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Diagnosis and classification of pelvic girdle pain disorders—Part 1: A mechanism based approach within a biopsychosocial framework

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Abstract

The diagnosis and classification of pelvic girdle pain (PGP) disorders remains controversial despite a proliferation of research into this field. The majority of PGP disorders have no identified pathoanatomical basis leaving a management vacuum. Diagnostic and treatment paradigms for PGP disorders exist although many of these approaches have limited validity and are uni-dimensional (i.e. biomechanical) in nature. Furthermore single approaches for the management of PGP fail to benefit all. This highlights the possibility that 'non-specific' PGP disorders are represented by a number of sub-groups with different underlying pain mechanisms rather than a single entity.

This paper examines the current knowledge and challenges some of the common beliefs regarding the sacroiliac joints and pelvic function. A hypothetical 'mechanism based' classification system for PGP, based within a biopsychosocial framework is proposed. This has developed from a synthesis of the current evidence combined with the clinical observations of the authors. It recognises the presence of both specific and non-specific musculoskeletal PGP disorders. It acknowledges the complex and multifactorial nature of chronic PGP disorders and the potential of both the peripheral and central nervous system to promote and modulate pain. It is proposed that there is a large group of predominantly peripherally mediated PGP disorders which are associated with either 'reduced' or 'excessive' force closure of the pelvis, resulting in abnormal stresses on pain sensitive pelvic structures. It acknowledges that the interaction of psychosocial factors (such as passive coping strategies, faulty beliefs, anxiety and depression) in these pain disorders has the potential to promote pain and disability. It also acknowledges the complex interaction that hormonal factors may play in these pain disorders. This classification model is flexible and helps guide appropriate management of these disorders within a biopsychosocial framework. While the validity of this approach is emerging, further research is required.

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1. Pelvic girdle pain disorders

Pelvic girdle pain (PGP) disorders represent a small but significant group of musculoskeletal pain disorders. Pain associated with the sacroiliac joints (SIJs) and/or the surrounding musculoskeletal and ligamentous structures represent a sub-group of these disorders. *Specific* inflammatory pain disorders of the SIJs, such as sacroiliitis, are the most readily identified PGP disorders (Maksymowych et al., 2005). However, PGP disorders more commonly present as '*non-specific*' (no identified pathoanatomical basis), often arising during or shortly after pregnancy (Berg et al., 1988; Ostgaard et al., 1991; Bastiaanssen et al., 2005) or following traumatic injury to the pelvis (O'Sullivan et al., 2002a; Chou et al., 2004). Frequently these pain disorders are misdiagnosed and managed as lumbar spine disorders, as pain originating from the lumbar spine commonly refers to the SIJ region. However, there is growing evidence that PGP disorders manifest as a separate sub-group with a unique clinical presentation and the need for specific management.

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A number of PGP disorders do not resolve (Ostgaard et al., 1996; Larsen et al., 1999; Albert et al., 2001; Noren et al., 2002; To and Wong, 2003), becoming chronic despite the absence of pathoanatomical abnormalities on radiological examination or signs of a systemic or inflammatory disorder from blood screening (Hansen et al., 2005). This leads to a broad diagnosis of a 'non-specific' PGP disorder and leaves a diagnostic and management vacuum. These PGP disorders are commonly associated with signs and symptoms indicating that the pain originates from the SIJs and/or their surrounding connective tissue and myo-fascial structures (Berg et al., 1988; Kristiansson and Svardsudd, 1996; Mens et al., 1999; Albert et al., 2000; Damen et al., 2001; Vleeming et al., 2002; O'Sullivan et al., 2002a; Laslett et al., 2003). However, identification of a painful structure does not provide insight into the underlying mechanism(s) that drives the pain (O'Sullivan, 2005a).

A number of theoretical models have been proposed with regard to potential underlying pain mechanisms in PGP. Chiropractic, Osteopathic and Manual Therapy models commonly propose that the SIJs can become 'fixated' or 'displaced' leading to positional faults. There are a series of complex clinical procedures proposed to identify these so-called 'positional faults' and treatment with manipulation, mobilisation and/or muscle energy techniques has been suggested to rectify them (Don-Tigny, 1990; Sandler, 1996; Kuchera, 1997; Oldreive, 1998; Cibulka, 2002). Although manual and manipulative techniques can result in short term pain modulation (Wright, 1995), there is little evidence for the long term benefits of SIJ manipulation or other passive treatments used in isolation for the management of chronic PGP disorders (Stuge et al., 2003). The selection of these techniques is often directed by treating the signs and symptoms of the disorder rather than a valid and clear diagnostic and classification paradigm based on the mechanisms that underlie the pain disorder.

More recently emphasis has been placed on enhancing motor control deficits in PGP disorders. This is based on the premise that deficits in lumbo-pelvic motor control result in impaired load transference through the pelvis and thereby contribute to a peripheral nociceptive drive of symptoms (Mens et al., 1996; Vleeming et al., 1996, 1990b; O'Sullivan et al., 2002a; O'Sullivan and Beales, 2007). There is growing evidence based on outcome studies that some PGP disorders do indeed respond well to specifically targeted motor training interventions (Stuge et al., 2004a, b; O'Sullivan and Beales, 2007). However, not all PGP disorders respond to these interventions (Stuge et al., 2006). Relevant to this inconsistency in outcome, is the existence of different patterns of motor control impairments in PGP subjects. For instance increased pelvic floor activation has been documented in subjects with peripartum PGP consistent with SIJ involvement (Pool-Goudzwaard et al., 2005),

while another group of subjects with SIJ pain (with a positive active straight leg raise test (ASLR)) demonstrate impaired control of the pelvic floor (O'Sullivan et al., 2002a; O'Sullivan and Beales, 2007). These findings highlight that; (i) there may be various underlying mechanisms that drive different PGP disorders, and (ii) the need for a classification based approach which guides targeted interventions for sub-groups of subjects with PGP, which is based upon the underlying pain mechanism(s) that drives the disorder.

2. Challenging the beliefs regarding the sacroiliac joints and the pelvis

The SIJ perhaps more than any other joint complex in the body has been shrouded by an enormous amount of mystique within the field of Manual Therapy—with complex, poorly validated and often confusing theories and treatment approaches associated with it. Beliefs of the clinician (that the pelvis is 'displaced' or 'unstable') commonly become the beliefs of the patients. For many patients these clinical labels can be detrimental with the potential to render the patient passively dependent on someone to 'fix them', elevating anxiety levels, reinforcing avoidance behaviours and promoting disability. Increased passive dependence and fear/anxiety has the potential to further increase the central drive of pain, contributing to disability and the chronic pain cycle. It is therefore important to be clear on the *'facts'* regarding the SIJs and put them into the context of current knowledge. The basic anatomy, biomechanics and stability models proposed for the SIJ are documented elsewhere and as such will not be reviewed in full here (Pool-Goudzwaard et al., 1998; Lee and Vleeming, 2000; Vleeming et al., 2006).

2.1. The facts regarding the SIJs

- The SIJs are inherently stable (Vleeming et al., 1990a, b; Snijders et al., 1993a).
- The joints are designed for load transfer (Kapandji, 1982; Gray and Williams, 1989) and can safely transfer enormous compressive loading forces under normal conditions (Snijders et al., 1993a).
- The SIJ has little movement in non-weight bearing (average 2.5 degrees rotation) (Sturesson et al., 1989; Brunner et al., 1991; Jacob and Kissling, 1995; Vleeming et al., 1992a, b), and even less in weight bearing (average 0.2 degrees rotation) (Sturesson et al., 2000).
- Movement of the SIJ cannot be reliably assessed by manual palpation, particularly in weight bearing (Sturesson et al., 2000; van der Wurff et al., 2000a, b).
- Due to its anatomical makeup, intra-articular displacements within the SIJs are unlikely to occur. No

study utilising a valid measurement instrument has identified positional faults of the SIJ—in fact the converse is true (Tullberg et al., 1998).

- Distortions of the pelvis observed clinically are likely to occur secondary to changes in pelvic and trunk muscle activity, resulting in directional strain and not positional changes within the SIJs themselves (Tullberg et al., 1998).
- No study utilising a valid measurement tool has demonstrated that pelvic manipulation alters the position of the pelvic joints (Tullberg et al., 1998)—pain relief from these procedures is likely to result from nociceptive inhibition based on neuro-inhibitory factors and/or altered patterns of motor activity (Wright, 1995; Pickar, 2002).
- Asymmetrical laxity of the SIJs, as measured with Doppler imaging, has been shown to correlate with moderate to severe levels of symptoms in subjects with peripartum PGP (Damen et al., 2001). Generalised SIJ laxity is not associated with peripartum pelvic pain (Damen et al., 2001).
- When clinical signs of reduced force closure have been identified (positive ASLR), the increased movement is identified at the symphysis pubis—not the SIJs (Mens et al., 1999). It is likely that the torsional forces occurring at the SIJs can cause strain across pain sensitised tissue.
- Pain from the SIJ is located primarily over the joint (inferior sulcus) and may refer distally, but not to the low back (Fortin et al., 1994a, b; Schwarzer et al., 1995; Dreyfuss et al., 1996; Maigne et al., 1996; Slipman et al., 2000; Young et al., 2003; van der Wurff et al., 2006).
- SIJ pain disorders can be diagnosed using clinical examination (Laslett et al., 2003; Young et al., 2003; Petersen et al., 2004; Laslett et al., 2005a, b). This includes the finding of pain primarily located to the inferior sulcus of the SIJs, positive pain provocation tests for the SIJs and an absence of painful lumbar spine impairment.
- The SIJ has many muscles that act to compress and control it (force closure), thereby enhancing pelvic stability (creating stiffness) allowing for effective load transfer via the pelvis during a variety of functional tasks (Vleeming et al., 1990a, b, 1995; Snijders et al., 1993a, b; ; Snijders et al., 1998; Damen et al., 2002; Richardson et al., 2002; O'Sullivan et al., 2002a; Pool-Goudzwaard et al., 2004; van Wingerden et al., 2004; Mens et al., 2006; Snijders et al., 2006).
- PGP disorders may be associated with 'excessive' as well as 'insufficient' motor activation of the lumbopelvic and surrounding musculature (O'Sullivan et al., 2002a; Hungerford et al., 2003; Pool-Goudzwaard et al., 2005; O'Sullivan and Beales, 2007).

3. Classification of pelvic girdle pain disorders

Chronic pain disorders are complex, multifactorial and need to be considered within a biopsychosocial framework. A different cluster of potential physical, pathoanatomical, psychosocial, hormonal and neurophysiological factors is associated with each disorder (Fig. 1). Needless to say the interactions between these factors are very complex. This highlights the need for a flexible classification and management approach for each disorder.

Although the SIJs and the surrounding ligamentous and myofascial structures are potentially nociceptive structures (Fortin et al., 1994a, b; Vilensky et al., 2002), from a neurophysiologic perspective it is well known that ongoing pain can be mediated both peripherally and centrally, and the forebrain can greatly modulate this process (Zusman, 2002; Woolf, 2004). It is therefore logical that PGP disorders can potentially be both peripherally or centrally induced/maintained, with a different balance or dominance of peripheral and central factors associated with each disorder (Elvey and O'Sullivan, 2005).

Furthermore with PGP there is the potential contributing role of sex hormones. There are a number of possible pathways by which hormones may influence PGP (Fig. 2). There is some evidence that sex hormones are active in pain modulation (Aloisi and Bonifazi, 2006). Sex hormones are also known to influence the inflammatory process in inflammatory pain disorders (Schmidt et al., 2006). Furthermore sex hormones may alter collagen synthesis (Kristiansson et al., 1999), thereby effecting the load capacity of the pelvis. There is some evidence to support the role of hormones in PGP disorders, with higher serum levels of progesterone and relaxin in early pregnancy being found in subjects who develop peripartum PGP compared to those who do not (Kristiansson et al., 1999). Via these processes sex hormones have the potential to contribute to PGP in different clinical presentations (Fig. 2). Further research is required to clarify how the role of hormones may differ in these various presentations of PGP.

The proposed classification model for PGP disorders is based on the potential mechanisms that can drive the PGP. This classification approach is not exhaustive but rather provides a framework to guide the clinician. Based on the mechanism(s) that underlie these disorders and operating within a biopsychosocial framework, the classification model aims to facilitate the diagnosis, classification (Fig. 3), and targeted management of these disorders.

3.1. The clinical examination

The clinical examination is critical to the clinical reasoning process that underpins this diagnosis and

Patho-anatomical factors
- structural pathology
- ligamentous laxity
- identification of peripheral pain generator (sacroiliac joint / pubic symphysis, myofascial / connective tissue)

Physical factors
- mechanism of injury if present
- disorder history (pregnancy related)
- disorder stage – acute, sub-acute, chronic
- area of pain – local / generalised / referred
- pain behaviour – intermittent vs constant
  – provocative and relieving factors
- mechanical vs non-mechanical provocation
- +ve active straight leg raise
- SIJ provocation tests
- adaptive vs mal-adaptive movement behaviours
- motor control impairments (↑ or ↓ motor activation)
- disability levels
- activity levels / conditioning / strength / muscle endurance
- work / home environment / lifestyle
- ergonomic factors

Genetic factors
- potentially influencing all other domains

Pelvic Girdle Pain

Neuro-physiological factors
- peripheral sensitisation
- hormonal factors
- central sensitisation
- sympathetic nervous system activity
- somatic complaints
- glial cell activation

Social factors
- relationships – family, friends, work
- caring for children
- work issues
- medical advice and treatment
- support structures
- compensation (emotional, financial)
- cultural factors
- socio-economic factors
- stress

Psychological factors
- personality type
- beliefs & attitudes towards pain disorder
- coping strategies – passive vs active
- hyper-vigilance
- pacing
- fear avoidance behaviour
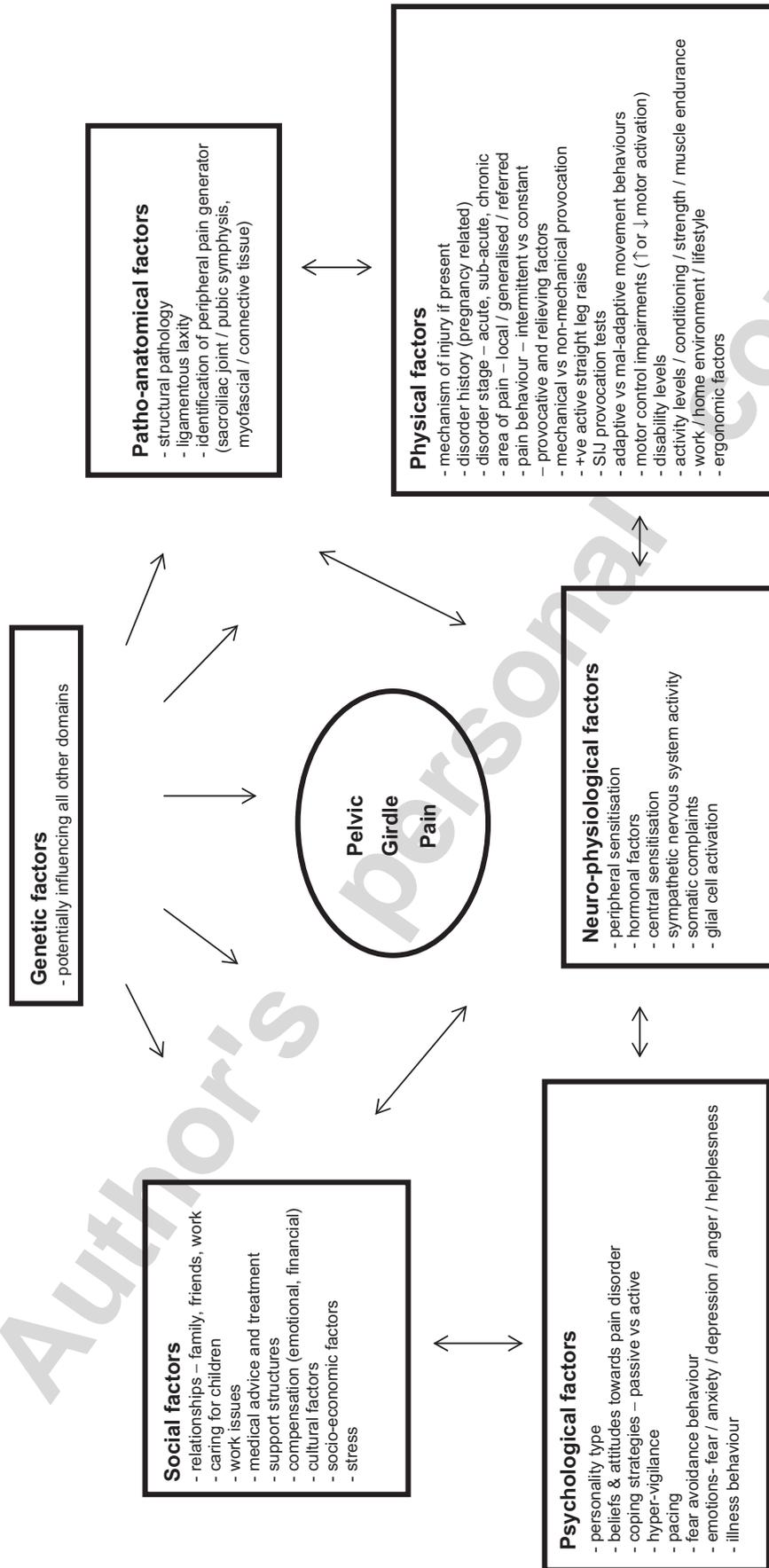- emotions- fear / anxiety / depression / anger / helplessness
- illness behaviour

Fig. 1. Factors that need consideration within a biopsychosocial framework for the diagnosis and classification of chronic pelvic girdle pain disorders.
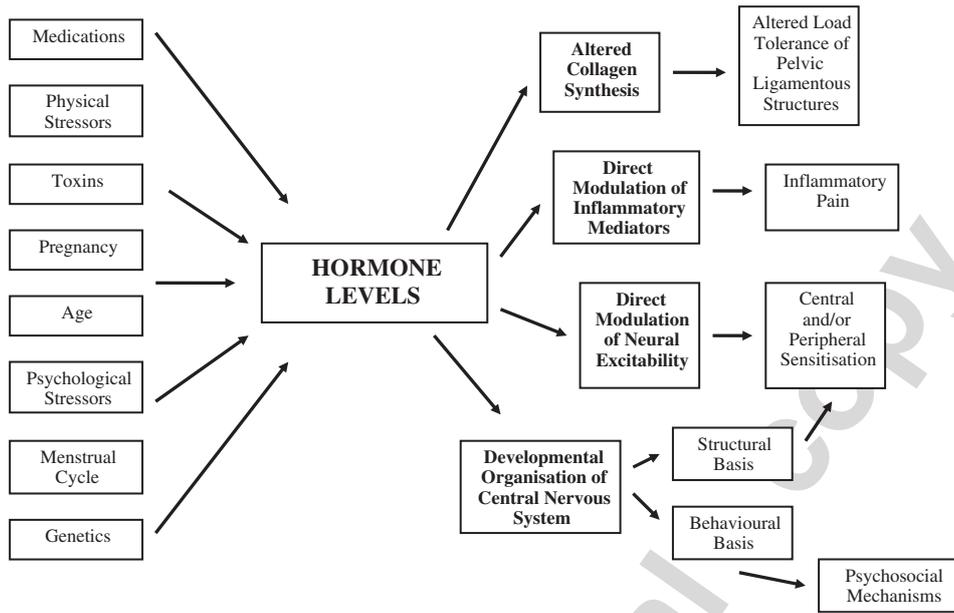
Fig. 2. Possible actions of hormones in the development and maintenance of pelvic girdle pain. Factors affecting hormone levels are also presented.
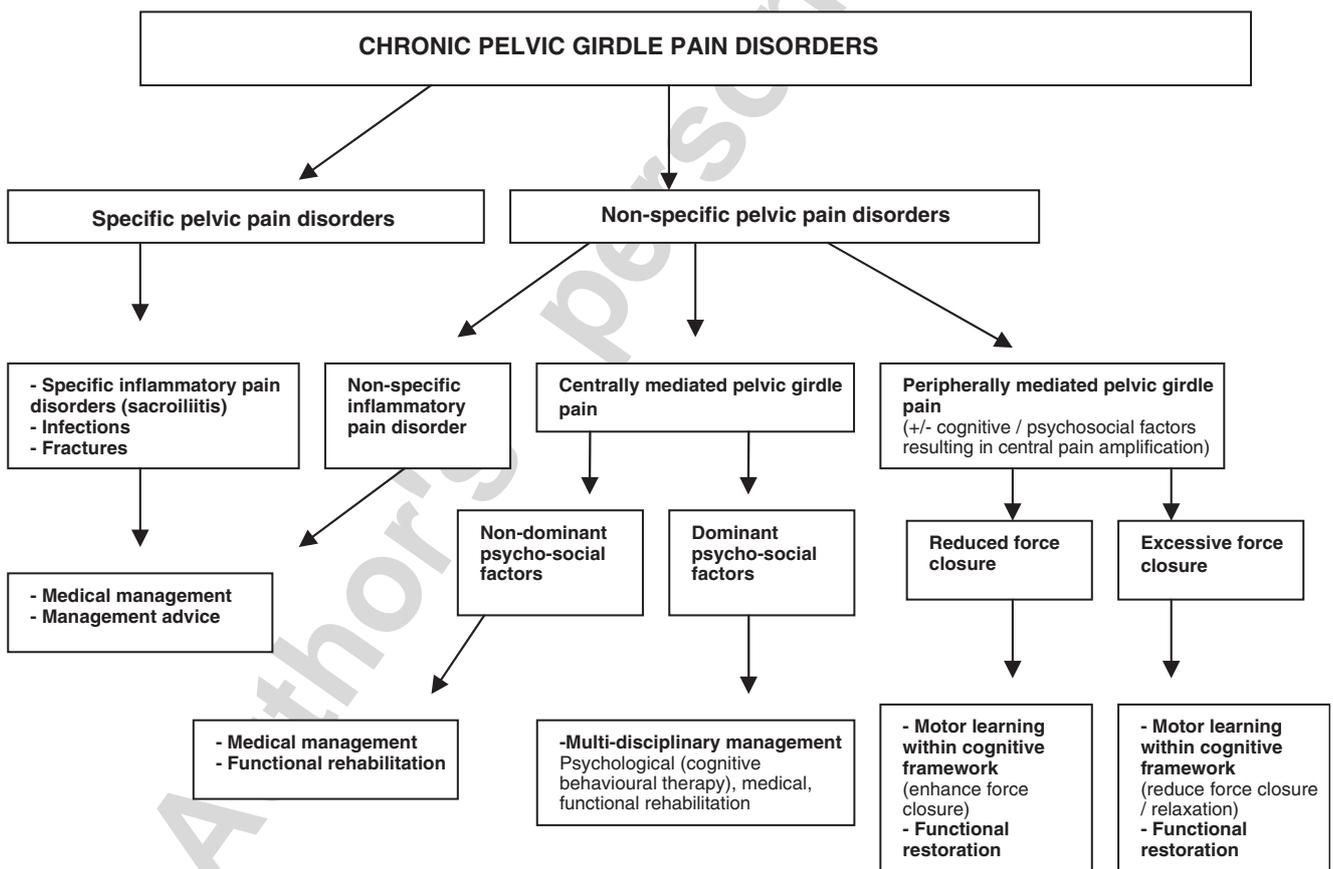
Fig. 3. Mechanism based classification and management of chronic pelvic girdle pain disorders.

classification framework. In the interview process all the following need to be considered:

- the pain area (localised versus generalised pain can indicate peripheral from central pain drive),

- pain pattern (intermittent versus constant, 24 hour pain pattern, sleep disturbances),
- pain intensity,
- pain behaviour (specific movements and postures that provoke and relieve pain),

- levels of disability and impairment,
- specific pain history (specific and surrounding events that may have contributed to the development of symptoms),
- family history of PGP,
- the patient's pain coping strategies (active versus passive coping),
- the patient's pain beliefs,
- presence of avoidant behaviours due to fear of movement and other psychosocial factors including present and past history of anxiety and depression,
- pacing patterns and
- concurrent presence of disorders of continence and/ or sexual dysfunction.

Review of radiology if present and screening for specific causes of PGP may be indicated from this process. This allows for a determination as to the area and nature of the pain.

A thorough physical examination is then required to determine the pain source and behaviour in relationship to the patient's movement behaviour. Physical tests should include:

- Palpation of the inferior sulcus of the SIJ and surrounding pelvic ligamentous and myo-fascial structures.
- Provocative tests for the SIJ and surrounding ligamentous and myofascial structures (Laslett et al., 2003, 2005a, b; Young et al., 2003; Petersen et al., 2004).
- The ASLR test in supine and prone as a test of load transfer, with a positive test resulting in normalisation of ASLR with the addition of pelvic compression (Mens et al., 1999; O'Sullivan and Beales, 2007).
- Careful analysis of the pain provoking and relieving activities and postures (functional impairments) highlighted from the interview to identify the presence of impairments of movement and motor control as well as avoidance behaviours and to determine their relationship to the pain disorder. Determining whether altered motor patterns are adaptive/protective (pain is aggravated when motor control patterns are normalised) or mal-adaptive (pain is relieved when motor control deficits are normalised) is essential.
- Tests for specific muscle function for the pelvic floor, the abdominal wall, the back muscles, iliopsoas, quadratus lumborum, the gluteal muscles and piriformis.

In addition the adjacent areas of the lumbar spine (including neural tissue) and hip joints should be thoroughly investigated to rule out involvement of these areas or to assess for coexisting pathology/dysfunction in these regions.

Correlating the patient's reported pain behaviour, beliefs and levels of impairment with his/her clinical presentation (observing for avoidance behaviours, catastrophising, etc.) is important to determine whether cognitive issues such as fear of movement are present and dominant. On synthesis of this material a diagnosis and classification of the PGP disorder can be made.

4. Specific pelvic girdle pain disorders

Pelvic girdle pain disorders associated with *specific* pathological processes include inflammatory arthritis, sacroiliitis, infections and fractures. These disorders are amenable to specific diagnosis with appropriate blood screening and radiological investigation. They can be associated with altered patterns of motor control behaviour that are *'adaptive'* and/or protective of the underlying disorder. Treating the signs and symptoms of these disorders by manual therapy and/or specific exercise interventions is generally not appropriate as it does not address the underlying pain mechanism of the disorder. Physiotherapy may be limited to management of the sequelae of the underlying disease/pathological processes especially in disorders such as ankylosing spondylitis.

5. Non-specific pelvic girdle pain disorders

5.1. Non-specific inflammatory pelvic girdle pain disorders

There appears to be a group of PGP disorders that present as being inflammatory in nature, rather than mechanical. They are characterised by constant, disabling and non-remitting pain, located in the SIJs, that is provoked with weight bearing, pelvic compression (such as a SIJ belt) and with SIJ pain provocation tests. These disorders may show areas of increased uptake on bone scan but are not linked to a specific inflammatory disorder diagnosis based on blood screening. They may be relieved with rest, anti-inflammatory medications and local steroid injections to the SIJ, but are resistant to physical interventions.

Although the exact underlying mechanism for these PGP disorders is unknown it is possible that hormonal factors play a role, particularly given their common onset in the first trimester of pregnancy or pain modulation with hormonal cycles or changes. Although the role of sex hormones is purely speculative in this group of patients, further research into their effect is warranted.

5.2. Peripherally mediated (mechanically induced) pelvic girdle pain disorders

These disorders are characterised by localised pain that has a defined anatomical location (SIJ and associated connective tissue and myofascial structures +/−symphysis pubis). The pain is intermittent in nature and is provoked and relieved by specific postures and activities related to vertical or directional loading in weight bearing positions. They are not usually asso-

ciated with spinal movement related pain and/or spinal movement impairment. A specific pain source at the SIJ and its surrounding structures can usually be identified by specific provocative manual tests (Laslett et al., 2003, 2005a, b; Young et al., 2003; Petersen et al., 2004). These disorders are usually associated with consistent local motor control changes (inhibition or excitation). These disorders usually have a clear mechanism or time of onset (either repeated strain or direct trauma to the pelvis or peripartum PGP). It is proposed that these

Mal-adaptive chronic pelvic girdle pain disorders → where motor control impairments represent dominant underlying driving mechanism for pain

Tissue injury / localised pain

Motor response

Factors that may influence pain and motor response

pathoanatomical
ligamentous laxity
physical
motor control
neurophysiological
hormonal
psychosocial
coping strategies
beliefs
fear avoidance
compensation
genetic

Resolution of the disorder

Non resolution
mal-adaptive patterns adopted
poor coping strategies
prolonged neuromuscular response
excessive ↔ reduced force closure

abnormal tissue loading

Excessive force closure classification
- hyper-activity of pelvic muscles with excessive joint compression

Management
- education – regarding pain mechanism
  - identify factors that drive motor system
  - cognitive behavioural approach
- relaxation of motor system
- relaxation strategies
- graded movement restoration
- functional restoration
- normalise movement behaviour

Reduced force closure classification
- motor control deficit of pelvic stabilising muscles with loss of force closure

Management
- education – regarding pain mechanism
- cognitive behavioural motor control intervention
- pain control (avoid provocation)
- specific motor activation
- retrain faulty postures and movements
- normalise movement behaviour
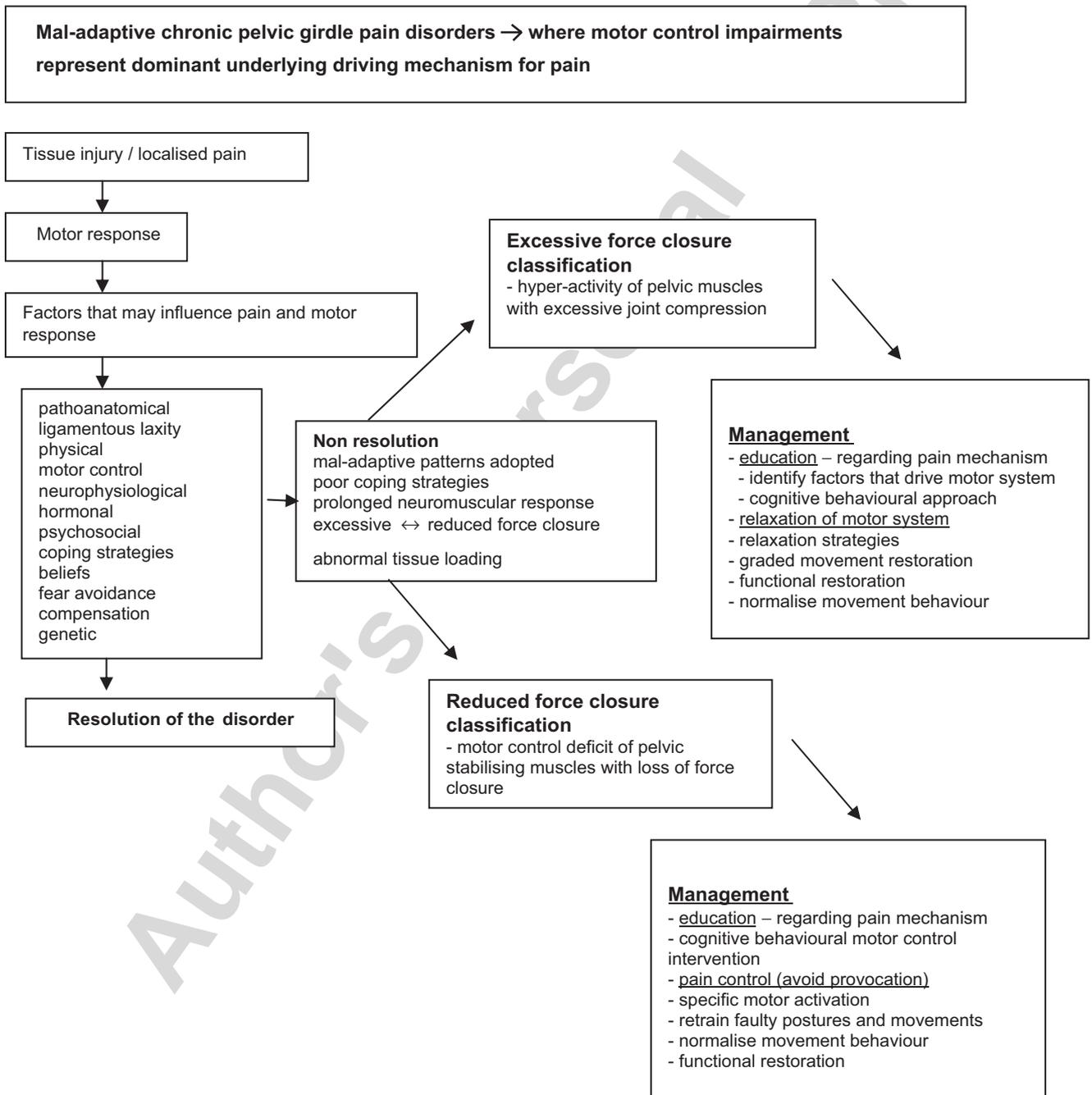- functional restoration

Fig. 4. Sub-classification of pelvic girdle pain disorders with a primary peripheral nociceptive drive. Peripheral drive is perpetuated by mal-adaptive motor control dysfunctions.

disorders may be classified into two clinical subgroups (Fig. 4).

5.2.1. Reduced force closure

The first group represents disorders where the peripheral pain drive is associated with excessive strain to the sensitised SIJs and/or surrounding connective tissue and myofascial structures secondary to ligamentous laxity (Damen et al., 2001), coupled with *motor control deficits* of muscles that control force closure of the SIJs (O'Sullivan et al., 2002a; Hungerford et al., 2003; O'Sullivan and Beales, 2007). These motor control deficits may have originally developed secondary to the pain disorder, but now their presence is *mal-adaptive* as the resultant 'reduced forced closure' leads to impaired load transfer through the pelvis, acting as a mechanism for ongoing strain and peripheral nociceptive drive for the pain disorder. Hormonal influences on collagen synthesis may be an important factor in this group.

These disorders are commonly associated with post-partum PGP and present with a positive ASLR test (normalised with pelvic compression) (O'Sullivan et al., 2002a; Stuge et al., 2004a). The motor control deficits that present in these disorders are variable and are linked to a loss of functional patterns of co-contraction of the local force closure muscles of the pelvis (such as the pelvic floor, the transverse abdominal wall, the lumbar multifidus, iliopsoas and the gluteal muscles). This is commonly associated with attempts to stabilise the lumbopelvic region via co-activation of other trunk muscles (quadratus lumborum, thoracic erector spinae, diaphragm, external oblique, rectus abdominis and vertical fibres of internal oblique). Their primary functional impairments are associated with pain in weight bearing postures such as sitting, standing and walking, or loaded activities inducing rotational pelvic strain associated with coupled spine/hip loading activities (i.e. cycling and rowing resulting in posterior rotational strain on ilium). These patients commonly assume postures that are associated with inhibition of the local pelvic muscles (pelvic floor, transverse abdominal wall, lumbar multifidus and the gluteal muscles) such as 'sway' standing, 'hanging off one leg', 'slump' sitting or 'thoracic upright' sitting (O'Sullivan et al., 2002b, 2006; Dankaerts et al., 2006; Sapsford et al., 2006) and present with a loss of lumbopelvic control (inability to disassociate pelvic from thoracic movement). These disorders may be relieved with a SIJ belt (Ostgaard et al., 1994; Mens et al., 2006), training optimal alignment of their spino-pelvic posture and functional enhancement of local co-contraction strategies across the pelvis with relaxation of the thoraco-pelvic musculature (O'Sullivan and Beales, 2007). These disorders may gain short term relief from mobilisation, muscle energy techniques, soft tissue massage and manipulation of the SIJs (clinical observation) although these in isolation tend not to benefit the long term outcome of the disorder. There is evidence that long lever exercise regimes may aggravate these disorders (Mens et al., 2000). These disorders can be further subgrouped based on their pattern of motor control dysfunction. Different combinations of motor control deficits may be found within the local lumbopelvic muscles such as is observed in low back pain disorders that result in different directional (vertical, rotational) strain patterns within the pelvis (O'Sullivan, 2005b).

Management of these disorders focuses on functionally enhancing force closure across the pelvic structures based on the specific motor control deficits present. The aim of the intervention is to provide functional activation of the motor system in order to control pain and restore functional capacity (Fig. 4). There is good evidence to support the efficacy of this type of approach in these disorders (Stuge et al., 2004a, b; O'Sullivan and Beales, 2007).

5.2.2. Excessive force closure

The second group is defined by a group of PGP disorders where the peripheral nociceptive drive is based on excessive, abnormal and sustained loading of sensitised pelvic structures (SIJs and surrounding connective tissue and/or myofascial structures) from the *excessive activation of the motor system local to the pelvis (excessive force closure)*. This patient group presents with localised pain to the SIJs and commonly also the surrounding connective tissue and myo-fascial structures (such as the pelvic floor and piriformis muscles) as well as positive pain provocation tests. However this group of patients has a negative ASLR (no feeling of heaviness). Compression (manual or using a SIJ belt), is often provocative, as is local muscle activation (pelvic floor, transverse abdominal wall, back muscles, iliopsoas, gluteal muscles). They commonly hold habitual erect lordotic lumbopelvic postures associated with high levels of co-contraction across various muscles such as the abdominal wall, pelvic floor, local spinal muscles (lumbar multifidus, psoas major) and in some cases the gluteal and piriformis muscles which may become pain sensitised. These motor control responses often become habitual secondary to excessive cognitive muscle training and/or muscle guarding of the lumbopelvic muscles, and are themselves *mal-adaptive* (provocative). These patients report pain relief from cardiovascular exercise, relaxation, assuming passive spinal postures (which they seldom do), as well as short-term relief with stretching, soft tissue massage, manipulation, muscle energy techniques and cessation of stabilisation exercises. These disorders are commonly associated with the patient's belief that their pelvis is 'unstable' or 'displaced' and that more muscle contraction or 'pelvic re-alignment' is beneficial. This is commonly reinforced by the treating therapist's beliefs. These disorders may be induced by

intensive 'stabilisation exercises', Pilates, ball exercise, and cognitive muscle exercise training of the abdominal wall, lumbar multifidus and pelvic floor. Patients with these disorders are commonly anxious, under high levels of stress, highly active and seldom rest.

Management of these disorders focuses on reducing force closure across the pelvic structures (Fig. 4). This is carried out with a combination of approaches such as: general as well as targeted relaxation strategies, breathing control, muscle inhibitory techniques, enhancing passive/relaxed spinal postures, pacing strategies, hydrotherapy, cessation of stabilisation exercise training, and a focus on cardiovascular exercise. Anecdotally this approach appears very effective although clinical studies are required to validate this.

5.2.3. Psychosocial influences on peripherally mediated pelvic girdle pain

It is known that chronic pain and PGP disorders are commonly associated with not only physical but also psychosocial and cognitive impairments (Main and Watson, 1999; Bastiaenen et al., 2004, 2006; Linton, 2000, 2005) (Fig. 1). Even in the presence of a dominant peripheral nociceptive drive to PGP (such as described above), cognitive and psychosocial factors are invariably linked to these disorders influencing pain amplification and disability levels to varying degrees. This highlights the need for a biopsychosocial (behavioural) approach to understanding and managing chronic PGP disorders even when they are peripherally mediated in nature.

Psychosocial factors have the potential to both 'up' regulate or 'down' regulate pain. For example, a classification of 'reduced force closure' may be associated with cognitive impairments such as faulty beliefs, elevated anxiety levels and passive coping strategies that amplifies pain via the central nervous system and promotes high levels of disability associated with the pain disorder. In this case the intervention must address the cognitive impairments associated with the disorder within the motor learning intervention such as by promoting accurate beliefs, relaxation techniques and active coping strategies. On the other hand, if the same 'reduced force closure' classification is associated with positive beliefs, active coping strategies and limited functional impairments, then the primary focus can be placed more on the physical impairments of the disorder to establish pain control.

Similarly a classification of 'excessive force closure' may be associated with underlying stress and anxiety. In this case dealing with these cognitive factors with relaxation, breathing strategies, pacing and cardiovascular exercise is a critical adjunct to the motor learning management of these disorders. Where the psychosocial/cognitive components of the disorders are resistant to change, complementary psychological and/or medical intervention may be essential.

5.3. Central nervous system driven pelvic girdle pain disorders

The mechanisms of central nervous system sensitisation and/or glial cell activation and their involvement in the maintenance of chronic pain states are well known (Woolf, 2004; Hansson, 2006), and may persist even once a peripheral nociceptive drive is removed or has resolved. In this way chronic PGP can be potentially mediated largely or entirely via the central nervous system. In these disorders, the pain may have initially presented as a peripherally driven disorder, but once chronic, the pain does not have a presentation consistent with a peripheral pain source. These pain disorders are commonly associated with widespread, severe, and constant pain that is non-mechanical in nature. They lack a specific detectable peripheral nociceptive drive or pathological basis and are commonly associated with widespread allodynia. These disorders are associated with high levels of physical impairment and social impact, and may be associated with widespread and inconsistent motor control disturbances and abnormal pain behaviours that are secondary to the pain state and do not clearly drive the pain disorder. These disorders are often associated with dominant psychosocial factors (somatisation, catastrophising, pathological fear and/or elevated anxiety, depression, as well as significant social factors such as past history of sexual abuse etc).

Although these disorders appear to represent a small sub-group of chronic PGP disorders, they are highly disabling and resistant to physical interventions. Management of these disorders must be multidisciplinary involving medical and psychological management as a primary approach. Functional rehabilitation should aim to enhance normal general body function and address abnormal pain behaviours without a focus on pain. Passive treatments and rehabilitation that focuses on specific muscle control strategies may simply act to reinforce abnormal pain behaviours and hyper-vigilance in these patients.

5.4. Genetics and pelvic girdle pain

The role that genetics play with non-specific PGP disorders is largely unknown although its potential must be recognised. Subjects with PGP are more likely to have a mother or sister who also has PGP (Mogren and Pohjanen, 2005; Larsen et al., 1999) which may implicate a genetic link although social influences may also mediate this effect. A genetic predisposition in PGP patients related to changes in action of relaxin is proposed as one mechanism of genetic influence on PGP (MacLennan and MacLennan, 1997). Clearly further research into genetic influences is required.

6. Summary

This paper provides a broad clinical classification model for the management of chronic PGP disorders. It is a flexible, mechanism-based approach within a multi-factorial biopsychosocial framework. The classification model directs appropriate management based on the underlying mechanism/s that drives the pain. Although there is growing support for the validity of this approach, further research is required into this area.

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