I. Rocker (Ed.)

Pelvic Pain in Women
Diagnosis and Management

Foreword by L.P. Thomas

With 32 Figures

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Foreword

Pelvic pain in the female patient is common in gynaecological practice, but the specialties of general surgery, urology and orthopaedics provide a significant number of patients and problems. These patients may suffer a multitude of symptoms, and only careful analysis and investigation of each individual problem by the doctor concerned will lead to correct diagnosis and management.

The subject matter of this book lies in the practice of many specialties, and all are combined here in a coherent whole. This emphasises the close collaboration necessary between family practitioners, junior hospital staff and consultants. The authors are consultants who work together in a busy district general hospital, and their experience and collaboration is evident in the approach to the diagnosis and management of pelvic pain in the female. Emphasis is laid on the careful evaluation of history and examination and the correct interpretation of diagnostic investigations. Full details of radiology, ultrasound scanning, endoscopy, peritoneoscopy and bacteriological investigation are given. Full consultation between members of staff who have special experience in these investigative procedures is of paramount importance. Details of treatment for relief of pain are important to all doctors concerned with this aspect of clinical management, and this section will be of particular value. The blending of these specialties allows full consideration of the problems affecting the patients. Careful management leads to better treatment for the patient and better satisfaction for the doctor.

Acute pelvic pain is easier to diagnose than chronic pain, and management is often more straightforward. Careful attention has been paid to the consideration of chronic pain with a multitude of symptoms. The psychological aspect of these problems is also fully covered, with emphasis on the consideration of the whole woman and her worries. The authors show considerable understanding of the difficult and sometimes delicate problems involved.

The book is directed to family practitioners, junior hospital doctors and medical students, and it is hoped that the full consideration of the many facets of pelvic pain will lead to a better understanding of the condition.

Newport, 1989 L. P. Thomas, M Ch, FRCS

Preface

Female pelvic pain is a common medical problem which in its acute, recurrent or chronic effects taxes the diagnostic and communication skills of all associated with patient care.

The changing pattern of life over the past 30 years has reduced parity and subsequent prolapse. Antibiotic therapy has changed the natural history of pelvic inflammatory disease, so that the inoperable frozen pelvis of the 1950s is now rarely encountered. Effective contraception has played a part in the increasing incidence of sexually transmitted disease and cervical intraepithelial neoplasia. While the number of live born has fallen in the past 20 years, there has been an increase in elective terminations of pregnancy, which now number 1 in 4 or 1 in 5 of all pregnancies. In addition there has been a rise in the number of one-parent families and their attendant social problems. Endometriosis is increasing in prevalence, and ovarian malignancy and bowel cancer have become, respectively, the commonest gynaecological and second commonest intestinal causes of death. There is also the additional expectation of diagnosis leading to treatment.

The primary health team successfully manages the majority of these problems, possibly with the backup of specialist services, but there remains a residual group of women who have chronic pelvic pain without obvious cause or acceptable explanation.

This handbook is the result of the combined experience of hospital consultants working in a large district general hospital, an association that for some began in their medical student days and for others 15 to 25 years ago. They have therefore coordinated their approach not only by cross referral but also by their teaching to graduate and postgraduate students. This book is intended as a guide for the management of common problems and particularly as a basis for communication to reduce the risk of vague explanation given in good faith but which increases anxiety.

My thanks are due to my colleagues for their willing cooperation, to Mrs Val Reed for the typing and to the Medical Illustration Department of the Royal Gwent Hospital for their sterling efforts.

Newport, 1989 I. Rocker

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1 General Aspects

J.M. HUGHES, D.E. STURDY and G.D. THOMAS

General Anatomy and Innervation of the Female Pelvis

D. E. Sturdy

The female pelvis is a basin-shaped cavity bounded anteriorly by the pubic bone, laterally by the ilium and ischium and posteriorly by the sacrum and coccyx (the true or lesser pelvis). The pelvic cavity is in direct communication with the abdominal cavity, and its upper limits are the tips of the iliac crests (the transtrochanteric line) related anteriorly to a surface point 3 to 4 cm below the umbilicus and posteriorly to the upper border of the fifth lumbar vertebra (Fig. 1.1). Its anterior wall is part of the musculature of the abdominal cavity. The lateral walls of the pelvic cavity are covered by the iliopsoas, iliacus and obturator muscles, and inferiorly the outlet is guarded by the levator ani and pubococcygeus muscles, which, with the corresponding muscles of the opposite side, form the pelvic diaphragm. Passing through this muscular diaphragm are three channels which open into the perineum, i.e. the urethra anteriorly, the vagina in the middle and the anal canal posteriorly. Anatomically, the visceral contents of the female pelvis can be separated into three compartments – anterior (urinary), middle (genital) and posterior (intestinal).

Within the pelvic basin the bladder and urethra lie anteriorly; the vagina, uterus, broad ligaments, Fallopian tubes and ovaries in the middle; and the anal canal and rectum posteriorly (Fig. 1.2). In addition, within its cavity, the pelvis contains loops of ileum, the sigmoid colon and frequently the greater omentum, the caecum and vermiform appendix. The ureter is the only structure which traverses from one compartment to the other. In its course along the lateral wall of the pelvis it lies in the middle compartment in close proximity to the ovary and in its terminal 4 cm it sweeps anteriorly into the anterior compartment. In this part of its course it is closely related to the lateral fornix of the vagina, especially on the left side. The interior

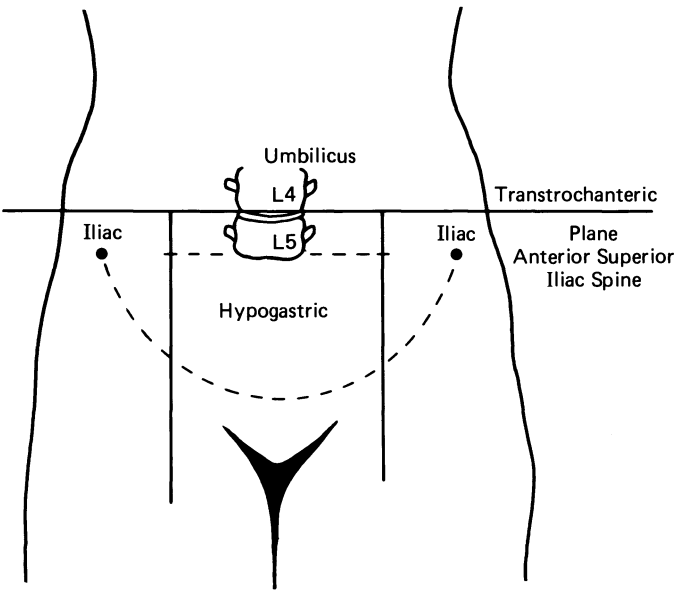

Fig. 1.1. Surface projection of upper limits of pelvis.

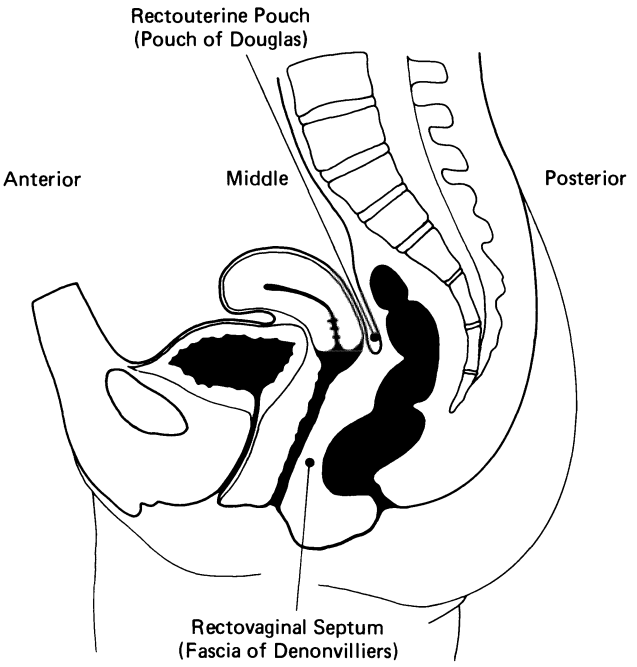

Fig. 1.2. Vertical section of female pelvis; compartmentalisation of pelvic viscera.

of the pelvic cavity is lined with peritoneum, which sweeps downwards on the internal surface of the anterior abdominal wall to cover the upper third of the bladder, the main bulk of the body of the uterus (forming the broad ligament which ensheathes the Fallopian tubes) and the anterior surface of the upper third of the rectum. At the rectosigmoid junction its two fused layers form the mesentery of the sigmoid colon. Where the peritoneum clothes a pelvic organ it forms an integral part of the layered structure of that organ and receives its innervation from the same nerve supply as the viscus.

The peritoneal reflection from the back of the upper part of the vagina to the anterior surface of the rectum forms a pouch of variable depth – the rectouterine pouch of Douglas – and the fused layers of peritoneum continue downwards as a thick fascia towards the perineum between the rectum and vagina – the fascia of Denonvilliers (Fig. 1.2).

Innervation of the Pelvic Viscera

The pelvic viscera are innervated by both autonomic and somatic nerves. The cord segments for sympathetic innervation are T11, 12, L1, 2, and the cord segments involved in parasympathetic supply are S2, 3, 4 (pelvic splanchnics). Segments S2 and S3 form the somatic pudendal nerve, which supplies the perineum and the terminal 2.5 cm of both the vagina and the anal canal (Fig. 1.3). Preganglionic sympathetic fibres synapse in ganglia along the major arteries of the posterior abdomen whereas the parasympathetic preganglionic fibres establish synaptic connections in ganglia close to, or in the wall of, the viscus that they supply.

Anterior Compartment, Bladder and Urethra

The bladder and urethra are innervated by a vesical plexus of sympathetic and parasympathetic nerves, each of which contains efferent (motor) and afferent (sensory) fibres. The hypogastric plexus (Fig. 1.3) also provides autonomic nerves to the bladder via the internal iliac, superior and inferior vesical and uterine arteries. Pain fibres, which are stimulated by distension, spasm, or inflammation of the bladder, run in both sympathetic (T11, 12 L1, 2) and parasympathetic (S2, 3, 4) nerves, mainly the latter.

Middle Compartment; Uterus, Fallopian Tubes, Ovaries and Vagina

The uterus and Fallopian tubes receive their autonomic nerve supply from the hypogastric plexus with branches of the uterine artery. These autonomic fibres have a common origin with those supplying the bladder. The ovaries and lateral 2 cm of the tubes receive their autonomic nerve supply from ovarian plexuses: the nerves reach the ovaries along with the ovarian arteries (Fig. 1.3). The ovarian plexuses are formed from branches of the renal, aortic and hypogastric plexuses (T11, 12, L1, 2). The upper three-quarters

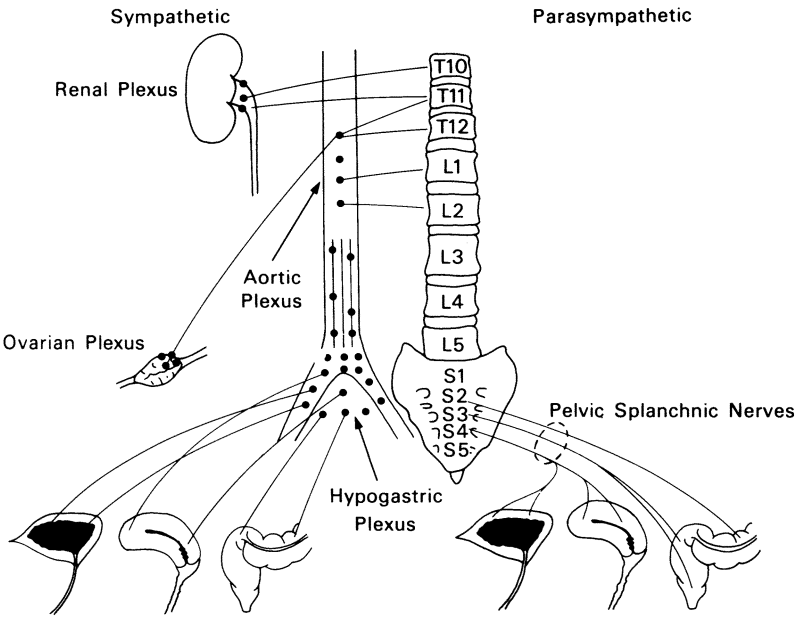

Fig. 1.3. Innervation of the pelvic viscera.

of the vagina has the same nerve supply as the uterus, whereas the distal quarter derives its supply from the pudendal nerve (S2, 3), in common with the distal 2.5 cm of the squamous portion of the anal canal.

Posterior Compartment; Rectum and Anal Canal

The rectum and upper half of the anal canal receive their vasomotor sympathetic innervation from the aortic and hypogastric plexuses along with branches of the inferior mesenteric and superior rectal (haemorrhoidal) arteries (T11, 12, L1, 2). Parasympathetic innervation is supplied by the pelvic splanchnic nerves from L2, 3, 4, which join the hypogastric plexus and are distributed to the rectum, internal half of the anal canal and internal anal sphincter muscles, together with branches of the superior and middle rectal blood vessels.

The sensory supply to the external 2.5 cm of the anal canal is from the somatic pudendal nerve (S2, 3) which, together with the perineal branch of the fourth sacral nerve (S4), innervates the external sphincter muscle (Fig. 1.3).

In view of the common sympathetic and parasympathetic innervation of the three compartments of the pelvic cavity, it is not surprising to find in clinical practice that a patient is unable to say whether visceral pain is emanating from the bladder, the genital tract or the rectum. Ancillary symptoms and physical signs will, in most instances, guide the clinician to the source of the patient's pelvic pain.

Pelvic Peritoneum

Visceral pelvic peritoneum, which covers the upper third of the bladder, the body of the uterus and the upper third of the rectum and the rectosigmoid junction, is an integral part of these organs and is innervated by autonomic nerves supplying these viscera. It is insensitive to touch but responds with pain on traction, distension, spasm or ischaemia of the viscus. Parietal pelvic peritoneum, which covers the upper half of the lateral wall of the pelvis and the upper two-thirds of the sacral hollow, is supplied by somatic nerves. These somatic nerves also supply corresponding segmental areas of skin and muscles of the trunk and anterior abdominal wall. Painful stimulation of the parietal pelvic peritoneum may cause referred segmental pain and spasm of the iliopsoas muscle and muscles of the anterior abdominal wall.

Pelvic Ureter

The abdominal ureter crosses into the pelvis at the bifurcation of the common iliac artery and is posterior to the ovary at this point. The ureter follows a course in the medial part of the broad ligament along the lateral pelvic wall towards the ischial spine and then deviates forwards and medially towards the base of the bladder, in close proximity to the uterine artery and lateral vaginal fornix. The pelvic ureter receives its autonomic nerve supply from the hypogastric plexus, which in turn is connected to the renal and aortic plexuses.

Sympathetic fibres emanate from the lower three thoracic (T10, 11, 12) and first lumbar (L1) segments of the cord and parasympathetic fibres from the second, third and fourth sacral segments (S2, 3, 4). All these fibres are thought to be mainly sensory. Distension and spasm of the ureter, e.g. by an impacted calculus, causes ureteric colic, which is referred to segmental areas T10, 11, 12, L1. The severe pain experienced by the patient starts in the loin, with radiation to the groin and labium majus. In some patients pain traverses into the upper thigh by referred radiation along the genitofemoral nerve (L1, 2).

Surgical Aspects of Female Pelvic Pain

On the basis of innervation of the pelvic organs, two types of pelvic pain are recognised — *visceral pain* due to stimulation of autonomic nerves (T11, 12, L1, 2, S2, 3, 4) and *somatic pain* due to stimulation of sensory nerve endings in the pudendal nerve (S2, 3). Pain in the female pelvis and perineum may be visceral or somatic or rarely a mixture of both. Pain in the pelvis may be acute, chronic, or referred, and, whereas this chapter highlights chronic conditions producing pain, a knowledge of acute causes of pain and their diagnosis is necessary because many of these acute conditions become chronic in due course.

Gynaecological/Obstetric Aspects of Pelvic Pain

By early adolescence the vagina, cervix and uterus have developed to functional maturity. The ovary has changed from intermittent to regular ovulation, and the menstrual cycle has started. The ovary undergoes cyclical enlargement and selective follicle maturation, culminating in the release of (usually one) ovum and minimal blood loss into the pelvic cavity some 12 to 14 days before menstruation. Retrograde menstruation through the patent Fallopian tubes may also occur. The patency of the uterine cavity and Fallopian tubes also allows infection direct access to the peritoneal cavity. These factors form a basis for peritoneal irritation, which will cause pain. There is the possibility of unilateral ovarian supremacy, but transpelvic spread of blood can produce ipsilateral, contralateral, and sometimes bilateral pain. The upper vagina is in direct contact with the lower margin of the peritoneal cavity and the pouch of Douglas and so can be sensitive to the pressure of coitus. This effect can be transmitted to adjacent organs, particularly the bladder, ovary, and rectum.

Neurophysiology of Pain

G.D. Thomas

Pain is one of the commonest reasons for medical consultation. In spite of an explosion in research and information relating to the neuroanatomy and neurophysiology of pain over the past decade, the ability to assess a patient's complaint of pain accurately remains imprecise and difficult. The following section outlines the current concepts in the anatomophysiology of pain and explains how this knowledge may be exploited.

Neuroanatomy and Physiology

Peripheral Receptors and Afferents

Nociceptive receptors, which are widespread in the tissues, detect mechanical, thermal and chemical stimuli. When the degree of stimulation becomes actually or potentially harmful (noxious), depolarisation occurs and is propagated centripetally to the neuraxis. Nociceptors have a relatively high threshold and are unaffected by stimuli that can excite the more sensitive tactile and articular mechanoreceptors.

Chemical irritation of nociceptors can be induced with a variety of agents, including lactic acid and K^+ ions (following ischaemia) and histamine, 5-hydroxytryptamine (serotonin), bradykinin and prostaglandins (following tissue damage and inflammation). Prostaglandin E_2 (PGE_2) enhances the stimulating action of bradykinin on nociceptors, thus accounting for the extreme tenderness of many inflammatory reactions. (Non-steroidal anti-

inflammatory drugs act by inhibiting prostaglandin synthetase, thus reducing the concentration of PGE_2 and therefore the sensitivity of the receptors.)

The afferent fibres from the receptor system are of two types: small myelinated (A δ) fibres and (the majority) unmyelinated C fibres. This is therefore a small diameter (mostly less than 5 μm) afferent system, resulting in relatively slow conduction velocity, extreme sensitivity to local anaesthetic agents and a higher threshold to electric stimulation than fibres of larger diameter, such as mechanoreceptor afferents.

The receptors leading to the slightly faster conducting A δ fibres have lower thresholds than those leading to the unmyelinated C fibres. The A δ fibres also have a much more direct path to the thalamus, and it is these fibres which conduct the initial pinprick type stimulus which is always well localised and precedes the more diffuse information carried by the C fibres, which signal tissue injury.

Spinal Cord

On reaching the dorsal horn via posterior nerve roots, incoming impulses are subject to two important modulating influences, one segmental and the other from descending pathways from higher centres.

Segmental Mechanism. Most of the nociceptive C fibres terminate in the substantia gelatinosa of the dorsal horn. From this peripheral situation the nociceptive traffic passes centrally through different laminae before finally converging on transmission (T) cells, which are more deeply situated. The faster conducting A δ fibres have a more direct path to their transmitter cells. When sufficiently stimulated the T cell will fire, and the impulse is then conducted across the midline to ascend in the contralateral anterolateral funiculus. Low-threshold mechanoreceptor traffic travels in relatively thick myelinated fibres ascending in the dorsal columns. At their point of entry into the spinal cord, however, these fibres give off collaterals that penetrate the dorsal horn. These collaterals synapse in the deeper layers of the horn and exert an inhibitory effect on transmission between the substantia gelatinosa and the transmitter cells. When C fibre traffic is relatively light but large-fibre stimulation high, the effect of this so-called gate control mechanism is that the T cell will not be stimulated. In the reverse situation however, the T cell will fire and nociceptive information will be passed along the anterolateral tract. In conditions where large-fibre afferents become deficient, as may happen following an attack of herpes zoster, the preponderance of unopposed C fibre traffic can result in prolonged pain.

Descending Inhibitory Mechanism. Further modulation of nociceptive transmission is brought about by descending pathways from higher centres. The best described mechanism originates in the periaqueductal grey matter of the midbrain. This structure has an input of collaterals from spinothalamic axons and is particularly responsive to pinprick stimulation. Descending fibres from the periaqueductal grey matter pass along a serotoninergic

pathway to the nucleus raphe magnus and then via the dorsolateral tract of the spinal cord to synapse eventually with an inhibitory interneuron. This interneuron is interpolated between the substantia gelatinosa and the nociceptive final transmitter and so is capable of blocking transmission.

Ascending Pathways. When the transmitter cell situated in the deeper laminae of the dorsal horn is sufficiently excited, it will propagate an impulse which is conveyed across the midline to ascend in the anterolateral funiculus of the spinal cord. Within this tract the fibres carrying initial or pinprick type information ascend with relatively few interruptions to the neothalamus. From here the information is relayed to the postcentral gyrus and results in accurate localisation of the pain. The fibres carrying the slightly later information of tissue injury ascend via a less direct route to the brainstem reticular formation. Some spinoreticular fibres ascend in the ipsilateral anterolateral tract, whereas others ascend independently of the main pathways. This explains why anterolateral cordotomy is not always effective in interrupting contralateral pain pathways and why its effectiveness may diminish in time. The information carried by this system is eventually relayed via the anteromedial (palaeo) thalamus to the frontal lobes, limbic system and hypothalamus. It is these connections that result in the less precise component of pain perception and the emotional response to it. The hypothalamic connection initiates the autonomic response to pain.

Visceral Pain

The viscera are innervated by the autonomic nervous system, which has sympathetic and parasympathetic components. The efferent fibres of the sympathetic system emerge through the thoracic and upper lumbar spinal nerves, and the parasympathetic efferent fibres emerge through certain cranial and sacral spinal nerves.

Viscera are insensitive to cutting, crushing or bruising. Excessive distension, contraction and pulling, however, together with some pathological conditions, excite nociceptive afferents and may result in pain. The great majority of the nociceptive afferents run within the sympathetic system. These afferents are the peripheral processes of unipolar cells situated in spinal nerve ganglia, and their courses are not interrupted by synapses in the autonomic ganglia. The sensation aroused by stimulation of these afferents is usually a poorly localised ache or, when caused by excessive contractions, colic. Occasionally, however, the pain may be referred to the same dermatome as that innervated by somatic sensory nerves which share the same vertebral inlet. The segmental inlet levels of sympathetic pelvic afferents are as follows:

Sigmoid colon and rectum	L1–2
Ureter	T11–L2
Ovary	T10–11
Urinary bladder	T11–L2
Uterus	T12–L1
Fallopian tube	T10–L1