Chronic pelvic pain: the enigma of gynaecological practice

Chronic pelvic pain is a debilitating condition with a complex and poorly understood aetiology. It is an important cause of chronic ill health and a major reason for time off work. This review focuses on how a systematic approach to its management significantly reduces morbidity and the consequences thereof.

Chronic pelvic pain (CPP), variously referred to as the 'bête noire' of gynaecology or 'a gynaecological headache', has been known for over 150 years. It is not a disease but a symptom with a diverse and poorly understood aetiology, causing physical discomfort and lifestyle disruption to those affected. The pain is mainly pelvic and in women hence upper genital tract disease is commonly suspected but, as abdominal or other pelvic structures may play an aetiological role (Gunter, 2003), a multidisciplinary approach to its effective management is often necessary.

Definition, prevalence and modelling

Pelvic pain of at least 6 months' duration is defined as CPP. This has been modified to include pelvic pain of at least 3 months' duration since an acute inflammatory process would have resolved by this time (Prentice, 2000). The exact prevalence is difficult to ascertain, but a population-based survey of women aged 18–50 years in the USA reported a CPP prevalence rate of 14.7% (Mathias et al, 1996); 15% of those employed reported time lost from paid work and 45% reported reduced work. The estimated medical cost for outpatient visits for CPP in the USA is $881.5 million per year. In the UK a community postal (questionnaire) survey of women of reproductive age reported a prevalence of 24% (Zonderman et al, 2001).

An integrated model of CPP, which provides an important base for its proper management, includes three domains. These are psychological, personality and social, all of which can influence the apparent intensity of painful stimulation (*Figure 1*). Descending influences from the brain modulate noxious input from the periphery and the perceived pain is not merely a sensation but a multidimensional experience involving the physiological, subjective and behavioural systems.

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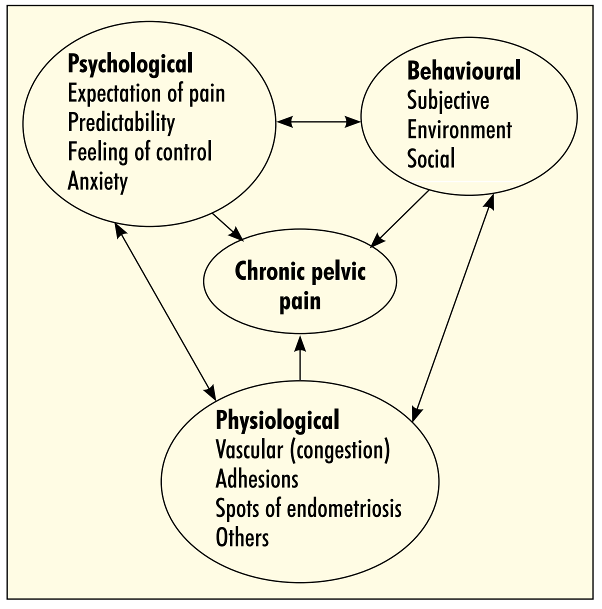
CPP patients have discordance in these three systems. The physiological component includes vascular changes or other pathology, e.g. adhesions or a small spot of endometriosis. The subjective component consists of afferent input from the physiological component, which can be labelled as pain under certain circumstances. Psychological variables are known to influence pain perception. These variables include individual expectation of pain, predictability, feeling of control over pain stimuli and anxiety. The behavioural component comprises responses that result from subjective perception of pain. Again psychological and social variables determine the nature and extent of behavioural responses. Parental behaviour or the presence of partners or caring friends, for example, can influence pain perception. Factors that maintain pain behaviour responses may not be the same as those that initiated them.

Aetiology and theory of aetiopathogenesis

The recognized causes of CPP are gynaecological, non-gynaecological, psychological and socioenvironmental. Although these may be isolated, an interplay between these factors is common (*Figure 2*). In a large proportion of women, however, CPP is idiopathic. Various theories have been proposed to explain the aetiopathogenesis of CPP.

Figure 1. Model for pelvic pain illustrating the complex interrelationships between various factors.



Theory 1: pathological

Undetected pathology proposes that there is a causal but undetectable pathology, either because it is in the early stages or current investigative techniques are not sensitive enough for its detection. Examples include laceration of uterine ligaments and subtle localized histological inflammation. The presence of a pathology does not necessarily imply a cause of CPP.

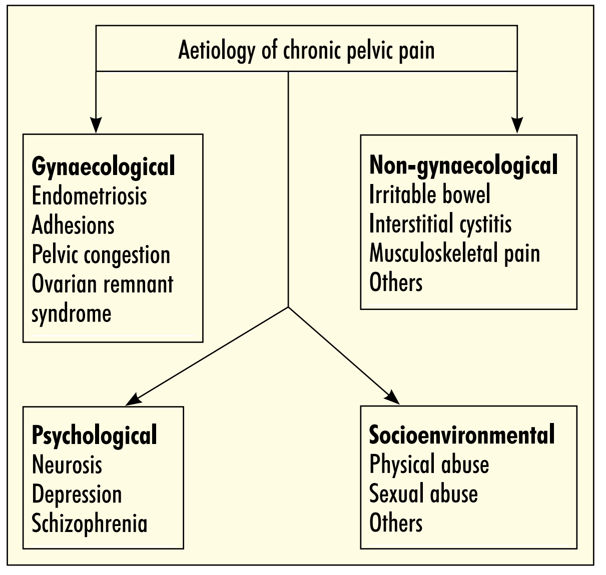
Theory 2: psychophysiological

Psychophysiological factors proposes that physiological changes not associated with pathology or disease processes (analogous to the aetiology of migraine or tension headache) may cause pelvic pain via psychological mechanisms. It has been suggested that such pain may result from stress-related pelvic vascular congestion.

Theory 3: psychogenic

This theory proposes that the pain is secondary to psychogenic factors (no sensory input has been demonstrated from the pelvis of these patients). In fact psychological abnormalities ranging from mild neurosis to schizophrenia in patients with CPP are thought to be explained by these theories which have been reported. Studies have demonstrated increased scores for hysteria, hypochondriasis, depression and neuroticism in women with CPP. Some of these have sexual difficulties and concerns about female identity and anxieties about sex. Gross et al (1980–81) reported that a significant proportion had been subjected to early traumatic sexual experiences like incest. These earlier reports have methodological problems as many patients were unmatched for the duration of pain, comparisons were often made between those with and without pathology or between women with pain and non-pain conditions. Overcoming these methodological problems has resulted in less enthusiasm for this theory. Psychological and personality disturbances are believed to be more likely a consequence rather than a cause of CPP.

Figure 2. Causes of chronic pelvic pain and their interactions.



Common types of CPP in gynaecology

Pelvic congestion syndrome

This is characterized by pelvic varicosities causing stasis and congestion within pelvic organs with resultant pain similar to that from varicose veins in the legs. Varicosities have been reported in 91% of women with unexplained pelvic pain but only rarely in pelvic pain-free controls (Beard et al, 1977). Pelvic congestion may be secondary to defective veins or to pelvic venous dilatation induced by vasoactive substances. Vasodilator properties of oestrogens have been suggested as responsible for these vascular changes. Although oestrogen levels are not high in these women, they often have uterine and endometrial hypertrophy suggesting an excess of oestrogen or an excessive response to it. That such women benefit from progesterone treatment or surgical removal of their ovaries supports this concept.

The typical woman with pelvic congestion syndrome is in her late twenties or early thirties, often multiparous, complaining of a dull aching pain in the pelvis, worsened by walking, standing, lifting or bending. There may be deep dyspareunia but more typically it is a postcoital ache. Dysfunctional uterine bleeding and congestive dysmenorrhoea are also common. There is often a history of acute episodes provoking hospital admission. Ovarian point tenderness can be elicited and ultrasound scan may reveal an enlarged uterus, with a thickened endometrium and multiple cysts in the ovaries. Usually at laparoscopy no lesions are found. Transuterine pelvic venography shows dilated ovarian veins and delay in the disappearance of the dye.

Ovarian remnant syndrome

This is defined as pelvic pain in the presence of residual ovarian tissue after a salpingo-oophorectomy. Incomplete extirpation is likely when the ovary is densely adherent to the pelvic side-walls, rectosigmoid and cul-de-sac by endometriosis, adhesions or pelvic inflammatory disease. As the residual ovarian tissue continues to function under gonadotrophin stimulation, cystic changes can cause pain by exerting pressure on the adjacent tissues.

Patient evaluation

History

A detailed history and thorough physical examination is important in women presenting with CPP (*Table 1*). Characteristic features of the pain including intensity, site, character, exacerbating and relieving factors, and the relationship to menstruation should be ascertained. It is also important to enquire about the coexistence of dysmenorrhoea and dyspareunia. Dyspareunia may be associated with any of the following: endometriosis, pelvic floor dysfunction, vulvodynia, interstitial cystitis and irritable bowel syndrome (Gunter, 2003).

Severe dysmenorrhoea, focal pelvic tenderness or deep dyspareunia with cyclical exacerbation are features suggestive of endometriosis. Direct questions regarding bowel and urinary symptoms are mandatory. Women with irritable bowel syndrome admit to alternating constipation and diarrhoea, abdominal distension, mucus per rectum, improvement in pain after bowel movement and a sensation of incomplete evacuation after defecation. Interstitial cystitis on the other hand manifests as pain, urgency, frequency, nocturia and a history of frequent culture negative urinary tract infections (Gunter, 2003).

Screening for psychological abnormalities is essential not only because of its aetiological importance but its impact on response to therapy (Gunter, 2003). A detailed social history is necessary to exclude domestic violence since victims have a higher incidence of chronic pain (Eisenstat and Bancroft, 1999).

Physical examination

A thorough abdominal examination may identify tender and trigger points. Patients should be asked to graphically depict their pain on a full torso diagram. The trigger points can then be confirmed by 'pinching subcutaneous tissues' beneath the point of maximal tenderness (Reiter, 1998). Careful inspection of the external genitalia should precede a pelvic examination. Pain and tenderness in the vestibular area in the absence of abnormality is characteristic of vulvodynia. A one finger digital examination should be performed to identify trigger points in the pelvic floor musculature and also to check for tenderness in the anterior vaginal wall – a feature of interstitial cystitis. This is followed by a gentle speculum examination and finally, a bimanual examination to assess the uterus and adenexa.

Table 1. Management of patients presenting with chronic pelvic pain

History	Pain characteristics
	Dysmenorrhoea
	Dyspareunia
	Bowel symptoms
	Bladder symptoms
	Depression
Examination	Abdominal
	Pelvic
	Trigger points
Investigations	Urine culture
	Vaginal swabs
	Laparoscopy
	Microlaparoscopy
	Hormone suppression
	Cystoscopy
Treatment	Reassurance after laparoscopy (to exclude pathology)
	Counselling and psychotherapy
	Progestogens
	Invasive treatment
	Hysterectomy with bilateral salpingo-oophorectomy
	Laparoscopic uterosacral nerve ablation or presacral neurectomy
	Trigger point injections

Investigations

Essential investigations include a mid-stream urine for culture and sensitivity, cervical and vaginal swabs to rule out pelvic infection and an ultrasound scan if clinically indicated. Laparoscopy is often undertaken by a gynaecologist in the UK. A major limitation of conventional laparoscopy is the risk of automatically regarding as causative any pathology directly visualized. On the other hand, patients with no visualized pathology are likely to be labelled disease free.

Microlaparoscopy performed with a 2 mm endoscope under local anaesthesia and supplemental intravenous sedation has been advocated (Palter, 1999). During the procedure, major pelvic organs are grasped systematically and areas of pathology probed. The patients rate the pain caused by the grasping on a scale of 0–10, allowing mapping. The sedation may, however, affect pain perception. There are currently no randomized controlled trials of this approach with conventional diagnostic laparoscopy.

In women with suspected endometriosis, administration of a short course of a gonadotrophin-releasing hormone (GnRH) agonist has been proposed as a diagnostic test. A cost–benefit analysis comparing this and laparoscopy for clinically diagnosed endometriosis concluded that GnRH agonists as a diagnostic tool will avoid laparoscopy in 50% of patients (Carter, 1995). Women with CPP and coexistent urinary symptoms such as dysuria, frequency, urgency, nocturia and a tender bladder base on vaginal examination may require a cystoscopy (to exclude interstitial cystitis) after a urine culture and sensitivity. This is best undertaken by urologists.

Management of chronic pelvic pain

Patients may fall into one of the following categories after investigations:

1. CPP with an identifiable pathology likely to be causative
2. CPP with an identifiable pathology but of uncertain significance, e.g. filmy adhesions or a small spot of endometriosis
3. CPP with no obvious pathology.

The precise proportion of women with CPP in the above categories is uncertain. However, Kresch et al (1984), in a comprehensive study of 100 British women undergoing laparoscopy for CPP, identified 31% with a pelvic pathology, the most common of which was adhesions (38%) and endometriosis (32%). Since treatment of women with CPP and an identifiable pelvic pathology is

determined by the latter, this review will focus mainly on the management of those with CPP and a negative laparoscopy, i.e. categories 2 and 3. The management of dysmenorrhoea is not discussed in keeping with the published literature on this subject.

Interventions undertaken for CPP are numerous and often unsatisfactory as is typical of conditions where the aetiology is not well defined. The Cochrane review (Stones and Mountfield, 2000) identified seven randomized controlled trials on interventions for CPP of which only four were of good methodological quality. The parameters that have been used to assess effectiveness in most studies are varied and include pain scores (VAS, McGill questionnaire) (Melzak, 1975), mood scales like hospital anxiety depression score, health-related quality of life measures like short form 36 health survey questionnaire, disturbance of daily activities like sexual function, time off work and resource utilization. The interventions discussed here include:

1. Reassurance after laparoscopy (to exclude pathology)
2. Counselling and psychotherapy
3. Progestogens
4. Invasive treatments:
   a. Hysterectomy with bilateral salpingooophorectomy
   b. Interruption of nerve pathways by laparoscopic uterosacral nerve ablation (LUNA) or presacral neurectomy (PSN)
   c. Trigger point injections.

Reassurance after laparoscopy

This may be all that is required in a group of women. Elcombe et al (1997) showed improvements in pain 3 and 6 months after a negative laparoscopy. Psychological variables like beliefs about pain and the change in each woman's evaluation of the seriousness of her condition were the only predictors of pain improvement identified. Although the study was non-randomized, short-term and the sample size was small, it highlighted the importance of psychological factors in the aetiology and management of CPP. Rasmussen et al (1993), in a longer term study, found that only 22% of such patients were subjectively free from pain and concluded therefore that with the exclusion of significant pathology laparoscopy was not in itself a satisfactory long-term treatment option.

Counselling and psychotherapy

This approach is best offered by a multidisciplinary team after a negative laparoscopy. Treatment commonly consists of counselling and reassurance (Albert, 1999). The multidisciplinary approach offers better long-term results than counselling by a single therapist.

Medroxyprogesterone

This has been shown to significantly improve pain scores especially in those with pelvic congestion syndrome (Farquhar et al, 1989). The effect of progestogens, however, is not long lasting.

Invasive treatment

Hysterectomy

There are no randomized trials assessing the effectiveness of hysterectomy for CPP although it is widely offered as a treatment option. In 308 women who underwent hysterectomy for CPP the symptoms resolved in 74%. However, a 40% recurrence rate was observed in those women in whom the hysterectomy was performed without identifiable pathology (Hillis et al, 1995). Total abdominal hysterectomy with bilateral salpingo-oophorectomy has been reported to be an effective treatment for pain as a result of pelvic congestion. In an observational study, Beard et al (1991) reported that 24 out of 36 patients had complete relief and 12 had some pain at 1 year. In the latter group only one patient had pain affecting daily life. Hysterectomy should therefore not be offered as the primary treatment as its failure to eradicate the psychological and emotional component of CPP often means a high recurrence rate. There is insufficient evidence to recommend hysterectomy for the majority of patients with CPP, especially in the absence of documented pathology.

Interruption of nerve pathways

PSN and LUNA are widely used in the treatment of CPP. A Cochrane review (Proctor et al, 2005) concluded that there was some evidence that LUNA and PSN were effective in CPP. In a well-controlled prospective study, PSN was found to achieve success rates of 73% in relieving dysmenorrhoea, 77% in relieving dyspareunia, and 63% in relieving other types of pelvic pain in women who failed to respond to the combined oral contraceptive pill and non-steroidal anti-inflammatory drugs (Lee et al, 1986). In a double-blind randomized controlled trial of LUNA for women with CPP, Johnson et al (2004) concluded that it was ineffective for non-dysmenorrhoeic CPP. The complications of PSN include catastrophic haemorrhage from the middle sacral vessels, long-term constipation and urinary retention and those of LUNA are damage or transection to the ureter and severe bleeding from the veins, which lie just medial to the uterosacral ligament.

Trigger point injections

This has been used to treat myofascial abdominal wall pain. Agents which have been used for this include long-acting local anaesthetic agents such as bupivacaine and botulinum toxin A. The genitofemoral and ilioinguinal nerves might be implicated in groin pain post-hysterectomy and an appropriate nerve block with a long-acting anaesthetic may be therapeutic (Gunter, 2003).

Conclusions

CPP is a poorly understood enigmatic condition with a diverse aetiology and may involve more than one organ system. An integrated multidisciplinary approach is essential in the evaluation and management of an individual with CPP. Good communication from the initial consultation with the GP can contribute to a successful outcome.

Available good quality evidence of effective treatment of CPP is limited. Management often requires a combination of therapeutic interventions, namely psychological, medical and surgical. A systematic approach starting in the community and culminating in a coordinated multidisciplinary standard approach will result in successful treatment in a significant proportion of women. BJHM

Conflict of interest: none.

Albert H (1999) Psychosomatic group treatment helps women with chronic pelvic pain. *J Psychosom Obstet Gynecol* **20:** 216–25

Beard RW, Besley EM, Leiberman BA, Wilkinson JC (1977) Pelvic pain in women. *AM J Obstet Gynecol* **128**: 566–70

Beard RW, Kennedy RG, Gangar KF (1991) Bilateral oophorectomy and hysterectomy in the treatment of intractable pelvic pain associated with pelvic congestion. *Br J Obstet Gynecol* **98:** 988–92

Carter JE (1995) Laparoscopic treatment of chronic pelvic pain in 100 adult women. *J Am Assoc Gynecol Laparoscop* **2:** 255–62

KEY POINTS

- Chronic pelvic pain (CPP) is defined as pelvic pain of at least 6 months' duration and affects about 15% of women.
- The aetiology is diverse in origin and may be idiopathic or related to an organ system.
- The aetiopathogenesis is poorly understood and may be an interplay of psychological, physiological and social factors.
- Patient evaluation must include a detailed history, thorough examination and laparoscopy.
- Microlaparoscopy is an attractive option but its usefulness has to be substantiated by randomized trials.
- Counselling and psychotherapy form an important aspect of management.
- The effectiveness of hysterectomy for idiopathic CPP is debatable.
- Laparoscopic uterosacral nerve ablation and presacral neurectomy have been shown to be of benefit in some cases of CPP.
- Trigger point injections are effective to treat myofascial pain.
- A multidisciplinary approach by a dedicated specialized team will provide the most successful management.

Eisenstat SA, Bancroft L (1999) Domestic violence. *N Engl J Med* **341:** 886–92

Elcombe S, Gath D, Day A (1997) The psychological effects of laparoscopy on women with chronic pelvic pain. *Psychol Med* **27:** 1041–50

Farquhar CM, Rogers V, Franks S, Pearce S, Wadsworth J, Beard RW (1989) A randomized controlled trial of medroxy progesterone acetate and psychotherapy for the treatment of pelvic congestion. *Br J Obstet Gynaecol* **96:** 1153–62

Gross RJ, Doerr H, Caldirola D, Guzinski GM, Ripley HS (1980–81) Borderline syndrome and incest in chronic pelvic pain patients. *Int J Psychiatry Med* **10**: 79–96

Gunter J (2003) Chronic pelvic pain: an integrated approach to diagnosis and treatment. *Obstet Gynecol Surv* **58:** 615–23

Hillis SD, Marchbanks PA, Peterson HB (1995) The effective of hysterectomy for chronic pelvic pain. *Obstet Gynecol* **86:** 941–5

Johnson NP, Farquhar CM, Crossley S, Yu Y, Van Peperstraten AM, Sprecher M, Suckling J (2004) A doubled-blind randomised controlled trial of laparoscopic uterine nerve ablation for women with chronic pelvic pain. *Br J Obstet Gynaecol* **111:** 950–9

Kresch AJ, Seifer DB, Sachs LB, Barrese I (1984) Laparoscopy in 100 women with chronic pelvic pain. *Obstet Gynecol* **64**: 672–4

Lee RB, Stone K, Magelseen D, Belts RP, Benson WL (1986).Presacral neurectomy for chronic pelvic pain. *Obstet Gynecol* **68**: 517–21

Mathias SD, Kupperman M, Liberman RF, Lipschutz RC, Steege JE (1996) Chronic pelvic pain: prevalence, health related quality of life, and economic correlates. *Obstet Gynecol* **87:** 321–7

Melzak R (1955) The McGill questionnaire: major properties and scoring methods. *Pain* **1**: 277–99

Palter SF (1999) Microlaparoscopy under local anaesthesia and conscious pain mapping for the diagnosis and management of pelvic pain. *Current Opin Obstet Gynaecol* **11:** 387–93

Prentice A (2000) Medical management of chronic pelvic pain. *Baillieres Best Pract Res Clin Obstet Gynecol* **14**: 495–9

Proctor ML, Latthe PM, Farquhar CM, Khan KS, Johnson NP (2005) Surgical interruption of pelvic nerve pathway for primary and secondary dysmenorrhoea. *Cochrane Database Syst Rev* **2005(4):** CD001896

Rasmussen KL, Hansen RM, Hansen K, Rasmussen ID (1993) Long term prognosis for women with chronic pelvic pain without laparoscopically demonstrable cause. *Ugeskr Laeger* **155:** 849–51

Reiter RC (1998) Evidence-based management of chronic pelvic pain. *Clin Obstet Gynecol* **41:** 422–35

Stones RW, Mountfield J (2000) Interventions for treating chronic pelvic pain in women. *Cochrane Database Syst Rev* **2000(4):** CD000387

Zonderman KT, Yudkin PL, Vessey MP et al (2001) The community prevalence of chronic pelvic pain in women and associated illness behaviour. *Br J Gen Pract* **51:** 541–7