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Chronic Pain: Peripheral or Centralized?

By Daniel Clauw, M.D.

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Clinical practitioners sometimes examine patients with pain and other somatic symptoms that they cannot adequately explain based on the degree of damage or inflammation noted at that site of the body. The goal of a typical physical evaluation is to determine the cause of the pain. If no cause is found, an individual is often given a diagnostic label that merely connotes that the patient has chronic pain without an identifiable cause in a specific area of the body, e.g., chronic low back pain, headache, or vulvodynia. In other cases, the diagnosis alludes to an underlying pathologic abnormality that may or may not be responsible for the individual's pain, e.g., endometriosis. In the worst case scenario, doctors tell patients that there is nothing wrong with them and the disorder is "all in their head".

Until recently, these unexplained pain syndromes perplexed researchers, clinicians and patients. Research over the past decade on chronic pain, however, has led

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Pudendal Neuralgia: An Unrecognized Cause of Pelvic Floor Pain

By Michael Hibner, M.D.

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Pudendal neuralgia is an important but often unrecognized and undiagnosed cause of pelvic floor pain. Its incidence is unknown, and there is relatively little data and scientific evidence in the literature on its diagnosis and treatment. However, I believe that a significant number of women who have burning pain in the vulva, clitoris, vagina, perineum, or rectum – including women who are diagnosed with interstitial cystitis, pelvic floor muscle spasms, vulvodynia, or other conditions – may in fact have pudendal neuralgia. Indeed, pudendal neuralgia is largely a diagnosis of exclusion, and such conditions often must be ruled out. But the neuropathic condition

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us to rethink our approach. Our current paradigms for diagnosing and treating chronic pain are antiquated. We should think of pain along a continuum from peripheral or nociceptive (pain resulting from damage to tissue or nerves) to centralized pain, which originates from or is amplified by the Central Nervous System (CNS), i.e., the brain and/or spinal cord.

Peripheral or nociceptive input might be partly responsible for most chronic pain, but in some individuals (with or without identifiable nociceptive input), CNS factors likely amplify the pain. In this situation, the patient's "volume control" for pain is set too high. Volume control is set by a variety of factors, including the levels of neurotransmitters that facilitate pain transmission (turn up the volume control) and those that reduce pain transmission (turn down the volume control). Centralized pain appears to be due in part to an imbalance in levels of these neurotransmitters. This imbalance may also result in fatigue, memory problems and sleep and mood disturbances, because the same neurotransmitters that control pain and sensory sensitivity also control sleep, mood, memory, and alertness.

Individuals with centralized pain conditions display diffuse hyperalgesia (increased pain from normally painful stimuli) and/or allodynia (pain from normally non-painful stimuli) along with sensitivity to multiple sensory stimuli, such as noises, odors, and bright light. The pain is often described by individuals as, "gnawing," "burning" or "stabbing". These individuals often have childhood and adolescent histories of chronic pain, e.g., menstrual cramping, abdominal pain, growing pains. Individuals with this pattern are also likely to have a family member(s) with a pain syndrome. Recent studies have shown that a variety of chronic pain conditions are familial, and specific genes that increase or decrease pain processing are rapidly being identified.

Centralized pain can be triggered or exacerbated by stressors, including infection with Lyme disease, Epstein–Barr virus, and viral hepatitis; surgery; deployment to war; physical trauma, e.g., motor vehicle accidents; and peripheral pain syndromes, e.g., osteoarthritis. Psychological stress may also exacerbate centralized pain. As a result of the pain, individuals typically begin to reduce daily activities and have difficulty fulfilling their responsibilities. They may neglect spouses or children and experience difficulty with work inside or outside the home. This can lead to maladaptive illness behaviors, such as isolation, cessation of pleasurable activities, poor sleep and reduction in activity and exercise, which can then lead to increased pain.

Evaluation and Diagnosis

The evaluation of an individual with chronic pain is a complex process. Unlike most other medical problems,

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The National Vulvodynia Association is a nonprofit organization that strives to improve women's lives through education, support, advocacy and research funding.

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simply arriving at a diagnosis is insufficient to guide treatment. That is because with any pain diagnosis there is tremendous heterogeneity with respect to the causes, contributing factors, and effective treatment. In particular, individuals with chronic pain may have peripheral or nociceptive contributors, e.g., tissue damage or inflammation, plus central non-nociceptive contributors, e.g., pain amplification. Therefore, the differential diagnosis of chronic pain involves identifying which of these factors are present in which individuals. This knowledge leads to the administration of appropriate pharmacologic and non-pharmacologic therapies or medical/surgical procedures.

During a health care visit, it is essential to provide a detailed history of chronic pain. As mentioned before, individuals with centralized pain often begin having chronic pain in multiple areas of the body at a relatively young age and the pattern continues throughout their lives. What often appears to one specialist as a new pain episode in a specific area of the body is often one in a series of chronic pain conditions that have occurred over the lifetime of an individual.

It is also important to focus on the characteristics of an individual's pain that can help distinguish it from other disorders. Centralized pain is typically diffuse or multifocal, often waxes and wanes, and is frequently migratory. These characteristics are quite different from those of peripheral pain, in which both the location and severity of pain are typically more constant. Patients with centralized pain may complain of discomfort when they are touched and may experience dysesthesias (unpleasant abnormal sensations produced by normal stimuli) or paresthesias (prickly, tingling sensations).

Critical information can be gleaned from the history and physical examination findings that can further help identify individuals with centralized pain. For example, individuals with centralized pain states very often demonstrate altered noxious thresholds (the point at which a sensory experience such as pressure, heat, or sound becomes unpleasant) for virtually every type of sensory stimulus. This can be easily understood by patients and clinicians alike when the phenomenon is conceptualized as increased volume control in the brain for any sensory stimulus. Individuals with centralized pain states often complain that they find noises, odors, and bright lights bothersome, and this altered sensory sensitivity may explain many of the visceral symptoms these individuals experience, e.g., indigestion, heartburn, abdominal pain, urinary urgency and frequency. Understanding sensory augmentation can be very helpful to patients because they may be less concerned when they develop new symptoms that follow this same pattern—symptoms that before might trigger a frustrating search for the cause of the current pain.

Treatment

Once it has been determined whether a peripheral or central mechanism (or both) is causing an individual's pain, an appropriate treatment plan can be instituted. Peripheral or nociceptive pain is typically more responsive to nonsteroidal anti-inflammatory drugs (NSAIDs) and opioids as well as certain medical and surgical procedures. Centralized pain is thought to be responsive to neuroactive compounds that alter the levels of neurotransmitters involved in pain transmission. Because the neurotransmitters that control pain sensitivity are also known (in different and overlapping brain regions) to control sleep, level of alertness, memory and mood, when a patient receives an appropriate dose of a centrally acting analgesic, he or she typically experiences improvement in many symptoms other than pain. For all chronic pain, it is important to use symptom-based pharmacologic therapy together with non-pharmacologic therapies. Pharmacologic treatments typically target symptoms, whereas non-pharmacologic treatments target the functional consequences of the symptoms, such as decreased activity, isolation and poor sleep.

Pharmacologic therapy

Effective pharmacologic therapies for centralized pain generally work in part by reducing the activity of neurotransmitters that facilitate pain transmission or

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by increasing the activity of inhibitory neurotransmitters that reduce pain transmission. Several drugs, or classes of drugs, have strong evidence for effectively treating centralized pain, including tricyclic compounds (amitriptyline, cyclobenzaprine), gabapentinoids (pregabalin, gabapentin), serotonin norepinephrine reuptake inhibitors (duloxetine, milnacipran), and gamma-hydroxybutyrate. Gamma-hydroxybutyrate is a scheduled substance due to its abuse potential as a date rape drug and has not been approved by the U.S. Food and Drug Administration as a pain treatment because of safety concerns. It is available in the United States, however, to treat sleep disorders. Individuals whose pain is not being controlled by other medications may wish to see a sleep specialist to determine if they have a sleep disorder warranting a prescription for gamma-hydroxybutyrate, which may have the added benefit of reducing their pain.

Drugs with more limited evidence of efficacy for centralized pain include older selective serotonin reuptake inhibitors with greater noradrenergic activity when used at higher doses (e.g., fluoxetine, paroxetine, sertraline), low-dose naltrexone, esreboxetine (a serotonin norepinephrine reuptake inhibitor not available in the U.S.), and cannabinoids (marijuana or synthetic cannabinoids). There are an increasing number of states that have legalized use of marijuana for medicinal purposes, specifically for individuals with chronic pain living in these states. Synthetic cannabinoid has not been approved in the U.S. for pain control, but has been approved in many other industrialized nations. Without synthetic cannabinoids, it is difficult for health care providers to offer guidance on appropriate dosages. It is known, however, that oral ingestion of cannabinoids (e.g., baked into foods, brewed in tea), rather than the synthetic version, is needed to maintain pain control over an extended period of time.

Conversely, individuals with centralized pain conditions typically do not respond well to therapies that are effective for acute pain or pain caused by damage to or inflammation of tissues (e.g., NSAIDs, opioids, injections, surgical procedures). In fact, there is evidence that opioids might worsen centralized pain states, leading to opioid-induced hyperalgesia. Individuals who are taking a moderate to high dose of opioids every day may wish to think about their initial pain level and functional status when they first started taking the drug. Individuals whose pain intensity and level of functioning have not improved should consider talking with their health care provider to determine whether opioids are the best class of drugs for their pain.

Non-pharmacologic therapy

Many individuals with centralized pain respond well to simple interventions that relieve stress, improve sleep patterns and increase activity and exercise. The body's two most potent internal analgesic systems are sleep and exercise. At present, it is known that poor sleep can contribute to centralized pain symptoms, therefore increasing the amount of sleep is an important

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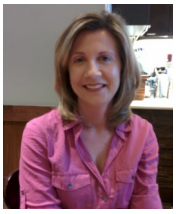
If you participate, you can help us raise thousands of dollars for a worthy cause, at no cost to you! Please sign up today.

NVA Welcomes New Executive Director



For more than 20 years, Lisa Goldstein has been advocating for women's health. Ms. Goldstein earned her Bachelor of Arts degree in Psychology and Economics and a Management Certificate from the University of Rochester in Rochester, NY. She continued her education there, obtaining a Master's of Science degree in Public Policy Analysis with an emphasis on Health Policy in 1994. While at the university, she worked in the Department of Ob-Gyn Residency Education Office, which led to her interest in women's health and to her career at the American College of Obstetricians and Gynecologists (the College). Ms. Goldstein began at the College as an intern, working her way up through the organization to become a Senior Director. In her position as Senior Director, Ms. Goldstein directed the Committee on Health Care for Underserved Women and the Committee on Adolescent Health Care, providing leadership, expertise, and direction to the College in these areas. Her work frequently required research, analysis, writing and editing; facilitation of meetings designed for strategic planning and consensus building; and information sharing to increase awareness of women's health care needs that are not fully met by the health care system. Given the unmet needs of women with vulvodynia, this experience and her commitment to women's health made Ms. Goldstein an ideal candidate for the position of Executive Director. We welcome her to the NVA. Ms. Goldstein is looking forward to working with you to advance the mission of the NVA and can be reached via email at lisa@nva.org.■

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should be suspected in women who have burning pain in any area along the distribution of the pudendal nerve. Awareness of the nerve's anatomy and distribution, and of the hallmark characteristics and symptoms of pudendal neuralgia, is important, because earlier identification and treatment appears to provide better outcomes. Most of the patients in our practice have pudendal neuralgia caused by mechanical compression – what is referred to as pudendal nerve entrapment.

The condition is sometimes referred to as cyclist syndrome because, historically, the first documented group of patients with symptoms of pudendal neuralgia was competitive cyclists. There is a misconception, however, that the condition only occurs in cyclists. In fact, pudendal neuralgia and pudendal nerve entrapment specifically may be caused by various forms of pelvic trauma, from vaginal delivery (with or without instrumentation) and heavy lifting or falls on the back or pelvis, to previous gynecologic surgery, such as hysterectomy, cystocele repair, and mesh procedures for prolapse and incontinence.

Pudendal neuralgia is multifactorial, involving not only compression of the nerve, for instance, but also muscle spasm and peripheral and central sensitization of pain. Treatment involves a progression of conservative therapies followed by decompression surgery when these conservative treatments fail.

Symptoms

In most cases, patients will describe neuropathic pain – a burning, tingling, or numbing pain – that is worse with sitting, and less severe or absent when standing or lying down. Initially, pain may be present only with sitting, but with time pain becomes more constant and severely aggravated by sitting. Many of my patients cannot tolerate sitting at all. Interestingly, patients usually report less pain when sitting on a toilet seat, a phenomenon that we believe is associated with pressure being applied to the ischial tuberosities rather than to the pelvic floor muscles. Pain usually gets progressively worse throughout the day.

Patients often will report the sensation of having a foreign body, frequently described as a golf or tennis ball, in the vagina, perineum, or rectum. Pain with urination and/or bowel movements, and problems with frequency and urgency, also are often reported, as is pain with intercourse. Dyspareunia may be associated with sexual

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arousal, penetration, orgasm, or any combination. Some patients report feeling persistent sexual arousal.

Occasionally, patients report having pain in regions outside the areas of innervation for the pudendal nerve, such as the lower back or posterior thigh. The presence of sciatica, or pain that radiates down the leg, for instance, should not rule out consideration of pudendal neuralgia. Just as worsening pain with sitting is a defining characteristic, almost all patients also have an acute onset of discomfort or pain; their pain can be traced to some type of traumatic event. Many of our patients trace the onset of their symptoms to immediately after gynecologic surgery, particularly vaginal procedures for prolapse or incontinence. Some patients report a more gradual onset of symptoms after surgery.

Diagnosis

The most important element of the diagnosis of pudendal neuralgia is the history, particularly regarding the onset of pain, the location of pain, and the nature of symptoms. History and physical examination both are important for ruling out other reasons for pain, including vulvodynia, pelvic floor tension muscle spasm, and interstitial cystitis. A pelvic exam often will reveal significant tenderness in the pelvic floor muscles. Patients with pudendal neuralgia often have a trigger point – a place of maximal tenderness and pain – at the ischial spine. Palpation of this area to produce what's known as a Tinel's sign (with pain and symptoms) thus should be part of the exam.

Also key to diagnosis are computed tomography–guided blocks of the pudendal nerve. In our practice, we consider any degree of pain relief, for any duration of time after the block, as supportive of a diagnosis of pudendal neuralgia. Patients who do not experience immediate relief from a block are thought not to have the condition. These image-guided blocks must be performed by experienced interventional radiologists with a local anesthetic.

To date, there are no imaging studies that are reliable for diagnosis. Ongoing advances in magnetic resonance imaging (MRI) and magnetic resonance neurography (MRN) may make these modalities valuable in the future, but currently these techniques yield too many false negative results. Pudendal nerve motor terminal latency, which measures the conduction velocity of electrical impulses, is not useful given a high rate of intra- and inter-observer variability and variations among patients who have had previous vaginal deliveries or pelvic surgery. Sensory threshold testing also has questionable reliability.

Initial Treatments

The initial approach to pudendal neuralgia should be conservative. Surgical decompression is the treatment of choice in patients with likely nerve entrapment, but determining the likelihood and extent of entrapment is a process. First, time must be spent trying to identify and address the factors causing pain, and trying to break the vicious cycle that occurs when neuropathic pain causes spasm of the pelvic floor muscles, which in turn leads to increased compression of the nerve and subsequent increase in pain level.

While there are no official treatment algorithms, we have found – based on available data and our experience in treating more than 500 patients with pudendal neuralgia – that particular therapies can lead to marked improvements for many patients.

For some patients, especially those in whom bicycling or specific exercises initially caused the pain, avoidance of activities that worsen the pain, and other lifestyle modifications, can be helpful. Medical therapy with analgesics/pain management (such as oral pregabalin) and muscle relaxants also may be helpful for some patients. We have tried all kinds of muscle relaxants and have found that a vaginal suppository combining diazepam and baclofen is superior. The most important treatment modality, however, is pelvic floor physical therapy. Such therapy is key because many patients have significant muscle spasm and subsequent muscle shortening. Therapists who are specially trained to work with pelvic floor muscle dysfunction can address these and other problems largely through various hands-on techniques, exercises, stretching, and patient education.

Botulinum toxin A (Botox) injections also are often a key part of therapy for patients with significant muscle

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NVA Celebrates 20th Anniversary

By Phyllis Mate, NVA co-founder and President

In August 1994, five women sat on a porch drinking lemonade, wondering whether they could be the only women in the world with constant burning pain in the vulva. They shared several distressing symptoms including pain with sexual intercourse, sitting for extended periods, gynecological exams and wearing fitted pants. Most had visited more than five doctors seeking a diagnosis and treatment. Cortisone topicals prescribed by their gynecologist or dermatologist didn't work. Fortunately, some had recently found vulvovaginal specialist, Stanley C. Marinoff, M.D., or his colleague, dermatologist Maria Turner, M.D., both of the Washington, DC area, who had written an article on a condition called Vulvodynia ("burning vulva"). As it turned out, Marinoff and Turner were pioneers in the field and didn't think that women with pain in the external genitalia were imagining their symptoms. Dr. Turner worked at the National Institutes of Health (NIH) and was not in private practice, so she suggested that neurologist Helene Emsellem, M.D., might be able to treat the pain aspect of the condition. Dr. Emsellem, realizing how isolating it was to suffer from an unknown disorder, asked her five vulvar pain patients if they wanted to speak with each other. By bringing us together, Dr. Emsellem was instrumental in the creation of the National Vulvodynia Association (NVA).

Over the next five years that small group of women, with an initial donation of $5,000 from our wonderful benefactor, Mona Schlossberg, and the guidance of Dr. Marinoff, helped to establish Vulvodynia as a legitimate disorder among doctors, provided information and support to thousands of suffering women and began a public awareness campaign. We had created the non-profit NVA, the only doctor-supported national vulvodynia organization that would continue to serve thousands of women and health care providers worldwide.

Now that NVA has reached its 20th anniversary, we want to take a moment to reflect on some past accomplishments and acknowledge the people responsible for them. We have successfully put Vulvodynia on the map at several Institutes at the NIH as well as on Capitol Hill. In 1998, after contacting women's health advocate Senator Tom Harkin (D-Iowa), and especially his then chief of staff, Peter Reinecke, strong language promoting Vulvodynia research was included in the NIH budget appropriations report for the first time. NIH, with encouragement from Capitol Hill, has held three Vulvodynia conferences for health care providers and researchers in the past 14 years and started funding Vulvodynia studies in 2000. During the same time period, NVA made significant progress in obtaining media coverage by issuing press releases, which generated stories on Vulvodynia in hundreds of magazines and newspapers and led to segments on TV shows such as Oprah, Dr. Oz, 20/20 and Sex in the City. If you Google Vulvodynia today, there are hundreds of websites to choose from, which was certainly not the case in the late 1990s.

As one of the original group of five, I am both amazed and grateful for how far we've come. Although I am the only co-founder still active, I have not forgotten the contributions of the other four: Harriet O'Connor, Support Director; Rhonda Brunell, Treasurer; Marjorie Veiga, Public Relations and Webmaster; and Jacqueline Smith, our first Executive Director. Thanks very much to each of you. There were also many others who volunteered their time. I don't have the space to name everyone, except to acknowledge our very first part-time staffer, LuEllen McCormack, who managed the growing NVA database and always had a smile on her face and a tiny poodle in her purse. Over the years, we've had many terrific women (and one man) on our volunteer Board of Directors. I'd just like to mention Andrea Hall, J.D., who spent more than a decade laying out and proofing all our newsletters with me, and Maurice Kreindler, Treasurer, who has taken excellent care of our financial assets for the last 15 years.

Our Board is most proud of the many researchers we have managed to fund, enabling some to obtain grants of $1 million or more from the NIH or other sources.

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This has been our greatest gift to women who currently have Vulvodynia and those who will come afterwards. A dedicated group of researchers from all over the world has been focused on designing studies intended to shed light on the cause(s) of Vulvodynia. My hope is that one or two major findings are only five to ten years away. That knowledge should jump-start a race to find novel effective treatments for Vulvodynia sufferers, and eventually lead to a cure for some women. Meanwhile, the NVA-funded National Vulvodynia Registry has been underway for four years, with the participation of at least eight doctors from across the US. These doctors submit information on willing Vulvodynia patients to a centralized database, which can then be analyzed by experts. This information includes which treatments have been tried on different types of vulvodynia patients and how successful they have been. Thanks to a grant NVA obtained from the Patty Brisben Foundation, for the past 18 months the Registry's scope has expanded to collect data on postmenopausal as well as younger women.

On the educational front, we developed and published four guides for patients, the most popular of which is our self-help guide. Our website, www.nva.org, is a gold mine of information for patients, health care professionals and the public. The patient tutorial on our site is an all-in-one tool for women to use to educate themselves before or after they receive medical help. With assistance from our dedicated medical advisory board, former executive director Christin Veasley developed the only online CME-accredited tutorial on the diagnosis and treatment of Vulvodynia for health care providers. This tutorial has been very successful, with approximately 1,400 providers completing the post-test every three months.

In April 2014, NVA was joined by Lisa Goldstein, M.S., our new executive director, who is a 20-year veteran of the American College of Obstetricians and Gynecologists. Lisa's current major task is overseeing the revision of our website, which has been generously funded by Purdue Pharma, a long-time supporter of NVA projects. (Our only other full-time staffer is our conscientious administrator, Gigi Breechen, who has managed the database, fulfilled thousands of mailings and responded to countless inquiries for almost 11 years.) This past spring, our Board of Directors invited Andrew Goldstein, M.D., director of the Centers for Vulvovaginal Disorders and the Immediate-Past President of the International Society for the Study of Women's Sexual Health, to join our Medical Advisory Board. We are confident that both Goldsteins (no relation) will continue to be strong advocates for women with Vulvodynia.

Our Hope for the Future

While NVA continues to fund pilot research on Vulvodynia, we are optimistic that NIH and other institutions will increase their funding in this area. We are aware that Vulvodynia is associated with other conditions, such as interstitial cystitis and irritable bowel syndrome. It may turn out that some research findings on interstitial cystitis, or another chronic pain syndrome, also apply to Vulvodynia. If that is the case, it could save Vulvodynia researchers time and effort, and hasten the process of determining the cause(s). NVA hopes that once we have this valuable information, some pharmaceutical companies will spend their resources on developing novel treatments. Pharmaceutical companies should be encouraged to make that investment after examining the results of epidemiology studies that estimate 12-16 million women in the US alone experience Vulvodynia or its symptoms. By NVA's 40th anniversary, we hope to report that at least one pharmaceutical company has life-changing news for most of us. Once we have treatments that have been scientifically proven to relieve Vulvodynia and there is an effective treatment for everyone, the NVA can change its main focus to prevention. I believe that will happen in our lifetimes. ■

CORRECTION: In the article, "Why Oral Contraceptive Pills Can Cause Vulvodynia," in the previous issue of *NVA News*, Dr. Jill Krapf's last name was spelled incorrectly. Dr. Krapf was a co-author of this article. The NVA apologizes for this error.

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spasm. In our practice, we administer approximately 200 units in 20 injections using a pudendal nerve block needle, under anesthesia. Not only does the treatment aid in muscle relaxation (thus increasing the patient's tolerance to physical therapy), it also helps to differentiate between pain caused solely by muscle spasm, and pain caused by nerve injury and muscle spasm.

While patients that do not have neuralgia, whose pain is caused solely or almost solely by muscle spasm, will benefit significantly more from Botox injections, some patients with pudendal neuralgia benefit from occasional, repeated Botox treatment in lieu of surgical decompression therapy. Many of our patients have been receiving Botox injections every 3-4 months, for instance.

Similarly, many other patients get significant pain relief from CT-guided injections of the nerve. While an initial CT-guided injection of anesthetic and steroid serves both diagnostic and therapeutic roles, a second and third injection can be performed to deliver more steroid and anesthetic into the pudendal nerve canal (Alcock's canal) in a patient who responded to the first injection but whose pain has returned. Again, these injections must be performed by an experienced interventional radiologist in a CT scanner.

Injections are offered 6 weeks apart and some patients have significant pain relief for 4-5 months, or even longer, after CT-guided nerve blocks. Patients who experience long-term pain relief from CT-guided blocks will not be offered decompression surgery. One of our patients, for instance, is receiving nerve blocks every 8 months as part of her treatment.

Surgical Decompression

If patients do not have sufficient pain relief from conservative therapies (relief that enables them to return to normal daily function), surgical decompression of the nerve is indicated. An estimated 30-40% of all patients with pudendal neuralgia will benefit from surgery.

The transgluteal approach (through the buttocks) appears to be the most effective technique, allowing the best visualization of the pudendal nerve and the greatest extent of decompression along the length of the nerve. The main concern with this approach has been the required transection of the sacrotuberous ligament and the possible impact on stability of the sacroiliac joint. In our practice, however, we have made several modifications to the approach that minimize these concerns and, we believe, are improving recovery and outcomes.

The patient is placed in a prone jackknife position, and the electrodes of a NIMS monitor[1] are placed in the anal sphincter. An incision of approximately 7-10 cm in length is made across the gluteal region overlying the sacrotuberous ligament. The gluteus muscles are spread, with muscle fibers separated longitudinally, and once the ligament is reached, it is transected at its narrowest point. The pudendal nerve then can be identified immediately below the ligament with use of a surgical microscope and the NIMS. When the surface of the nerve is touched, we are alerted by the NIMS monitor (part of the nerve runs to the anal center). In some patients, the pudendal nerve may actually be attached to the anterior surface of the sacrotuberous ligament.

The nerve is then decompressed along its entire length. Neurolysis is performed along each of the nerve's branches until the nerve is completely free. In our practice, we most often find the nerve entrapped between the sacrospinous and sacrotuberous ligaments, which form a sort of "V" in the pelvis. Because the sacrospinous ligament does not serve any anatomic purpose, I transect the ligament so that I can transpose the pudendal nerve anteriorly to give it more room.

Repair of the sacrotuberous ligament was not traditionally performed as part of the transgluteal approach, but we believe that repair is important for stability of the sacroiliac joint. In other modifications to the traditional approach, we wrap a piece of NeuraGen Nerve Guide[2], a nerve-protecting sheath made of collagen, around the nerve to prevent the formation or reformation of scar tissue. To promote nerve healing, we then cover the nerve with platelet-rich plasma prepared from the patient's own blood.

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Before closure, we also place a pain pump catheter along the course of the nerve. We believe that infusion of bupivacaine for 10-20 days postoperatively decreases the risk of central sensitization to pain and allows patients to be more mobile after surgery, which we encourage. It also may reduce the risk of scar formation. When neuropathic central pain is believed to be a significant problem, as it often is in patients whose nerves have been injured by surgical mesh, we also administer ketamine. An infusion of this previously used anesthetic can erase or reverse the troubling phenomena of central sensitization to pain.

Outcome data from France show that approximately 30-40% of patients are pain-free after surgical decompression, with another 30% reporting improvement in pain and 30% reporting no change in their pain levels[3]. At our institution, we have found that 70% of patients who undergo transgluteal surgical decompression have at least a 20% improvement in pain. Within this broad category are a significant number of patients who are pain-free, and many who report improvements of 50% or more.

Interestingly, we have found that outcomes are similar among our much smaller number of "re-do" surgical patients. Thus far we have performed approximately 20 such transgluteal procedures – 17 on patients who had re-scarring of the nerve after surgery performed at other institutions, and 3 who had surgery many years ago in our practice, before we were able to optimally visualize the entire nerve and made modifications to improve the procedure. Just as with our first-time surgeries, approximately 70% of patients who underwent a second procedure had at least a 20% improvement in pain.

In all cases, the pudendal nerve recovers slowly, especially when it has been entrapped and injured for a long time, and improvements in pain often do not occur until about four months after surgery. Improvement typically continues for some time, up to 18 months after surgery. Patients may still have pain related to muscle spasms after surgery, so continued physical therapy and/or more Botox injections are often beneficial. Patients must also, of course, continue to avoid any offending factors or activities.

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therapeutic target. In addition, pain often gets worse due to less participation in physical activities, because aerobic exercise, as well as strengthening and stretching exercises, have been shown to relieve pain. For many, simply increasing daily activities is helpful before actually starting to exercise. Many individuals try to do too much too soon, leading to exacerbated pain. It is better for individuals to do the same amount of an activity each day, regardless of how they feel. For a symptom management program that provides a self-care approach, go to www.fibroguide.com. This website is designed for individuals with fibromyalgia, but can be helpful to anyone with a centralized pain state.

Selected References:

1. Arnold LM, Hudson JI, et al. Family study of fibromyalgia. *Arthritis Rheum* 2004;50:944-52.
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4. Buskila D, Atzeni F, et al. Etiology of fibromyalgia: the possible role of infection and vaccination. *Autoimmun Rev* 2008;8:41-3.

(A full set of references is available by request. Please email admin@nva.org or call 301-299-0775.) ■

Chronic Pain and Depression

On May 28-29, 2014, Lisa Goldstein, NVA Executive Director, attended the annual National Institutes of Health Pain Consortium Symposium in Bethesda, Maryland. The symposium, *Biological & Psychological Factors that Contribute to Chronic Pain,* included presentations on the association of chronic pain and depression, sleep disorders and neuro-immune function, as well as sessions on comorbid pain disorders. What follows is a summary of the three sessions on chronic pain and depression.

Daniel Clauw, M.D., University of Michigan

Dr. Clauw provided an overview of chronic pain and depression. He highlighted that almost all studies find that chronic pain increases the risk of depression, and that some also suggest that people with a history of depression are at a higher risk for chronic pain. He noted that even though depression is a risk factor for development of subsequent pain, it is not one of the most significant risk factors. The risk is very modest from a relative standpoint in population-based studies, but is high from an absolute standpoint, because depression is such a common disorder. In studies using the same methodology, many other factors are typically stronger predictors of subsequent pain than baseline depression. These other factors include early life stressors, prior pain, prior somatic symptoms, and poor sleep. In addition, Clauw discussed how individuals with centralized pain, originating from or amplified by the Central Nervous System (CNS), are more likely to have comorbid depression than those with nociceptive (peripheral) pain. This is due in part to an imbalance in certain neurotransmitters that control pain and sensory sensitivity and also control mood, sleep, memory, and alertness. (See Clauw article, p. 1.) He concluded that while depression is a common co-morbidity in chronic pain, we have historically focused too much on the affective component of depression. To achieve the biggest impact on the lives of chronic pain patients, we should instead focus on the CNS-mediated symptom cluster of multifocal pain, fatigue, memory problems and mood disturbances associated with centralized pain. This will help to determine the underlying origin of the pain condition and corresponding treatment(s).

Mary Davis, Ph.D., Arizona State University

Dr. Davis presented on the relationship between positive affect (emotion) and chronic pain. Data suggest that the presence of positive affect predicts positive outcomes better than negative affect, e.g., sadness, predicts negative outcomes in pain patients. Therefore, a clinical approach to consider for chronic pain patients is one that focuses on actively promoting positive emotions (or moods) rather than on efforts to reduce negative emotions. This concept is supported by a study recently published by Davis and colleagues[1]. In this study, 110 women with fibromyalgia and/or osteoarthritis were exposed to a stressful interview and then randomly assigned to either view a positive or neutral mood-inducing film clip. Among both depressed and non-depressed patients, the level of pain experienced increased during the stressful interview. After viewing the film clip, the mood of the non-depressed subjects improved, regardless of whether they saw a positive or neutral clip. However, the mood of the depressed women improved only if they viewed the positive clip. The change in mood was associated with reduced pain among depressed patients who viewed the positive film clip. This suggests that interventions to improve the mood of chronic pain patients with depression may help to reduce pain after women experience stressful situations.

Davis also described mindfulness-based therapy, a specific intervention designed to enhance positive affect, coping skills and resilience. This therapy uses cognitive-behavioral therapy methods with newer psychological strategies such as mindfulness meditation that focuses on becoming aware of thoughts and feelings and accepting them, but not attaching or reacting to them. Davis and colleagues showed that mindfulness therapy was more effective at reducing joint tenderness than cognitive-behavioral therapy in a 2008 study of 144 individuals with depression and rheumatoid arthritis[2]. In a more recent study comparing mindfulness training to general health tips that were both delivered online to 79 fibromyalgia patients, mindfulness training was more effective in improving patients' positive affect,

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enjoyment of family and pain coping skills than general health tips[3]. Although the effects were small, the results suggest the potential for improving pain by delivering low-cost and widely accessible psychological interventions online.

Courtney DeVries, Ph.D., Ohio State University

Dr. DeVries concluded the session on depression and chronic pain by discussing her study on the effects of social interaction on allodynia (pain from normally non-painful stimuli) and depression in mice, and offering a possible physiological explanation for Davis' findings. DeVries noted a reduction in allodynia and depressive behaviors in mice housed in pairs before nerve injury compared to mice reared alone. In addition, when the mice were paired after injury, there was a significant decrease in allodynia. This suggests that prior exposure to stress may alter the response to a subsequent nerve injury. It also shows that, even once pain is established, it is possible to add a positive stimulus to decrease the amount of pain experienced. Examining this from a physiological perspective, DeVries explained that nerve injuries cause neuroinflammation, which in turn causes pain and promotes the development of depressive-like behavior. Stress exacerbates these conditions through a corticosteroid-mediated pathway and social interaction relieves them by reducing the neuroinflammation associated with the nerve injury. This relationship may apply to other forms of neuropathic pain.

(***Ed. Note****:* Archived webcasts of the entire meeting, divided into Day 1 and Day 2, are available at: https://videocast.nih.gov/Summary.asp?File=18461&bhcp=1 and https://videocast.nih.gov/Summary.asp?File=18462&bhcp=1, respectively.)

References

1. Davis MC, Thummala K, Zautra AJ. Stress-related clinical pain and mood in women with chronic pain: moderating effects of depression and positive mood induction. *Ann Behav Med* 2014;48:61-70.
2. Zautra AJ, Davis MC, Reich JW, et al. Comparison of cognitive behavioral and mindfulness meditation interventions on adaptation to rheumatoid arthritis for patients with and without history of recurrent depression. *J Consult Clin Psychol* 2008;76:408-21.
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