tarium to aide in the laparoscopic treatment of fibroids. As noted in the article, we evaluate the cul-de-sac and identify those patients with adequate cul-de-sacs that allow us to deliver the uterus into the vagina.

Secondly, we have not had any difficulty in removing myomas that are on the anterior surface of the uterus, and indeed, in 1 patient we removed the myomas that were identified laparoscopically. This technique is especially indicated when multiple small submucosal myomas are present.

In addition, we attempt to map myomas using a pelvic magnetic resonance imaging (MRI) that gives us excellent insight into the exact number and location of the myomas, and in our experience, is far better than the usual abdominal and intravaginal ultrasound.

As far as the interpretation of postsurgery urinary retention, I believe that it is a reflex spasm secondary to the retroversion of the uterus. In all cases this urine retention was self-correcting. In addition, antibiotic prophylaxis was administered in all patients.

In summary, I believe that the laparoscopic assisted vaginal myomectomy is a technique that is appropriate for women who have a number of uterine myomas, especially deep intramural myomas with at least 1 large sentinel myoma. The sentinel myoma is removed laparoscopically; the culpotomy is then performed; the uterus is delivered into the vaginal area through culpotomy and the remainder of the myomas are removed.

Utilizing this technique, a precise 2 and 3 layer suturing can be carried out.

Thank you for your kind comments and your insightful evaluation.

Sincerely,
Herbert A. Goldfarb, MD
Montclair Reproductive Center, 29 The Crescent, Montclair, NJ 07042. Telephone: (973) 744-7470, Fax: (973) 743-1274, Email: HGoldfarb@HGoldfarb.com

---

Re: Current Concepts of Pelvic Congestion and Chronic Pelvic Pain

To the Editor:

I read with great interest the excellent paper by Dr C. Paul Perry, (Current concepts of pelvic congestion and chronic pelvic pain. *JSLS*. 2001;5:105-110). It is true that association between pelvic congestion (pelvic varicosities) and chronic pelvic pain is more of a diagnosis of exclusion (absence of other findings) rather than the primary finding we are looking for. The data on the incidence of pelvic varicosities in patients with chronic pelvic pain is scarce and often contradicts our assumption of such an association. Kresch et al found during laparoscopy that out of 100 women suffering from chronic pelvic pain 3% had large pelvic veins, but among 50 asymptomatic women undergoing tubal ligation large pelvic veins were found in 15%.1 The valvular insufficiency theory cannot be supported by the fact that pain due to pelvic varicosities disappears after menopause. If valvular incompetence is a primary reason, one would expect pain to increase with age. On the other hand, we all have been in the position of, during laparoscopic examination of patient with chronic pelvic pain, not finding endometriosis, occult or overt hernias, or adhesions, yet looking at the pelvis we were convinced that this patient had pelvic pain because the pelvis was congested. The congestion did not have to take the form of pelvic varicosities. It could appear as hundreds of dilatated small arteries and venules visible through the peritoneum.

Pelvic congestion is not a cause of pain, but rather the symptom of the condition maintaining the pain. I would like to offer a different explanation for the mechanism of chronic pelvic pain in some women, especially those with diffuse pelvic congestion.

Some women with chronic pelvic pain suffer from a form of reflex sympathetic dystrophy (RSD) and sympathetically maintained pain (SMP) involving the pelvis. For them, minimal endometriosis, occult hernias, and pelvic adhesions are the triggers rather than the cause of pain. Chronic pain is a complex system of maladjustments within the body and the nervous system and in particular the autonomic nervous system. The autonomic nervous system is the operating system of our body; it runs programs (routines) to
maintain homeostasis within our body despite changing external and internal environments. It is an interface between higher neurologic functions (thought, conscious and unconscious emotions) and effectors on the periphery (mechanical, immune system, hormonal system, circulation, etc.) In chronic pain, malfunction occurs within the autonomic nervous system, and new faulty routines are developed which instead of mitigating the pain, maintain it. Because of the unique position of the autonomic nervous system as an interface, occurrence and management of pain can be affected by emotional status, psychotherapy, psychopharmacology, surgical intervention, nerve ablations, acupuncture, physical therapy, and many other treatment methods. The format of this letter prevents me from fully describing this concept, but it certainly is worth looking into in the future.

Sincerely,
Thomas I. Janicki, MD
Department of Reproductive Biology
Case Western Reserve University
University Suburban Health Center
1611 South Green Rd #216
Cleveland, Ohio 44121
E-mail: tij@att.net

References:
1. Kresch, et al. Laparoscopy in 100 women with chronic pelvic pain. Obstet Gynecol. 1984;64(5):672-674.
2. Janig W. Systemic and specific autonomic reactions in pain: efferent, afferent and endocrine components. Euro J Anaest. 1985;2:319-346.
3. Baron R, Levine JD, Fields HL. Causalgia and reflex sympathetic dystrophy: does the sympathetic nervous system contribute to the generation of pain. Muscle Nerve. 1999;22: 678-695.

Author's Response:
I appreciate Dr Janicki's comments and find myself unable to disagree with his theory that pelvic varicosities are an effect and not a cause of chronic pelvic pain. The current theories of etiologies range from estrogen dependent neurotransmitter production, incompetent pelvic vein valves, orgasmic dysfunction, psychosomatic dysfunction, and iatrogenically induced varicosities.

His theory is that the pain from pelvic congestion is actually a type of reflex sympathetic dystrophy, which is producing the venous dilatation. The varicosities are, in his opinion, a result of the sympathetically maintained pain. Although this concept is just as valid as the previously unsubstantiated etiologies, several facts cause me to doubt its validity.

Reflex sympathetic dystrophy (causalgia) is more accurately called complex regional pain syndrome (CRPS). Hyperplasia and vascular changes are components in common with pelvic pain congestion syndrome. Most of what we know about CRPS comes primarily from causalgic pain in the extremities and has the following characteristics: (1) it is preceded by some noxious event or nerve injury, (2) spontaneous pain or hyperplasia is not limited to a single nerve distribution and is out of proportion to the inciting event, (3) there are blood flow changes in the distal limb, edema, or sudomotor abnormalities occur, (4) it is a diagnosis of exclusion.

It is unlikely that CRPS (if involved at all) is the only cause of pelvic congestion. The clinical observation that these patients benefit from oral medroxyprogesterone acetate and do not respond to neuropathic medications speaks against this theory. Also, we know that hysterectomy is very effective for pain due to pelvic congestion. If the CRPS were etiologic, surgery would exacerbate the pain.

C. Paul Perry, MD
Pelvic Pain Center
Brookwood Women's Medical Center
2006 Brookwood Medical Center Dr.
Birmingham, AL 35209
Telephone: (205) 877-2950
Email: pelvicpain@aol.com

References:
1. Birk F, Handwerker HO. Complex regional pain syndrome: how to resolve the complexity? Pain. 2001;94:1-6.