CME Approved Article

Basic Neuroanatomy for Understanding Pelvic Pain

Robert M. Rogers, Jr., M.D.

Objectives

To know which stimuli cause somatic pain and which result in visceral pain.
To explain the concepts of intermingling, cross-talk, and viscerosomatic convergence.
To understand the true nature of dermatomes.
To describe the five pathways that transmit nociceptive signals out of the pelvis.
To appreciate the complexities of individual perceptions of pain and the importance of patient education.

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Pretest Questions

1. ***Why is visceral pain dull, diffuse, and poorly localized?***
2. ***How can somatic trigger points mimic visceral pain?***
3. ***What are visceral nociceptors?***
4. ***What stimuli cause somatic pain?***
5. ***What stimuli cause visceral pain?***
6. ***Why are dermatomes not discrete sensory bands on the abdominal wall?***
7. ***How do cross-talk and viscerosomatic convergence amplify the sensations of pelvic noxious stimuli?***
8. ***What are two examples of intermingling of afferent fibers in the female pelvis?***
9. ***Where do the afferent (sensory) fibers synapse?***
10. ***How many distinct neuropathways transmit noxious stimuli out of the female pelvis?***

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Case Reports

Patient No. 1

A 52-year-old, para II woman was in her usual state of good health when she had a laparoscopic-assisted vaginal hysterectomy 5 years ago. She was discharged within 24 hours of the procedure, but severe lower abdominal pain resulted in rehospitalization for 9 days. Gastrointestinal, general surgical, neurosurgical, and neurologic consultations and evaluations were all negative.

The patient described the pain as being sharp, waxing and waning in the right lower quadrant. It was tolerable when she stood but was very irritating when she was sitting. The pain radiated laterally to the right hip, inferiorly to the upper anterior aspect of the right leg, and medially and deep toward the left lower quadrant. Physical examination elicited a focus of acute sharp tenderness in the right lower quadrant approximately 3 cm medial to the right anterior superior iliac spine, as well as another focus approximately 3 cm inframedial from the first spot. Each of these areas was injected with several milliliters of 0.5% bupivacaine plus dextrose to the trigger points. After several minutes, neither palpation of the areas nor the sitting position caused pain or tenderness in the right lower quadrant.

Approximately 8 hours after these injections the pain gradually returned. Two weeks later the areas were reinjected with a total 6 ml of a solution of 9 ml 0.5% bupivacaine HCl and 1 ml triamcinolone diacetate suspension 40 mg/ml, and again the right lower quadrant pain was alleviated. Two weeks later pain and tenderness had not returned. During an annual examination 1.5 years later the patient reported that the discomfort was much improved, and she had resumed all her normal activities.

This patient had several trigger points in the right lower quadrant of the anterior abdominal wall. Just as true deep pelvic pain can be referred to the anterior abdominal wall, such trigger points can refer pain back to the pelvis. This phenomenon may occur at the level of the posterior horn of the spinal cord as well as in the cerebral cortex. Because of prolonged, chronic irritation to afferent sensory nerve fibers, these pain pathways can be altered, causing a conditioned, preprogrammed response at the level of the spinal cord, as well as preconditioned neuroanatomic pain mapping in the cortex.

Neurologic pathways that transmit noxious stimuli out of the female pelvis are poorly understood by many gynecologists. This article should improve understanding of and provide insight into the confusing and complex afferent neuroanatomy of the female pelvis.

Patient No. 2

A 33-year-old, para II woman complained of right-sided deep dyspareunia present for the past several

From Women's Clinic, Ltd., West Reading, Pennsylvania.

months and worsening. Her periods were normal without dysmenorrhea, postcoital spotting, or pain during normal activities. She was athletically fit, running 4 miles at least five times a week. She had no history of sexually transmitted diseases, and her husband was her only sexual partner.

Her abdomen was benign, without masses or tenderness. The vagina was clean and well supported, the cervix was normal and not tender. The uterus was anterior, of normal size, and nontender. Adnexal examination was negative, without tenderness or masses, confirmed by rectovaginal examination. Further palpation of the right pelvic sidewall, particularly on the obturator internus muscle approximately 3 cm inferior to the ischial spine, caused acute tenderness similar to her deep dyspareunia. Vaginal probe ultrasound study showed no abnormalities.

The patient was advised to walk and not run for the next several weeks and to take nonsteroidal antiinflammatory drugs. She returned 4 weeks later stating she no longer experienced deep dyspareunia.

This case illustrates how irritation to the musculature and parietal fascia in the pelvis can easily be confused with deep pelvic visceral pain. In this acute situation, cross-talk and viscerosomatic convergence confuse both patient and clinician concerning the true etiology of pelvic pain.

Visceral Pain

Visceral pain, classically deep within the abdominal or pelvic cavities, is characteristically diffuse, dull, and poorly localized.[1] These sensations are associated with visceral responses such as nausea, anxiety, tachycardia, hypotension, and diaphoresis. Surprisingly, the source of such discomfort may be somatic trigger points[2] within the body wall, extremities, pelvic muscular basin, and perineum, as well as deep pelvic viscera themselves. Noxious stimuli in these areas can result from irritation to afferent fibers in these regions, from scar tissue or adhesions, neuromas, occult hernias, and inflammatory processes such as infection and endometriosis externa. Other irritating stimuli within the pelvis are benign and malignant tumors, vascular congestion, uterine prolapse and pelvic floor dysfunction, spasm within the muscular pelvic basin, and various vulvovaginal disorders. More commonly recognized causes of pelvic pain are irritable bowel syndrome, chronic constipation, dysmenorrhea, chronic pelvic inflammatory disease, urethral syndrome, detrusor instability, and interstitial cystitis. In addition, many patients have coexisting psychologic and psychiatric diagnoses.

How pain is caused by these irritants is not clear; however, it can involve mechanical, electrical, and thermal irritation. Chemical irritation is caused by released substances such as prostaglandins, potassium, histamine, serotonin, and substance P. These substances can activate action potentials in the terminals of afferent pain fibers in the immediate area. Visceral nociceptors are free, unmodified nerve endings attached to poorly myelinated or unmyelinated nerve fibers called A-delta and C fibers. They mediate both somatic and visceral noxious stimuli.

Body walls, extremities, and parietal peritoneum are sensitive to mechanical trauma, as well as to thermal and chemical stimuli.[3] Visceral peritoneum, mesentery, gastrointestinal tract, ureters, bladder, and urethra are all very sensitive to traction, rapid distention, smooth muscle spasm, and ischemia. These same structures are relatively insensitive to cutting, burning, chemical irritation, and mechanical crushing. However, inflammation in the mucosa of these organs significantly increases their sensitivity to touch, pressure, and chemical stimuli.

Although it is insensitive to touch or pressure, the fundus of the uterus is highly sensitive to myometrial contractions. Fallopian tubes and ovaries are primarily sensitive to traction and distention. Distention of the cervix gives rise to painful sensations referred to the lower abdomen, although pinching of the cervix evokes little pain sensation. Vaginal epithelium is relatively insensitive to mechanical irrigation, yet is very sensitive when inflamed, as mentioned.

Basic Neuroanatomic Concepts

To understand the dull and diffuse nature of pelvic pain, it is necessary to understand several neuroanatomic concepts: intermingling of afferent (sensory) fibers; intercommunication among nerve plexuses; the true nature of dermatomes; cross-talk; viscerosomatic convergence; larger areas serviced by visceral afferentes; and individual variations of anatomy and neurophysiology.

Intermingling of afferent fibers refers to different pathways that allow nociceptive stimuli in one area in the pelvis to exit the pelvis toward the spinal cord. For example, a noxious stimulus on the anterior wall of the rectum may irritate afferent fibers that exit the

pelvis through the middle rectal nerves to the inferior hypogastric plexus, and from there up the hypogastric nerves to the superior hypogastric plexus. The same noxious stimulus may exit the pelvis by way of the superior rectal nerves that feed back through the inferior mesenteric plexus to the spinal cord (Figure 1).[4] Various irritating stimuli in the pelvis can be picked up by certain afferent fibers that exit the pelvis through completely different routes.

Some visceral nerve plexus in the abdomen and pelvis can actually intercommunicate through fibers between them. To continue the example, there are intercommunicating fibers between the superior hypogastric plexus and inferior mesenteric plexus (Figure 2). The major abdominopelvic visceral nerve plexus, from superior to inferior anatomically, are celiac, superior mesenteric, aorticorenal, ovarian, inferior mesenteric, superior hypogastric, hypogastric, and inferior hypogastric to the vesical, uterovaginal, and middle rectal (Figure 3). Many variations in bilaterality, shape, size, location, and number of these plexus and ganglia have been observed.

The transverse cutaneous segment that is innervated by one somatic afferent from one spinal nerve is called a dermatome. It is traditionally taught that dermatomes are innervated by only one spinal nerve. In reality, however, each dermatome has sensory contributions from as many as four other spinal nerves (Figure 4). Thus they have a great tendency to overlap one another in cutaneous spatial distribution.[3] Referral is the process by which visceral pain is localized to a dermatome on the anterior abdominal wall. The mechanism of referred visceral pain can be explained by interaction of visceral and somatic afferent nerves in the spinal cord, which is better explained in discussions of cross-talk and viscerosomatic convergence that follow.

Because afferent fibers are poorly myelinated or not myelinated at all, and because they come into close physical proximity at various stages during their courses back to the spinal cord, a strong electrical action potential in one nerve can actually instigate action potentials in other nerves, even though they are not affected by the irritating stimulus. This phenomenon

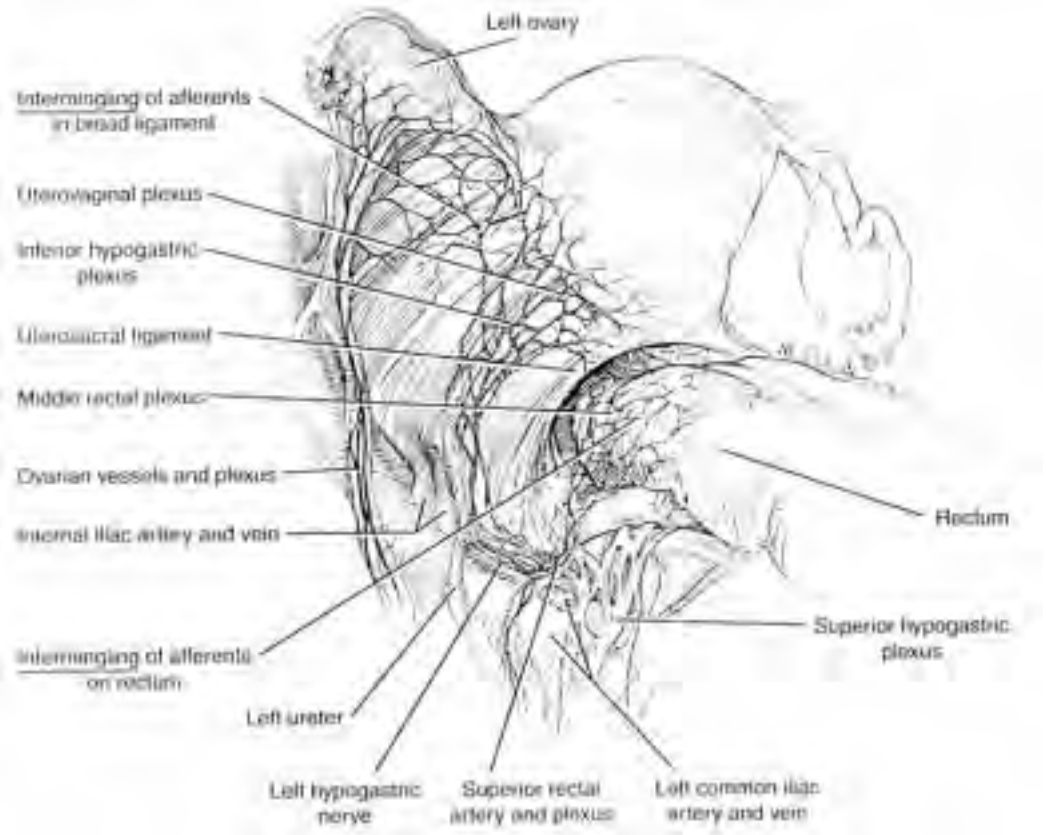

FIGURE 1. Intermingling of visceral nerves on pelvic viscera. (From reference 4, with permission.)

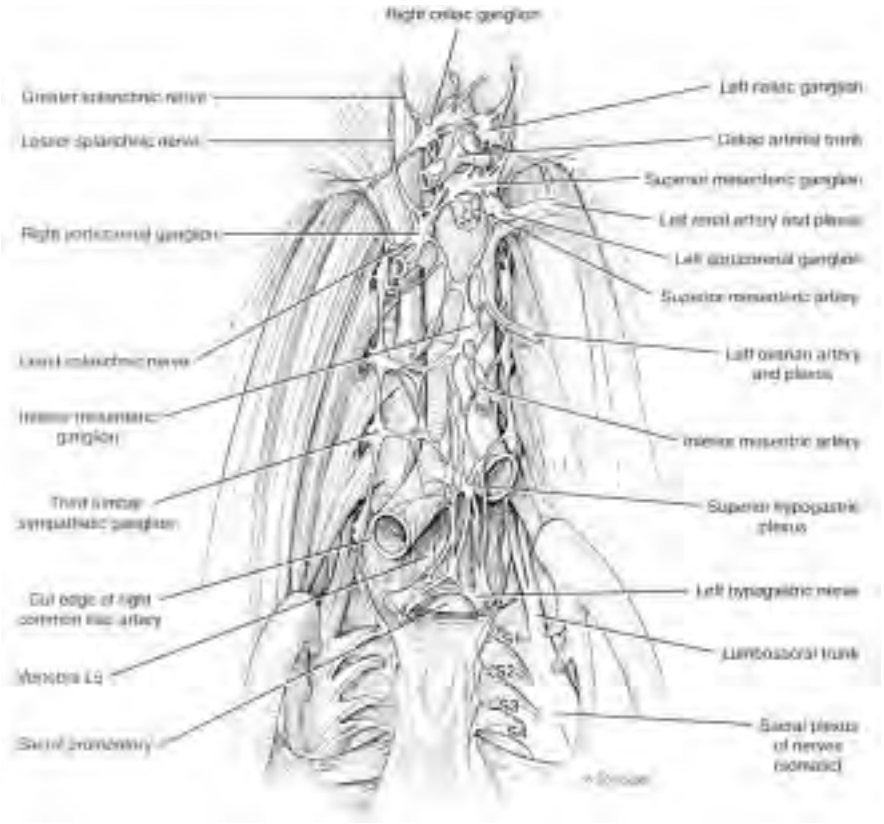

FIGURE 2. Visceral nerves in the abdomen. (From reference 4, with permission.)

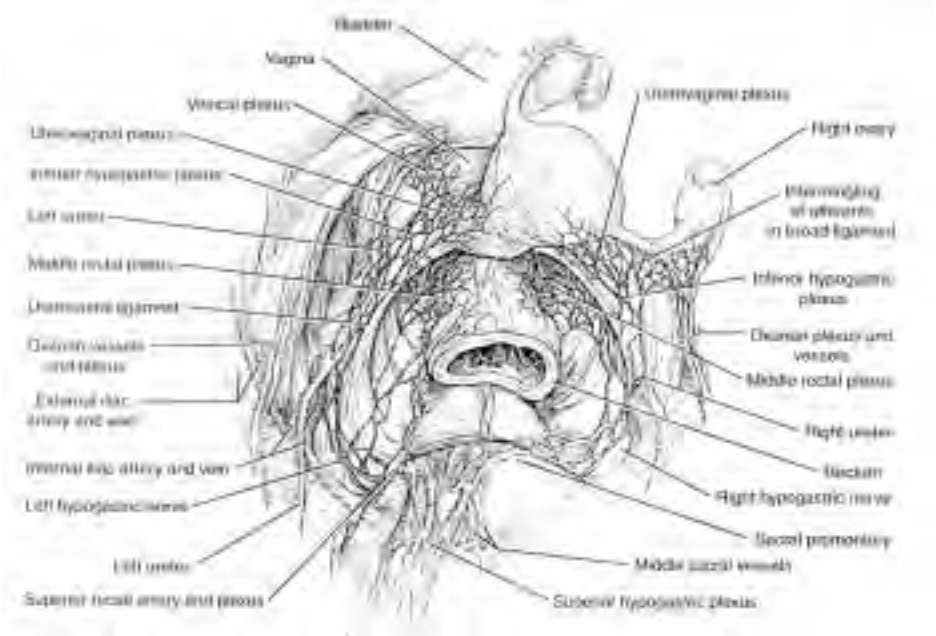

FIGURE 3. Visceral nerves of the pelvis. (From reference 4, with permission.)

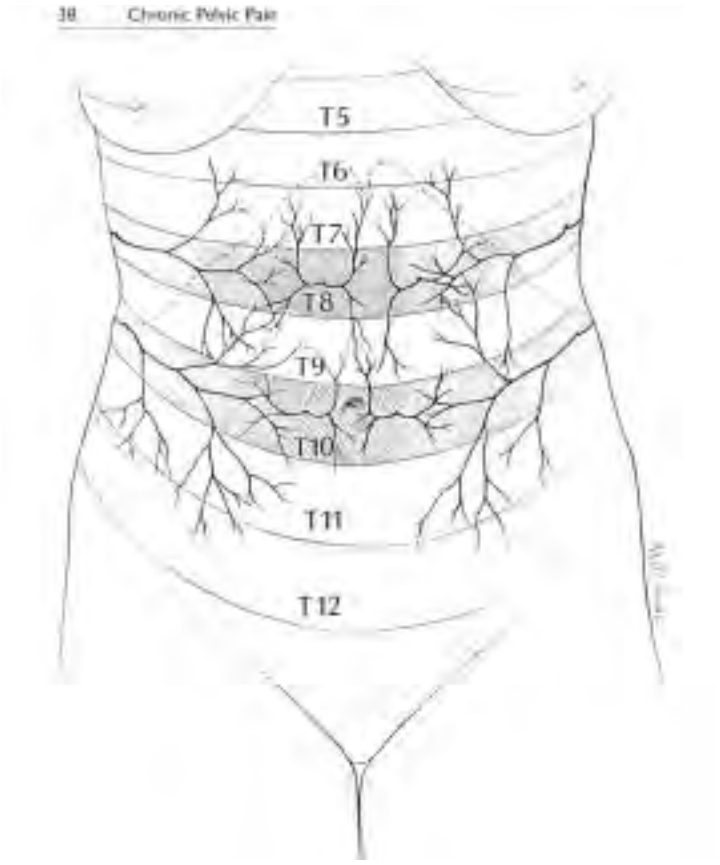

FIGURE 4. Anterior abdominal dermatomes. (From reference 4, with permission.)

is called cross-talk.[5] These nerves are very close to one another in several anatomic areas—in pelvic plexus of nerves, posterior or dorsal root ganglia leading to the spinal cord, and posterior horn of the spinal cord where somatic and visceral afferent fibers converge to synapse on second-order neurons. Posterior or dorsal root ganglia contain cell bodies from peripheral afferent (sensory) nerves, both somatic and visceral. These cells are known as unipolar neurons since each has only one peripheral process.

Only 2% to 7% of all afferent fibers passing through each dorsal root ganglion are visceral afferents.[6,7] The remaining 93% to 98% are somatic. Thus cross-talk can occur in the dorsal root ganglion as well as in the dorsal horn of the spinal cord where all these afferents stimulate second-order neurons. Strong noxious visceral stimuli may stimulate many somatic afferent fibers and are thus referred onto dermatomes. Similarly, strong noxious somatic stimuli may stimulate and irritate many visceral afferent fibers, leading to a false perception of visceral pain.

In addition, each preganglionic afferent fiber entering the dorsal horn of the spinal cord synapses with as many as 15 to 20 second-order neurons. Many of these neurons receive only somatic afferent input, although some receive both visceral and somatic input. Second-order neurons distribute their signals to the various dermatomes of the lower back, lateral sidewall, and anterior abdominal wall, as well as to dermatomes surrounding the female perineum. This viscerosomatic convergence[6] is a process of sensory modulation that is also influenced by conscious and unconscious centers in higher levels of the spinal cord, midbrain, and brain cortex.

Each visceral afferent nerve fiber that travels to one dorsal root ganglion is responsible for a much larger area within abdominal or pelvic visceral tissues than a corresponding somatic afferent fiber that innervates

somatic tissues such as skin or skeletal muscle. Therefore, it is much more difficult to localize visceral pain than it is to localize somatic pain in many cases.

Finally, to confuse the practitioner in diagnosing the source of pelvic pain, each patient has her own individual variations of neuroanatomy and neurophysiology. These variations are also affected by the manner in which noxious stimuli are processed in the subconscious and conscious areas of the spinal cord and brain.

Individual, familial, and social factors also affect emotional manifestations when noxious stimuli are perceived as pain in the unconscious and conscious centers of a woman's brain. Therefore, the patient's symptoms, signs, diagnoses, and treatments may be slightly or even greatly different from those of other patients with similar complaints.

Getting Noxious Stimuli out of the Pelvis

Five neuropathways transmit signals from noxious stimuli out of the female pelvis (Figure 5): inferior hypogastric plexus to the hypogastric nerves to the superior hypogastric plexus of nerves; pelvic (parasympathetic) splanchnic nerves (nervi erigentes), many of which pass through the inferior hypogastric plexus; sacral (sympathetic) splanchnic nerves from the inferior hypogastric plexus back to sacral extension of paravertebral sympathetic ganglia; superior rectal nerves leading to the inferior mesenteric plexus of nerves; and ovarian plexus of nerves leading back to the root of the ovarian vessels overlying T10 and T11. These visceral afferent fibers are very long and travel uninterrupted to their cell bodies in dorsal root ganglia without synapsing in any plexus or ganglia. Each afferent fiber embeds free nerve endings in the wall of a viscus or blood vessel.

There are two inferior hypogastric plexuses, one each in the left and right pelvic sidewalls. Each inferior hypogastric plexus is a weblike area approximately 2 to 3 cm x 3 to 5 cm within the endopelvic connective tissue surrounding the ureter and internal iliac vessels, lateral to the rectum and upper vagina at the base of the broad ligament. This area is just lateral to each uterosacral ligament. This dense web contains many

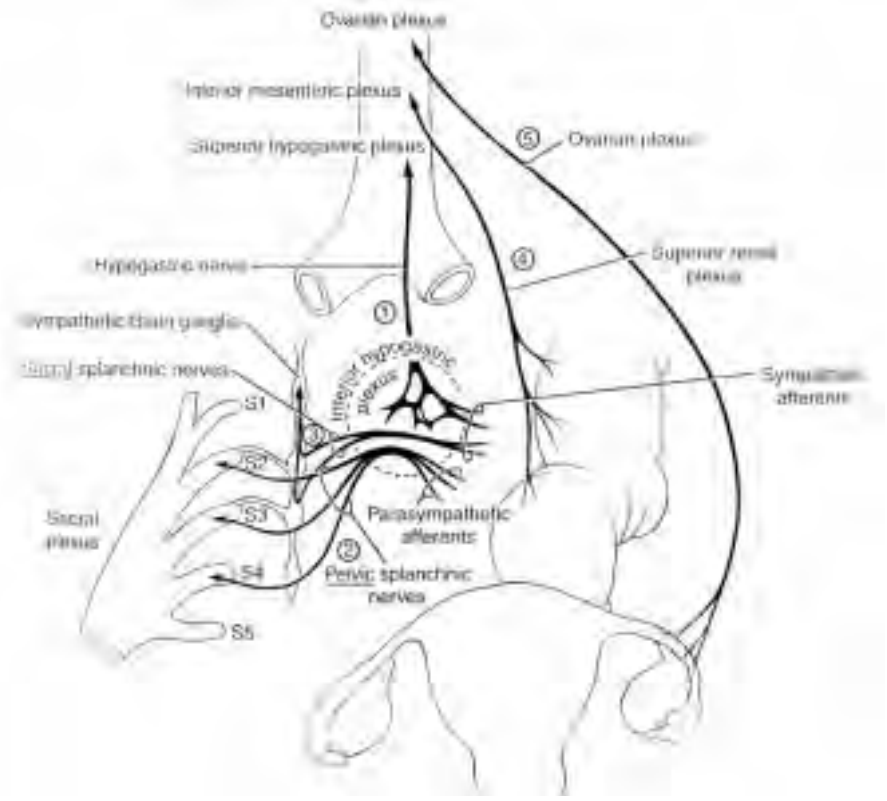

FIGURE 5. Transmission of nociceptive signals out of the pelvis. (From reference 4, with permission.)

small ganglia, primarily sympathetic and some parasympathetic. Many afferent fibers from the bladder, uterus, and middle and lower rectum pass through this region, following various routes to dorsal root ganglia and dorsal horns of the spinal cord.

The sympathetic efferent nerve supply to the inferior hypogastric plexus is derived from three sources. The first is downward continuation of the superior hypogastric plexus overlying the lower portion of the aorta and prelumbar vertebrae through each hypogastric nerve. The second is from preganglionic fibers from sacral splanchnic nerves that arise from downward or sacral extension of the sympathetic chain of paravertebral ganglia. The third sympathetic source consists of postganglionic sympathetic fibers, having synapsed in the sacral sympathetic ganglia chain.

The inferior hypogastric plexus also has a parasympathetic supply from pelvic splanchnic nerves, which are derived from anterior rami of spinal nerves S2, S3, and S4. These parasympathetic nerves are distributed to various plexus in the pelvis to give parasympathetic innervation to the vagina, cervix, uterus, tubes, but not ovaries, and to the urethra, bladder, lower ureters, rectum, and anal canal. In addition, parasympathetic and sympathetic innervations travel through pudendal nerves to areas of the vulva and clitoris that respond to sexual stimulation. The ovary obtains its own parasympathetic nerve supply from the vagus nerve (tenth cranial nerve).

Each inferior hypogastric plexus feeds into three other plexuses: middle rectal, uterovaginal (Frankenhauser), and vesical. The middle rectal plexus travels to the rectum by way of middle rectal blood vessels. Visceral afferent fibers from the middle rectal plexuses intermingle on the rectum with visceral afferent fibers from the superior rectal plexus. Therefore, any noxious stimulus, such as distention of the lower rectum, may be transmitted out of the pelvis through the middle rectal plexus back to the inferior hypogastric plexus, or through superior rectal nerves through the superior rectal artery back to the inferior mesenteric plexus.

The uterovaginal plexus supplies efferent and afferent innervation to the uterus, cervix, and vagina. It is contained within the endopelvic connective tissue surrounding the ureter and uterine vessels, just lateral to uterosacral ligaments as they insert into the uterus. Therefore, a paracervical block of local anesthesia should be placed just lateral to the insertion of each uterosacral ligament into the cervix. Many of these nerve fibers enter the lower uterine segment with uterine vessels and not through uterosacral ligaments. This is one reason why procedures that transect uterosacral ligaments have a relatively high failure rate for relief of uterine pain.[8] In addition, lateral transection of uterosacral ligaments may damage ureters by transecting them or by producing surrounding fibrosis and fixation. This increases the risk of ureteral injury during subsequent hysterectomy.

The uterovaginal plexus sends nerve fibers to the vagina along with the vaginal artery, to the cervix by way of the uterine artery, and to the lower uterine segment accompanying the ascending uterine artery. These visceral nerves also supply the upper part of the broad ligament, as well as the uterine tube, where they intermingle with visceral nerves from the ovarian plexus and other nerves directly from the inferior hypogastric plexus. Therefore, irritating stimuli in the broad ligament may proceed to the spinal cord through fibers from the uterovaginal plexus, through the inferior hypogastric plexus, or through the ovarian plexus along with the ovarian artery back to the ovarian plexus at the root of the ovarian artery on the aorta. This is another example of intermingling.

The vesical plexus travels with the inferior vesical artery and ureter to innervate the bladder. Remember, electric action potentials resulting from strong noxious stimuli in the bladder travel back through the inferior hypogastric plexus, where they may activate other action potentials in other afferent nerve fibers through cross-talk.

Afferent fibers that travel through each inferior hypogastric plexus can travel out of the pelvis by three separate routes. The first route is along the hypogastric nerve to the superior hypogastric plexus. The second route is with the pelvic (parasympathetic) splanchnic nerves (nervi erigentes) to sacral nerves 2, 3, and 4. The third route is with sympathetic nerves that travel back to the spinal cord by way of sacral splanchnic nerves from the sacral extension of the paravertebral chain of ganglia.

There are two other afferent neuropathways out of the pelvis. Superior rectal nerves, the fourth route, innervate the upper and middle portions of the rectum and leave the pelvis by way of the superior rectal artery, which leads to the inferior mesenteric plexus of nerves at the root of the inferior mesenteric artery

at the aorta. As mentioned, these nerves intermingle on the rectum with those from the middle rectal plexus of nerves.

The ovarian plexus of nerves, the fifth route, is a fine network of visceral nerves from the tenth and eleventh thoracic segments of the spinal cord. These nerves enter paravertebral sympathetic ganglia at the level of the fourth lumbar vertebra and follow ovarian vessels to the ovaries, tubes, and broad ligaments. Thus, these visceral afferent fibers intermingle in the broad ligament with visceral afferent fibers from the uterovaginal plexus and from intercommunications with the inferior hypogastric plexus itself. Ovarian pain traditionally is referred to T10 and T11 dermatomes.

Perception of Pain

Actual perception of and physiologic and psychologic response to pain is a complex process. The process is significantly affected by unconscious and conscious processing of nerve signals to the spinal cord, midbrain, and cortex that are received from visceral and somatic sensory afferents.[9] The individual's perception of pain also depends on the strength of the nociceptive stimulus, spreading of electric action potentials by cross-talk, and number of posterior horn interneurons stimulated by incoming first-order neurons directly from the site of stimulus.

Several receptors in the dorsal horn of the spinal cord can modulate processing of nociceptive signals at that level. For example, serotonin release appears to enhance release of endogenous opiates that certainly can suppress subjective perception of pain. Opiate receptors have also been found in the dorsal horns of the spinal column. Various drugs such as tricyclic agents and selective serotonin uptake inhibitors increase the concentration of serotonin in these areas and have a role in pain management. In addition, spinothalamic tracks are the primary ascending spinal routes for nociceptive information to the midbrain and cortex.

Within the thalamus, some painful sensations are brought to conscious perception and allow sensory discrimination of pain such as location, nature, and intensity. Other nociceptive signals remain in the unconscious and mediate visceral autonomic responses, as well as emotional responses such as arousal, fear, and general orientation. Feedback from the cortex and midbrain down descending spinal tracts may actually magnify or even suppress processing of noxious information at the level of the dorsal horn. Thus the brain has important input into processing this information at the level of viscerosomatic convergence at the dorsal horn of the spinal column.

Acute visceral pelvic pain usually has a specific cause, such as rupture of an ovarian cyst, tubal pregnancy, dysmenorrhea, acute cystitis, and infectious bowel syndrome, among others. Chronic pelvic pain, unfortunately, usually does not have a specific diagnosis. It is frustrating and discouraging to the patient, her family, and her physicians. Chronic pelvic pain is a result of a complex, poorly understood, abnormal physiologic interaction among noxious stimuli, both visceral and somatic; actual dysfunction within the nervous system itself; and adverse interplay with psychologic, family, and social relationships and interactions.[10] These many factors are in a dynamic state and can be manifested clinically by different characteristics at successive office visits.

Afterthought

Our knowledge of acute and chronic pelvic pain in women is based mostly on research in rats, cats, and monkeys and projected to the human experience. This article describes for the gynecologist the complex interactions and incompletely understood dynamics of the visceral nervous system and its relation to this disorder. Physicians can no longer tell a woman that her pelvic pain is "all in her head." Acute pelvic pain usually can be treated successfully. However, the chronic condition requires detailed evaluation with the primary goal of patient and family education and alleviation of suffering, rather than elimination of pain.

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Suggested Reading

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Williams PL, Bannister LH, Berry MM, et al, eds: Gray's Anatomy, 38th ed. New York, Churchill Livingstone, 1995

CME TEST 005
Pelvic Pain

(Test valid through August 1999)

The American Association of Gynecologic Laparoscopists (AAGL) is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians. The AAGL designates this continuing medical education activity for 1 credit hour in category 1 of the Physician's Recognition Award of the American Medical Association.

Instructions: Read the article on pelvic pain beginning on page 15, and select all answers that apply for each of the following questions. Record your answers by circling the appropriate letter on the test form located on the next page.

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1. Visceral pain is

a. Dull
b. Diffuse
c. Poorly localized
d. Accompanied by nausea
e. All of the above

2. Afferent (sensory) fibers

a. Are well myelinated
b. Travel in clear pathways
c. Synapse in preaortic ganglia
d. Have cell bodies only in the dorsal root ganglia

3. The viscera are very sensitive to all except one of the following:

a. Rapid distention
b. Ischemia
c. Burning
d. Traction

4. Intermingling of afferent fibers refers to

a. Electric action potentials in one nerve exciting electrical activity in another nerve
b. Overlapping of dermatomes by afferents from as many as four other spinal nerves
c. Interaction of nerves in midbrain and cortex
d. Afferent fibers from different neuropathways detecting the same noxious stimuli in the same visceral area

5. Cross-talk refers to

a. Intercommunication among various nerve plexus and groups of ganglia
b. Electric action potentials in one afferent nerve exciting electrical activity in another afferent nerve
c. The process by which intermingled afferent nerves can detect noxious stimuli in the same visceral area
d. Communication between subconscious midbrain and conscious cortex

6. The ratio of somatic afferents to visceral afferents in each dorsal root ganglion is

a. Over 9:1
b. Approximately 7:2
c. Approximately 5:5
d. Approximately 1:9

7. Each second-order neuron in the dorsal horn of the spinal cord

a. Stimulates only one dermatome
b. Primarily matches with one incoming preganglionic afferent
c. Is key to understanding viscerosomatic convergence
d. Is only 2% to 7% effective in its transmission

8. Uterosacral ligaments are important in understanding central uterine pain because

a. Most afferent fibers from the uterus pass through them
b. Transection procedures are effective in relieving central pelvic pain
c. Paracervical block should be placed within these ligaments through the vagina
d. Transection can seriously injure the ureter

9. All of the following statements regarding the ovarian plexus of nerves are true except

a. It receives its parasympathetic nerve supply from pelvic parasympathetics (nervi erigentes)
b. It enters sympathetic paravertebral ganglia at L4
c. It refers back to T10 and T11 segments
d. It also innervates the broad ligament

10. The purpose of management of chronic pelvic pain is to

a. Medicate the patient and sedate pain
b. Perform aggressive laparoscopy to lyse adhesions, obliterate endometriosis, and eliminate pain
c. Educate the patient and her family and help alleviate suffering
d. Have the patient focus on only one symptom

The Journal of the American Association of Gynecologic Laparoscopists, CME Test 005.

Pelvic Pain

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