Journal of Obstetrics and Gynaecology, February 2007; 27(2): 130 – 133

APPLIED ANATOMY

The inferior hypogastric plexus: A different view

R. SPACKMAN[1], B. WRIGLEY[1], A. ROBERTS[1] & M. QUINN[2]

[1]*University Department of Anatomy, Bristol and* [2]*Department of Obstetrics and Gynaecology, Hope Hospital, Manchester, UK*

Summary
The morphology of the inferior hypogastric plexus has been demonstrated in a series of dissections of nulliparous cadavers. Each cadaver was embalmed in a solution containing a significant proportion of methanol which preserved the pliability of the tissues enabling the nerve plexi to be clearly demonstrated. These structures are not normally encountered during general gynaecological surgery and clinicians are often unaware of the possible consequences of injury during vaginal delivery or sustained constipation. Denervation of pelvic organs, with subsequent reinnervation over the medium term, may account for a variety of obstetric and gynaecological syndromes.

Introduction

The innervation of the pelvis has been studied in both fresh and embalmed specimens (Hunter 1774; Lee 1841; Beck 1846; Frankenhäuser 1867; Davis 1933; Labate 1938; Krantz 1959; Quinn 2003). Despite these contributions, much remains to be understood about the complex pathways of the superior, middle and inferior hypogastric plexi as well as the intrinsic innervation of the respective viscera. Demonstration of the inferior hypogastric plexus (IHP) has not been a prominent feature of medical education since anatomical specimens are usually embalmed, and preserved, in formalin. Delicate nerves become less pliable and difficult to demonstrate with standard anatomical techniques. This technical subtlety may have contributed to limited clinical awareness of these important nerves.

Pelvic innervation is derived from the superior hypogastric plexus, sympathetic chain, parasympathetic fibres (S2 – 4) and the sacral plexus (S1 – S5). The superior hypogastric plexus is the downward continuation of the inferior mesenteric plexus over the lower aorta and sacral promontory. The hypogastric nerves are formed by the bifurcation of the superior hypogastric plexus and deliver this innervation to the inferior hypogastric plexi over the posterolateral pelvis lying almost parallel to the internal iliac arteries. Interconnecting fibres between the right and left hypogastric nerves constitute a middle hypogastric plexus though it is often difficult to differentiate from the superior hypogastric plexus. Visceral plexi associated with uterus, vagina, rectum and bladder are derived from the IHP. The uterus receives its primary innervation from the uterovaginal plexus, which is located near the transverse cervical ligament lateral to the cervix (Frankenhäuser 1867). Nerve fibres are distributed throughout the myometrium with the branches of the uterine artery with a significant nerve plexus at the endometrial-myometrial interface (Kranz 1959; Quinn 2003) and additional concentrations of nerve fibres in the subserosal layers (Quinn 2003). The nerve supply of the endometrium remains controversial though the cervix has a rich submucosal innervation (Stjernholm et al. 2000; Stjernholm et al. 1999).

Gynaecological interest in the innervation of pelvic viscera has been renewed by observations of widespread nerve fibre proliferation in many women with sensory pelvic symptoms (Quinn and Kirk 2002; Lerner et al. 1985; Christmas et al. 1990; Westrom and Willen 1998; Boreham et al. 2002; Chan et al. 2003).

The IHP is rarely seen during gynaecological surgery as it occupies the posterolateral walls of the pelvis when most benign surgery requires access to midline pelvic structures. Hysterectomy may be regarded as a denervatory procedure since the uterus is a midline viscus that is removed along with its nerve supply. Nerve preservation has been proposed to reduce comorbidity at radical hysterectomy. Injury to the nerve plexus during some patterns of vaginal delivery and sustained constipation has been proposed as a source of denervation-reinnervation causing a wide range of obstetric and gynaecological syndromes (Quinn 2004; Quinn 2005; Allen and Masters 1955; Quinn and Armstrong 2005).

Materials and methods

Serial dissection of three female cadavers bequeathed to the Department of Anatomy, University of Bristol, was performed under the terms of the 1984 Anatomy Act. Physical findings, including the shape of the external cervical os, the shape of the vagina, the integrity of the vaginal supports, etc. suggested that all were nulliparous subjects. Each cadaver was embalmed by inserting catheters into the femoral artery in the region of the femoral triangle; one directed distally from this point, and one proximally. A total of 20 litres of fluid were initially

Correspondence: M. Quinn, 10 Royal Victoria Park, Bristol BS10 6TD, UK. E-mail: mjquinn001@btinternet.com

ISSN 0144-3615 print/ISSN 1364-6893 online © 2007 Informa UK Ltd.
DOI: 10.1080/01443610601113839

delivered with a further 5 litres injected by hand into localised sites which had not been perfused by the embalming pump. The embalming fluid consisted largely of ethanol (80%) and contained a low formaldehyde content (1.4%) and high methanol content (4.3%), which maintained both the colour of the tissue, as well as a degree of elasticity. After embalming, the pelvis was divided from the torso at the level of the 4th lumbar vertebra, immediately inferior to the bifurcation of the abdominal aorta. The lower limbs were removed at mid thigh level, and then the pelvis was sectioned in the median sagittal plane. For 1 week prior to dissection, the pelvis was stored at 19°C, submerged in a 50% solution of industrialised methylated spirit. Small bowel was reflected and removed from the false pelvis and the parietal peritoneum was divided over the common iliac artery. The peritoneum could then be carefully separated from the underlying fascia. Each sagittal hemisection was prepared and mounted in a similar fashion, enabling full access to the pelvic side wall in each specimen.

Dissection was performed by blunt and sharp dissection starting at the bifurcation of the common iliac artery. Traction on the peritoneum and lower viscera provided planes of dissection in which the branches of the inferior hypogastric plexus could be demonstrated. Serial photographs were taken at different stages of the procedure.

Results

Sagittal hemisections provide optimal exposure for the demonstration of pelvic innervation though the superior hypogastric plexus and abdominal branches require a trunk for their full demonstration. Removal of the midline viscera allows improved access over surgical specimens, and, orientation by sacral segment is directly available. The extraperitoneal connective tissue is first separated from the plexus by blunt dissection and then removed, at which stage the full extent of the plexus becomes apparent (Figure 1).

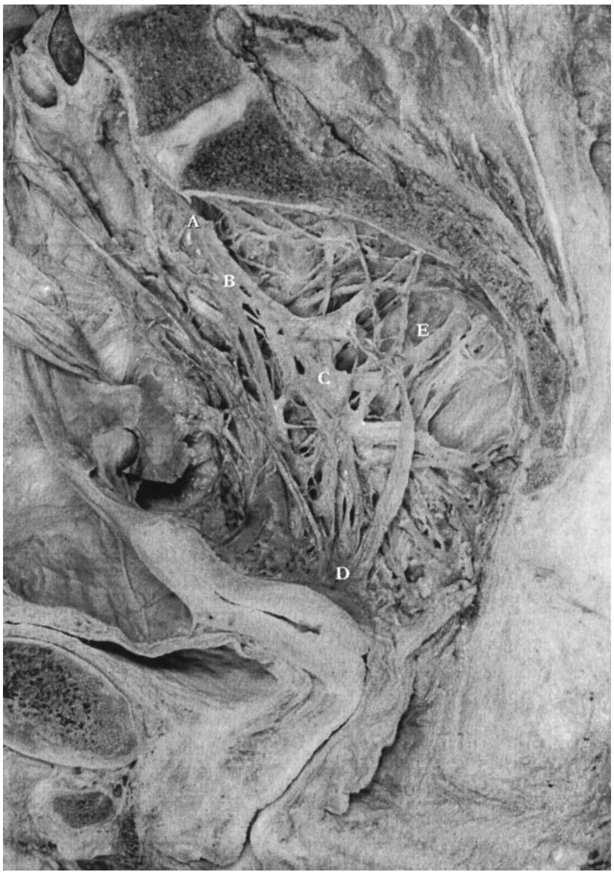

Figure 1. Pelvic innervation on the right pelvic side wall. (A) Superior hypogastric plexus, (B) right hypogastric nerve, (C) inferior hypogastric plexus, (D) uterovaginal nerve plexus, (E) sacral nerves.

The course of the hypogastric nerves

The right and left hypogastric nerves connecting the superior and inferior hypogastric plexi were 7–10 cm in length in these specimens. They were plexiform, occupied the connective tissue adjacent to the internal iliac artery and consisted of several strands in one of the specimens before terminating in the plexus at the level of the third sacral vertebra close to the origins of the uterosacral ligaments. In each of the six specimens, branches of the hypogastric nerve were closely associated with the ureter and branches of the anterior division of the internal iliac artery.

The inferior hypogastric plexi

In all hemipelves the IHP formed a single layered plate on the posterolateral wall of the pelvis. In an axial plane it occupied sacral levels S4–S5, in a sagittal plane it was found immediately lateral to the pelvic viscera medial to the vessels, and in the coronal plane it was found in the parametrial tissues lateral to the cervix. Each IHP was tethered at its origin on the side wall of the pelvis by varying amounts of connective tissue. Medially it was fixed by the branches to the viscera. Condensations of connective tissue in the form of named ligaments, e.g. uterosacral or cardinal ligaments, could be identified in these preparations and their course demonstrated by traction on contained vessels and nerves. In each of the hemipelves, the IHP contributed nerve fibres to the uterosacral ligament.

Each IHP was medial to the branches of the internal iliac artery lying in a plane medial to the vessels, and consisted of a fenestrated sheet of nerves in a curved plane with the majority of fibres travelling in a near parallel orientation with significant connections between them. Dimensions were difficult to derive with precision though they occupied an area of approximately 7 × 4 cm in the largest specimen. Branches of each plexus were closely related to the branches of the internal iliac artery. The most medial fibres of the plexus ran with the uterosacral ligament; the majority of these medially situated fibres converged toward the body and cervix of the uterus, while more lateral fibres diverged to pass toward the bladder and rectum. Individual plexi are associated with each of the lower viscera including the uterovaginal plexus (Frankenhäuser's plexus, Figure 1).

The visceral plexi

The visceral branches of the IHP were distributed anteriorly towards their target organs. Vesical branches were associated with the ureters, rectal branches were delivered to the lateral aspect of the rectum and uterovaginal (Frankenhäuser's) plexi were found lateral to the cervix (Figure 1). The uterovaginal plexus occupies an area approximately 2 cm^2, is circumscribed medially by the ampulla of the rectum and the lateral vaginal fornix; laterally by the uterine artery, superiorly by the broad ligament and inferiorly by fibres of the cardinal ligament. This plexus receives additional fibres from the sympathetic trunk and parasympathetic fibres from the pelvic splanchnic nerves. The innervation of the bladder reaches the viscus close to the ureters before sweeping across the surface of the viscus.

Discussion

These dissections demonstrate the topography of the inferior hypogastric plexus and its important anatomical features including the hypogastric nerves, inferior hypogastric and visceral plexi. The embalming technique enables the colour and pliability of the tissues to be preserved while providing for subsequent preservation in formalin. Patient dissection together with this combination of embalming and storage techniques provides prosected specimens that have been used for both undergraduate and postgraduate education for 12 months with some diminution in colour of the tissues though minimal loss of anatomical detail.

The convergence of major nerve trunks on the vaginal vault suggests they may be vulnerable at vaginal delivery (Allen and Masters 1955), or, through sustained attempts at defaecation (Quinn and Armstrong 2005). Premature, or prolonged, maternal voluntary efforts have been associated with avulsion of the levator ani from its fascial origin over the obturator internus (Allen 1971). The IHP lies medial to the pelvic musculature and may be susceptible to injury particularly if associated tissues become friable and oedematous during the course of prolonged labour. Successive vaginal deliveries may be part of the reason for the variable descriptions of the anatomical features in prior accounts since the IHP has been regularly described as asymmetrical or multilayered in previous morphological accounts (Davis 1933; Labate 1938).

The presence of mixed nerve fibres in some of the myofascial supports including the uterosacral ligaments has been noted previously (Campbell 1950). These nerves may also be compromised following vaginal delivery, since it is a common observation at laparoscopy to observe asymmetry of the uterosacral ligaments and loss of the interligamentous fibres in women with chronic pelvic pain (Quinn and Kirk 2004). Preservation of nerves may be desirable in some patterns of radical hysterectomy to maintain visceral function though extended studies of the clinical consequences are not yet available.

The morphology of the IHP suggests that nerves may be injured at different sites by different mechanisms. Reinnervation has been observed in all pelvic viscera in both nulliparous and multiparous women implying prior denervatory processes including sustained constipation and childbirth (Quinn and Kirk 2002; Lerner et al. 1985; Christmas et al. 1990; Westrom and Willen 1998; Boreham et al. 2002; Chan et al. 2003; Quinn and Armstrong 2005). Constipation may disrupt the sagittal axis of myofascial support along the axis of the uterosacral ligaments, rectovaginal septum and perineal body where interruption of nerves at fascial junctions may result in denervation-reinnervation and uterine (chronic pelvic pain) and vulval (vulvodynia) reinnervation (Quinn 2006a,b). Injury to the intrinsic innervation of the uterus including the endometrial-myometrial nerve plexus may also result from curettage or endometrial ablative techniques and lead to impaired uterine contractility and defective placentation (Quinn 2006b). Enhancing clinical awareness of the structure and function of the inferior hypogastric plexus may lead to an improved understanding of some obstetric and gynaecological syndromes.

References

Allen WM, Masters WH. 1955. Traumatic laceration of uterine support. American Journal of Obstetrics and Gynecology 70:500–513.

Allen WM. 1971. Chronic pelvic congestion and pelvic pain. American Journal of Obstetrics and Gynecology 109:198–202.

Beck T. 1846. On the nerves of the uterus. Philosophical Transactions of the Royal Society of London 136:213–235.

Boreham M, Wai CY, Miller RT, Schaffer JI, Word RA. 2002. Morphometric properties of the posterior vaginal wall in women with pelvic organ prolapse. American Journal of Obstetrics and Gynecology 187:1501–1509.

Campbell RM. 1950. The anatomy and histology of the sacrouterine ligaments. American Journal of Obstetrics and Gynecology 59:1–12.

Chan CLH, Facer P, Davis JB, Smith GD, Egerton J, Bountra C et al. 2003. Sensory fibre expressing capsaicin receptor TRPV-1 in patients with rectal hypersensitivity and faecal urgency. Lancet 361:385–391.

Christmas TJ, Rode J, Chapple CR, Milroy EJG, Turner-Warwick RT. 1990. Nerve fibre proliferation in interstitial cystitis. Virchows Archiv. A, Pathological Anatomy 416:447–451.

Davis AA. 1933. The innervation of the uterus. Journal of Obstetrics and Gynaecology of the British Empire 40:481–497.

Frankenhäuser F. 1867. Die nerven der Gebarmutter und ihrer Endigungen in den glatten Muskelfasern. Ein Beitrag zur Anatomie und Gynakologie. Jena, Germany: Mauke. pp 1–82.

Hunter W. 1774. Anatomica Uteri Humani Gravidi Tabulis Illustrata. Birmingham: Baskerville.

Kranz KE. 1959. Innervation of the human uterus. Annals of the New York Academy of Science 75:770–784.

Labate JS. 1938. The surgical anatomy of the superior hypogastric plexus – presacral nerve. Surgery Gynecology and Obstetrics 67:199–211.

Lee R. 1841. On the nervous ganglia of the uterus. Philosophical Transactions of the Royal Society of London 2:269.

Lerner EJ, Jaffe M, Ree HJ, McDuff HC. 1985. Proliferation of myometrial nerves in patients with severe dysmenorrhoea. Rhode Island Medical Journal 68, 265–267.

Quinn MJ. 2006a. Persistent constipation and posterior vulval pain. Journal of Obstetrics and Gynaecology 26:388–389.

Quinn MJ. 2006b. Sustained constipation and subsequent reproductive outcomes: Is there a link? Journal of Obstetrics and Gynaecology 26:366–367.

Quinn MJ. 2005. Preeclampsia and partial uterine denervation. Medical Hypotheses 64:449–454.

Quinn MJ. 2004. Obstetric denervation – gynaecological reinnervation: disruption of the inferior hypogastric plexus in childbirth as a source of gynaecological symptoms. Medical Hypotheses 63:390–393.

Quinn MJ. 2003. The endometrial-myometrial interface. American Journal of Obstetrics and Gynecology 188:857.

Quinn MJ, Armstrong G. 2005. Uterine nerve fibre proliferation in advanced endometriosis. Journal of Obstetrics and Gynaecology 24:932–933.

Quinn MJ, Kirk N. 2004. Uterosacral reinnervation in parous endometriosis. Journal of Obstetrics and Gynaecology 24:189–190.

Quinn MJ, Kirk N. 2002. Differences in uterine innervation at hysterectomy. American Journal of Obstetrics and Gynecology 187:1515 – 1520.

Stjernholm Y, Sennstrom M, Granstrom L, Ekman G, Yong L, Johansson O. 2000. Neurochemical and cellular markers in human cervix of late pregnant, postpartal and non-pregnant women. Acta Obstetricia Gynecologica Scandinavica 79:528 – 537.

Stjernholm Y. Sennstrom M. Granstrom L. Ekman G. Johansson O. 1999. Protein gene product 9.5 – immunoreactive nerve fibres and cells in human cervix of late pregnant, postpartal and non-pregnant women. Acta Obstetricia Gynecologica Scandinavica 78:299 – 304.

Westrom LV, Willen R. 1998. Vestibular nerve fiber proliferation in vulvar vestibulitis syndrome. Obstetrics and Gynecology 91:572 – 576.