of contractions, the amplitude of the muscle contraction, the speed with which the muscle returned to rest, the post-contraction resting baseline, and the muscle fibers used during contraction.

These results have important diagnostic implications. To date, vulvodynia and VVS have been diagnosed based on a description of symptoms, rather than physical findings or etiology. If it turns out that vulvodynia or VVS can be accurately diagnosed with sEMG biofeedback, this would be the equivalent of having a blood test that reveals a positive or negative result.

Additionally, the White, Jantos, and Glazer paper discussed the case of a vulvodynia patient who was referred for sEMG biofeedback, but upon evaluation presented with a normal pelvic floor muscle. Further medical testing revealed that the patient had an infectious condition. When treated for this condition, the patient's vulvar symptoms disappeared. Dr. Glazer has seen several patients diagnosed with vulvodynia and referred for sEMG biofeedback whose pelvic floor muscle appeared normal during evaluation. He has referred all of these patients back to their gynecologists for a more detailed workup. Although not empirically studied, in each case, a gynecological abnormality or external irritating factor that had not been addressed was determined to be the source of the patient's problem.

The ongoing Canadian study is producing other interesting findings. This is the first study to demonstrate controlled comparisons of surgery, biofeedback, and psychological/psychosexual approaches to the treatment of vulvodynia. Pure VVS patients, i.e., those with sharp vaginal en-

*See BIOFEEDBACK, page 11*

# Biofeedback

*(from page 10)*

try pain only, were randomly assigned to one of three groups: vestibulectomy, biofeedback, or talk therapy with a combination of group and sex therapy. By the three month follow-up, surgery produced the best results, with biofeedback a close second. Talk therapy produced little or no positive results. At six month follow-up, the effectiveness of surgery remained the same, the biofeedback group showed additional improvement, and talk therapy continued to show little or no benefit.

At the September 1997 meeting of the International Society for the Study of Vulvovaginal Disease, Glazer, Jantos, Hartmann, and Swencionis presented a poster comparing sEMGs of vulvodynia patients and normals. This current research demonstrates that, in addition to pelvic floor muscle stabilization measures, muscle fiber readings are emerging as a key factor in predicting pain reduction. These readings specify which type of muscle fiber the patient is contracting during training exercises.

## Summary

In summary, these research findings indicate that sophisticated analysis of the pelvic floor muscle using sEMG biofeedback equipment is helpful in the diagnosis and treatment of vulvovaginal pain. Pelvic floor muscle changes must not only include stabilizing the muscle, but strengthening the correct muscle fibers, to alleviate pain. Repeating the identical assessment procedure at each session allows for comparison over time to assure that rehabilitation is occurring. Some patients achieve pelvic floor muscle strengthening or relaxation, but not pain relief. A more detailed analysis may indicate that the patient is contracting the wrong fibers or that the pelvic floor is more relaxed, but still unstable.

## Selecting a biofeedback specialist

In selecting a biofeedback practitioner, it is necessary to keep in mind that the treatment of vulvovaginal pain with sEMG biofeedback is new. First, look for a specialist with specific training in sEMG biofeedback for the pelvic floor; ideally this will include training in the treatment of vulvovaginal pain conditions. Second, ask the practitioner if the office equipment and protocols include readings of muscle fibers, as well as muscle stability.

Insufficient practitioner training, or inadequate equipment and protocols, are likely to affect the quality of the biofeedback results. *Only sEMG biofeedback can measure the stability of the pelvic floor muscle at rest and specify which muscle fibers are used during contraction.* Dr. Glazer has heard patients say, "I've tried biofeedback and it didn't work," or "it had limited value." Frequently, these patients used instruments involving electrical stimulation of pelvic floor muscles, or alternative measurement devices such as manometers, instruments used for pressure readings. Vulvar pain patients who have had unsuccessful experiences with biofeedback, but did not use sEMG biofeedback, may want to consider trying it again with a practitioner who has appropriate training and equipment.

## References

1. Glazer, H.I., Rodke, G., Swencionis, C., Hertz, R., Young, A. Treatment of vulvar vestibulitis syndrome with electromyographic biofeedback of pelvic floor musculature. J. Reprod. Med. 1995; 40:283-90.

2. Romanzi, L., Poloneczky, M., Glazer, H. A simple technique for assessment of pelvic muscle function as a part of routine pelvic examination; validation by surface electromyography. Manuscript in preparation.

3. White, G., Jantos A., Glazer, H. Establishing the diagnosis of vulvar vestibulitis. J. Reprod. Med. 1997; 42:157-60. ■

# THE NVA NEEDS YOUR CONTRIBUTION

## I WANT TO SUPPORT THE NVA AND RECEIVE MORE INFORMATION ON VULVODYNIA.

Name _____

Address _____

_____

Phone (H) _____ (O) _____

The NVA needs the support of everyone: patients, families, and health care providers.

❑ $35 ❑ $50 ❑ $100 ❑ Other $ _____

❑ Yes, I would like to be contacted by other NVA supporters in my area.

❑ No, I do not want to be contacted. Please keep my name confidential.

Please send your check or money order, payable to NVA, together with your name, address and telephone number to: NVA, P.O. Box 4491, Silver Spring, MD 20914-4491.

N
V
A

NATIONAL VULVODYNIA ASSOCIATION

P. O. Box 4491 ❖ Silver Spring, MD 20914-4491