KNOWLEDGE – WHAT EVERY PRACTITIONER NEEDS TO KNOW FOR CLINICAL PRACTICE

All health practitioners spend a considerable number of years gaining the knowledge necessary for their chosen career. What types of knowledge are there and what do practitioners need on a daily basis? Knowledge can be categorized as:

1. propositional, theoretical or scientific knowledge (Higgs and Titchen 1995), also known as declarative knowledge (Jensen et al 2007) and

2. non-propositional or professional craft knowledge (knowing how to do something) (Higgs and Titchen 1995) or procedural (Jensen et al 2007). Non-propositional knowledge also includes personal knowledge or knowing oneself as a person and in relationship with others.

Propositional, or declarative, knowledge refers to the content knowledge that one’s profession is based on and includes factual information derived from formal research trials (Chs 4, 5). In addition, this category includes theoretical knowledge developed from existing empirical protocols and principles derived from dialogue with professionals in the same discipline and logic (Higgs 2004).

Non-propositional, or procedural, knowledge pertains to knowing how to do things pertaining to one’s profession (craft and personal knowledge), such as how to mobilize a joint, release a hypertonic muscle, rewire a neural network, train a movement pattern and/or motivate an individual to change. This knowledge is gained through reflection on both professional and personal experiences (what worked, what did not work and how could it have been ‘done’ or handled differently to achieve a different outcome). Historically, non-propositional knowledge formed the basis for both medicine and physiotherapy. All therapy is influenced by a practitioner’s perspective, their personal knowledge, values and beliefs. This factor contributes to the outcome of an intervention and is often not considered in clinical trials studying the efficacy of a particular treatment (i.e. a trial that aims to identify if manipulation or exercise is more effective for the treatment of low back pain).

Most practitioners continue to take post-graduate courses or attend professional conferences to improve their knowledge pertaining to clinical theory and research (propositional) as well as their technical skills (non-propositional or craft); however, Rivett & Jones (2004) note that there is a tendency in both courses and conferences to neglect an essential component of daily clinical practice - clinical reasoning. How should the practitioner integrate into clinical practice the newly learned
scientific and theoretical knowledge? Who is it appropriate for and when is the new skill appropriate to use? Clinical practice is, and always will be, a blend of science and ‘art’ with a healthy dose of logic and reasoning. Clinical expertise comes from reasoning, reflection, skill acquisition and the continual life-long pursuit of knowledge (propositional (declarative) and non-propositional (procedural and personal)) (Jensen et al 2007). This takes time, discipline and often mentorship and professional affiliation both with individuals and groups.

Recently, for best practice, there is increasing pressure for practitioners to become evidence-based when making all clinical decisions. However, it appears that this term, evidence-based practice, means different things to different people. What is evidence-based practice and what is its history?

EVIDENCE-BASED PRACTICE – WHERE DID IT COME FROM? WHERE IS IT GOING?

In 1989, when the first edition of this text was published, the term evidence-based practice had yet to be coined. Gaining access to anything published (clinical opinion or scientific research) meant a trip to the university library, scrolling through the index medicus looking for any relevant article and then heading to the stacks where the journal was filed hoping it was there. The next step was to wait in a long line for the photocopier with a fistful of nickels in hand. In 1989, the internet was used only by the Department of Defense; it was released for public use in 1992.
In the 1970’s and 1980’s, peer-appointed leaders in physiotherapy taught a variety of clinical theories, protocols and techniques largely based on their experience and expertise. Therapists seeking more knowledge for their clinical practice attended their short-term courses and often became advocates for their models. At that time, it was common to be asked what kind of therapist you were and which model you followed (i.e. Maitland, Cyriax, Kaltenborn or McKenzie). Fortunately, the founders of the Canadian Academy of Manipulative Therapy (Cliff Fowler, David Lamb and John Oldham) decided against embracing just one of these approaches and against personalizing an approach for themselves and subsequently developed an integrated curriculum that provided information on all models. Canadian manual therapists have been exposed to an integrated approach since the inception of postgraduate training in orthopaedic manual therapy in 1975 and have consistently used reasoning and critical thinking to understand both impairments and the mechanisms driving the patient’s pain experience.

The term ‘evidence-based’ was first used in 1990 by David Eddy and ‘evidence-based medicine’ in 1992 by Guyatt et al. The methodologies used to determine ‘best evidence’ were largely established by the Canadian McMaster University research group led by David Sackett and Gordon Guyatt. Professor Archie Cochrane, a Scottish epidemiologist, has been credited with increasing the acceptance of the principles behind evidence-based practice (Cochrane 1972). Cochrane’s work was honoured through the naming of centers of evidence-based medical research, Cochrane Centers, and an international organization, the Cochrane Collaboration. Since the early 1990’s there has been an explosion of research evidence and accessibility to this evidence has been facilitated for those involved in research or formal study through easy internet access to full text articles in indexed journals. Unfortunately, access to full-texted articles is still limited, or expensive, for clinicians not affiliated with research centers or universities.

Evidence-based medicine categorizes and ranks the different types of clinical evidence. The terms ‘levels of evidence’ or ‘strength of evidence’ refer to the protocols for ranking the evidence based on the strength of the study to be free from various biases. The highest level of evidence for therapeutic interventions is a systematic review, or meta-analysis, including only randomized, double-blind, placebo-controlled trials that involve a homogenous patient population and condition. Expert opinion has little value as evidence and is ranked the lowest due to the placebo effect, the biases inherent in both the observation, and reporting of the cases and difficulties in discerning who is really an expert.

Evidence-based practice (EBP) embraces all disciplines of health care (not just medicine) and has become synonymous with best practice, but what does the term really mean? To some, it appears that EBP means that a clinician can only use assessment tests and treatment techniques/protocols that have been validated through the scientific process with high ranking studies as valued by the ‘levels
of evidence’. This is difficult to adhere to for many reasons, one being that there is not enough evidence at this time. Indeed, could there ever be enough scientific evidence for every situation met in clinical practice? Sackett, Straus, Richardson, Rosenberg & Haynes (Sackett et al 2000) define evidence-based practice as ‘the integration of best research evidence, with clinical expertise and patient values.’ They note that,

External clinical evidence can inform, but can never replace individual clinical expertise, and it is this expertise that decides whether the external evidence applies to the patient at all, and if so, how it should be integrated into a clinical decision.

The Three Components of Evidence-Based Practice
Sackett, Straus, Richardson, Rosenberg & Haynes (2000)

Clinical expertise, as noted above, is comprised of both propositional (declarative) and non-propositional (procedural, craft and personal) knowledge; in other words, knowing what, and how, to do the right thing at the right time (clinical reasoning and skill). The type of knowledge gained from scientific studies contributes to building only one kind of knowledge. In evidence-based practice according to Sackett et al’s definition, clinical expertise plays an equal role alongside the research evidence. A key goal of the second section of this text is to facilitate the development of clinical expertise by exploring multiple types of knowledge. A third component of evidence-based practice is the patient’s values and goals, which come from the person for whom all of the research and expertise is intended to help.

Recently, the term ‘evidence-informed’ has surfaced, the intent being to suggest that since there is not enough research evidence for every situation met in clinical practice, the clinician should be
informed of what is known and make their clinical decisions accordingly. However, if we can agree with Sackett et al’s definition of EBP, there is no need to modify the term since clinical expertise (reasoning and skill) is considered part of the definition of best practice.

UNDERSTANDING PAIN – WHAT DO WE NEED TO KNOW?

Understanding the neurophysiology of pain mechanisms is essential knowledge for treating patients with lumbopelvic-hip pain. Since the proposal of the gate control theory of pain by Melzack and Wall in 1965, significant advances in pain research and therapy have occurred. It is not our intent to provide an in-depth coverage of this topic here, but instead to highlight key features and establish a common language to be used throughout this book.

What causes pain? Searching for the pain generator

The search for ‘the pain generator’ in peripheral tissues began when Descartes, in the 17th century, proposed that specific pathways existed from the peripheral tissues to the passive brain to transmit information notifying the brain of tissue injury. The premise that injury of the tissues (ligaments, connective tissue, bones, nerves, organs, etc.) is the cause of pain is the basis of the pathoanatomical or biomedical model of pain, and has prevailed in the assessment and treatment of pain until quite recently. This model has led to research and increased understanding about nociception, including the stimuli that can cause it (mechanical, thermal, and chemical), which peripheral tissues can be painful and the pain patterns they generate. Clinicians believed that if the tissues could heal or be fixed (by whatever means, including by anesthetic injection, anti-inflammatory medication, or removal of the offending tissue), then the nociception would stop, the pain would go away, and the patient would recover function.

However, it is now well recognized that the pathoanatomical model is limited in several ways. Lumbopelvic pain commonly exists in the absence of any findings on diagnostic tests (x-ray, CT scan, blood tests, nerve conduction tests, etc.), and damaged tissues can be identified in people who experience no pain (Nachemson 1999, Waddell 2004). Tissues heal and yet the pain experience persists. Furthermore, a focus on only treating ‘the painful tissue’ neglects to consider that other systems or structures, which may be dysfunctional but pain free, could be the underlying cause of excessive mechanical stresses on the painful structures, or the cause of decreased blood flow or nutritional supply. In order to resolve the pain, the pain free but impaired structures need to be treated for long term resolution. Identification of what tissue hurts does not provide insight as to why it hurts.
Finally, significant developments in neuroscience have changed our understanding of what pain is, and have required us to reframe and change our thinking.

We now understand that at any time in one patient there are many ‘pain generators’ that do not exist solely in the peripheral tissues. Rather than looking for one source of pain, we need to consider that multiple mechanisms are at play in the experience of pain in all our patients. These mechanisms can be broadly separated into peripherally mediated (nociception and peripheral neurogenic pain) or centrally mediated (related to processing in the central nervous system (CNS)) (Butler 2000), and will be discussed in more detail later in this section.

Classifying pain – timelines & mechanism of injury

Patients are commonly classified according to the timeline or duration of their pain experience, and the cause or mechanism of their injury. In general, problems are considered to be acute if they are within the first 6 weeks to 3 months (depending on the type of tissue injured) after an initiating incident (Brukner & Khan 2002, Magee et al 2007). Tissue injury results in a known sequence of events aimed at protecting and repairing the damaged structures. These stages of tissue healing occur in three overlapping stages that have been given multiple names but refer to the same processes:

1) acute inflammatory stage,
2) subacute or proliferation stage,
3) chronic or maturation and remodeling stage.

The term chronic is often used to indicate the persistence of pain beyond the normal timeline for tissue healing (Bonica 1953, Merskey & Bogduk 1994), as opposed to a stage of the tissue healing process. In the Classification of Chronic Pain (Merskey & Bogduk 1994) published by the International
Association for the Study of Pain, it is noted that the normal time of healing ‘may be less than one month, or more often, more than six months. With nonmalignant pain, three months is the most convenient point of division between acute and chronic pain, but for research purposes six months will often be preferred.’ Chronic pain is also further outlined as ‘a persistent pain that is not amenable, as a rule to treatments based upon specific remedies, or to the routine methods of pain control such as non-narcotic analgesics (Merskey & Bogduk 1994).’

More recently, the term persistent low back pain has emerged in the literature, to indicate pain that continues past the expected time frame for tissue healing. Others are suggesting that acute episodes of low back pain would be better termed recurrent episodes in a chronic problem as the underlying mechanisms contributing to recurrent low back pain are likely to be different from a first time traumatic episode of low back pain, and recurrence of pain after an acute episode is a common problem (Pengel et al 2003).

Acute pain, especially when related to a specific initiating incident, is commonly perceived as being relatively straightforward in terms of what pain mechanisms are at play. These are generally accepted to be types of peripherally mediated pain (nociceptive or peripheral neurogenic) related to tissue damage and the resultant inflammatory processes are aimed at restoring homeostasis in the body. However, is any pain experience truly simple? Consider the following report:

A builder aged 29 came to the accident and emergency department having jumped down on a 15 cm nail. As the smallest movement of the nail was painful he was sedated with fentanyl and midazolam. The nail was then pulled out from below. When his boot was removed a miraculous cure appeared to have taken place. Despite entering proximal to the steel toecap the nail had penetrated between the toes; the foot was entirely uninjured (BMJ 1995).

The initial logical hypothesis in this case was that acute trauma to the foot was causing severe nociceptive input from the damaged tissues. However, as physical examination revealed completely intact tissues, this cannot explain the patient’s pain experience. Clearly other pain mechanisms were at play, despite the timeline (acute onset) and mode of onset (traumatic) of the pain.

Empirical evidence now exists to explain these kinds of stories. A consistent factor that has emerged from the pain sciences is that the meaning of the pain experience, and especially the threat value of the experience, is significant. In other words, does the pain signify something harmful or not? While some may continue to function and keep going in spite of pain, others are completely debilitated by the mere thought of the sensation. There is increasing evidence to support that an individual’s experience of pain is significantly influenced by the way they think and feel about the situation as a whole, regardless of the severity of tissue injury. The story above illustrates these influences; his pain experience is an example of 100% centrally mediated pain, driven by his beliefs
(cognitive dimension) and emotional state (affective dimension) related to the event (having a nail driven through his boot). It is clear that we cannot separate the tissues from the person to which they belong; we are integrated beings and our experience of our body (whether positive or negative) is the result of complex interactions and processes occurring in the brain.

Thus, although the mechanism of onset and time frames related to the pain experience are important to know, we must take care that this information does not lead us to assume that certain timelines necessitate certain pain mechanisms. Acute pain can be largely driven by central mechanisms. Persistent pain can also be largely driven by peripheral mechanisms. That is, persistent or chronic pain states may have central components, but these are not necessarily the dominant mechanism for every patient simply because the pain experience has persisted for a long period of time. While evidence supports that ‘the relationship between pain and the state of the tissues becomes less predictable as pain persists (Moseley 2007b),’ we need to remember that the pain experience is uniquely individual. Regardless of whether the pain is a newly occurring event or a persistent experience, it is a multidimensional experience, and thus any person presenting with pain should be evaluated with a framework in mind that allows for the consideration of all these factors. As Butler (2000 p. 53) notes,

Overlap of mechanisms is the key feature because the boundaries are often fuzzy. There will be differing contributions of mechanisms to the injury state over time, person and injury.

**Classification by pain mechanisms**

So what are the different biological mechanisms that drive the pain experience? Pain mechanisms can be further categorized (Butler 2000, Gifford 1998) as they relate to:

a. input into the nervous system,

b. processing in the nervous system, and

c. output from the nervous and other systems.

The brain receives continuous information from the body and the environment (input mechanisms or all sensory pathways) that is assessed and interpreted (processing at both conscious and unconscious levels) prior to producing a response (output mechanisms). Some of this incoming information is nociceptive. There are many factors that determine an individual’s behaviour and pain experience (physical, cognitive, emotional) in response to nociceptive input, including:

- contextual factors of the immediate circumstance (i.e how dangerous is this sensation in the light of environmental and internal factors?) as well as
past experiences and personal knowledge that collectively contribute to the individual’s beliefs, attitudes, emotions and physical responses.

Input mechanisms as they pertain to pain include all the sensory information reaching the central nervous system (CNS) from the body internally and externally. This includes nociceptive pain from tissues including bones, ligaments, tendons, muscles, connective tissue, viscera, etc. (Butler 2000, Gifford 1998, Wright 2002) and peripheral neurogenic pain from neural tissue outside of the CNS. Processing occurs in the dorsal root ganglion and in the CNS. In the brain, an individual’s thoughts and feelings (cognitions + emotions = perception) are integrated and can influence the output mechanisms, which include:

1. somatic or motor (altered posture, altered motor control),
2. autonomic (increased sympathetic response for ‘fight or flight’),
3. neuroendocrine (increased stress, heightened emotions, hormonal changes) and
4. neuroimmune.

Thinking within the context of stress biology creates a broader framework for understanding pain. Gifford (1998), in proposing the mature organism model, notes that

…the sensation of pain is seen as a perceptual component of the stress response whose prime adaptive purpose is to alter our behaviour in order to enhance the processes of recovery and chances of survival. Stress biology and the stress response broadly considers the systems and responses concerned with maintaining homeostasis (Gifford 1998).
It has been proposed that continued activation of the stress-regulation systems and excessive or prolonged cortisol output has a destructive effect on peripheral tissues such as muscle, bone and nerve tissue, thereby perpetuating a vicious cycle of stress, pain, and tissue injury (Melzack 2005).

In his *Neuromatrix Theory of Pain* (2001, 2005), Ronald Melzack highlights the need to assess and treat the whole person, not just the painful parts.

*The body is felt as a unity, with different qualities at different times... [Together all outputs] produce a continuous message that represents the whole body [which he also describes] as a flow of awareness* (Melzack 2001, 2005).

Melzack’s model has four components (2001, 2005):

1) the body-self neuromatrix – an anatomical substrate in the brain of the body-self,  
2) cyclical processing and synthesis of nerve impulses which produces a neurosignature,  
3) the flow of neurosignatures is projected back to areas of the brain, the sentient neural hub, which converts them into the flow of awareness, and  
4) activation of an action neuromatrix occurs to provide the pattern of movements to bring about the desired goal.

The neuromatrix, distributed throughout many areas of the brain, comprises a widespread network of neurons which generates patterns, processes information that flows through it, and ultimately produces the pattern that is felt as a whole body. The stream of neurosignature output with constantly varying patterns riding on the main signature pattern produces the feelings of the whole body with constantly changing qualities....The final, integrated neurosignature pattern for the body-self ultimately produces awareness and action (Melzack 2005).
The figure above is a modification of Melzack's representation of the body-self neuromatrix to illustrate the sensorial, cognitive, and emotional dimensions of pain. Perhaps the best summary for this section highlighting the broad view we need to take when considering pain comes from this leader in the study of pain himself, Ronald Melzack (2001).

*We have traveled a long way from the psychosocial concept that seeks a simple one-to-one relationship between injury and pain. We now have a theoretical framework in which a genetically determined template for the body-self is modulated by the powerful stress system and the cognitive functions of the brain, in addition to the traditional sensory inputs. The neuromatrix theory of pain – which places genetic contributions and the neural-hormonal mechanisms of stress on a level of equal importance with the neural mechanisms of sensory transmission – has important implications for research and therapy. The expansion of the field of pain to include endocrinology and immunology may lead to insights and new research strategies that will reveal the underlying mechanisms of chronic pain and give rise to new therapies to relieve the tragedy of unrelenting suffering.*

It is very clear that as clinicians we need to be aware of all the possible mechanisms that can create pain and to challenge ourselves to have an open mind as we seek to understand each individual's unique pain experience in order to determine which mechanisms are primary for them in all stages of their rehabilitation process.

**CLASSIFICATION AND CLINICAL PREDICTION RULES – ARE WE SEARCHING FOR THE HOLY GRAIL?**

Given the multidimensional nature of pain, it is not surprising that using pain presentation (location, duration, onset) as the sole means to classify patients and determine best treatment has been ineffective. Fritz and colleagues (2007) report that despite over 1000 randomized clinical trials investigating the effectiveness of interventions for the management of low back pain, ‘the evidence remains contradictory and inconclusive (Fritz et al 2007).’ One key reason believed to contribute to this state of the evidence is the lack of classification of low back pain patients into subgroups, not only for studying treatment efficacy, but also for determining etiologic and prognostic factors (Gombatto et al 2007, Leboeuf-Yde et al 1997, Riddle 1998). Sahrmann in the late 1980’s noted,

*As we all know, general diagnoses such as low back pain or hip pain do not often relate to the cause or to the underlying nature of the condition (Sahrmann 1988).*

As clinicians have long recognized, it is now widely accepted that patients with low back pain, pelvic girdle pain, and hip pain do not form homogeneous populations, but consist of multiple
subgroups with different combinations of underlying impairments (physical and psychosocial), and these subgroups require different treatment approaches for best outcomes. Furthermore, given that multiple factors contribute to lumbopelvic or hip pain, it is also unrealistic to expect that one single type of treatment modality will resolve a patient’s presenting pain and functional limitations. Thus, the pursuit of valid ways to identify subgroups of patients with low back and pelvic girdle pain has become an increasingly prominent theme in the literature over the last three decades.

The classification for lumbopelvic pain has evolved since the pathoanatomically-based classification of MacNab (1977) with a variety of patient characteristics proposed for use in creating homogeneous subgroups (Bernard & Kirkaldy-Willis 1987, Coste et al 1992, Delitto et al 1995, Fritz et al 2007, Kirkaldy-Willis 1983, McKenzie 1981, O’Sullivan 2005, O’Sullivan & Beales 2007, Reeves et al 2005, Sahrmann 2001). O’Sullivan (2005) noted that a limitation of many classification systems is that only a single dimension (pathoanatomical, psychosocial, neurophysiological, motor control, signs and symptoms, etc.) is often used to create subgroups. Classification systems will be most useful in clinical practice if variables across multiple domains are used to create subgroups.

Features that have been incorporated into different systems include (note this is not intended to be an exhaustive list):

- presence or absence of identifiable underlying pathology (pathoanatomical, peripheral pain generator models),
- pain presentation (central, unilateral, with or without radiation of symptoms to the lower extremity) (signs and symptoms models),
- underlying pain mechanisms/neurophysiology,
- response of pain to movement (centralization or peripheralization) (signs and symptoms models) (movement impairment models),
- physical impairments such as loss or increase of mobility, altered motor control, altered posture/spinal alignment, and the relationship of symptom provocation to these impairments (motor control models, signs and symptoms models, movement impairment models),
- response to specific treatments (manipulation, stabilization exercises, specific exercises, traction), and
- psychosocial and cognitive features such as fear avoidance, coping strategies, and beliefs (biopsychosocial models).

In recent years, the development of clinical prediction rules (CPRs) has emerged as another method to classify patients. CPRs are derived statistically with the aim of identifying the combinations of clinical examination findings that can predict a condition or outcome. Thus, they are proposed to be a useful tool to assist in clinical decision making by improving the accuracy of diagnosis, prognosis, or
Clinical practice rules provide probabilities of a given diagnosis or prognosis but do not necessarily recommend decisions. Clinical prediction rules can be of great value to assist clinical decision-making but should not be used indiscriminately. They are not a replacement for clinical judgment and should complement rather than supplant clinical opinion and intuition (Beattie and Nelson 2006).

Research on specific subgroups and development of classification systems will definitely provide a much better understanding of the specific impairments, mechanisms, and psychosocial features that characterize subgroups and their response to treatment. As Melzack wrote about the evolution of the gate control theory of pain,

As historians of science have pointed out, good theories are instrumental in producing facts that eventually require a new theory to incorporate them (Melzak 2001).

However, it is important to recognize that there are limitations on how information gained from classification systems, CPRs, clinical trials, and indeed the findings of any scientific study, can be translated and applied into the reality of clinical practice. Firstly, statistical averages tell us about the average response of the group defined by the characteristics used in design of the study. Individual responses may be to a greater or lesser degree than the average, or even in the opposite direction of the reported response. Indeed, practicing clinicians are well aware of the many patients they have seen who do not fit the data from clinical trials or other studies. These clinical cases provide valuable insight and can generate questions for further research. Secondly, while the data provides relatively unbiased information, the interpretation and conclusions made from the data, and published alongside the data, are subject to bias just as much as clinical opinion is subject to bias. It is also important to recognize that a lack of data or science does not invalidate a technique or approach, nor does it mean that approaches that have been studied are necessarily superior. In clinical practice, application of any classification system/CPR requires care to ensure that it does not create a rigid, narrow mindset. Placing the patient ‘in a box’ could prevent the clinician from considering other
options for treatment that may be greatly beneficial. Neglecting to provide these other options could then result in sub-optimal outcomes.

Consider the one domain of underlying pain mechanism as a way to create subgroups. Butler (2000) notes that,

*The word “division” can be instant trouble because these mechanisms all occur in a continuum. All pain states probably involve all mechanisms, however in some, a dominance of one mechanism may become obvious. Pain mechanisms are not diseases or specific injuries. They simply represent a process or biological state.*

In their classification of pelvic girdle pain disorders, O’Sullivan and Beales (2007) categorize non-specific pelvic pain disorders into two groups, one that has centrally mediated pain, and one that has peripherally mediated pain. Although the group of centrally mediated pain is further classified into those with non-dominant psychosocial factors and those with dominant psychosocial factors, the treatment protocol for the subgroup of centrally mediated pelvic girdle pain is medical management (central nervous system modulation), psychological (cognitive behavioural therapy), and functional capacity rehabilitation. Specific interventions directed at identified physical impairments in the periphery are not recommended, and yet it is highly unlikely that many patients will have 100% centrally mediated pain. In the authors’ experience, even in patients with a strong contributor of central sensitization to their pain experience, careful assessment often reveals specific meaningful tasks that relate to a consistent reproduction of symptoms (see case study Julie K - chapter 9). It is reasonable to suggest that even if peripheral mechanisms only contribute 20% to the complete picture, addressing that 20% in addition to the other approaches will provide the greatest chance for the best outcome. Furthermore, it is likely that by addressing the physical impairments, psychosocial variables will also be impacted, further advancing the goals of treating drivers of central sensitization.

It is also crucial to recognize that our patients change as a result of their changing life circumstances and our interactions with them (both physical and personal). Thus, during the course of treatment continual re-evaluation is necessary to adapt the treatment program accordingly. Sticking to a rigid plan based on an initial placement into a subgroup may result in the provision of sub-optimal care.

Finally, in our quest for better classification schemes and science to support and test our clinical approaches, it is important to remember that at the end of the day no matter how detailed and well defined our classification schemes, the person presenting to the clinician is a unique individual with unique life experiences. There will never be one recipe for treatment that is the best fit for all patients. Furthermore, patient values and beliefs are central to the treatment process, and if they do not want to receive what is considered ‘best practice’ from the current evidence, we cannot force it on them. Given
the same impairment in the tissues, no two individuals will have exactly the same perception and presentation (experience and behaviour) because ‘how they manifest their pain or illness is shaped in part by who they are (Jones & Rivett 2004).’ A reminder, the highest level of evidence for therapeutic interventions is a systematic review, or meta-analysis, of only randomized, double-blind, placebo-controlled trials which involve a homogenous patient population and condition. Is this possible in the light of what is known about pain? Do homogeneous populations really exist in clinical practice?

Science can provide us with an abundance of knowledge to challenge, refine, reshape, and validate our clinical practice, but it cannot provide all of the information needed in any individual patient encounter; it does not paint the whole picture of the patient. In order to effectively treat patients, therapists need to have well-organized knowledge including propositional (knowledge ratified by research trials), non-propositional (professional craft or ‘knowing how’ knowledge) and personal (knowledge gained from personal experiences) (Jones & Rivett 2004).

Understanding and successfully managing patient’s problems requires a rich organization of all three types of knowledge. Propositional knowledge provides us with theory and levels of substantiation by which the patient’s clinical presentation can be considered against research-validated theory and practice. Non-propositional professional craft knowledge allows us the means to use that theory in the clinic while providing additional, often cutting-edge (albeit with unproven generality) clinically derived evidence. Personal knowledge allows a deeper understanding of the clinical problem to be gained within the context of the patient’s particular situation and enabling us to practice in a holistic and caring way (Jones & Rivett 2004).

Personal and craft knowledge cannot be learned from RCTs, mechanistic studies, basic physiology studies, or clinical prediction rules. Ultimately, it is the development of clinical expertise that creates optimal patient care. According to Ericsson & Smith (1991) expertise has been defined as ‘having the ability to do the right thing at the right time.’

Clinical expertise has two components: skill acquisition (do the right thing) and clinical reasoning (at the right time). Clinical reasoning skills facilitate the organization and integration of knowledge gained both in and out of the clinic, and the wise application of that knowledge for each individual patient. The development of clinical expertise is discussed further in chapter 9, and is the focus of the second part of this book.

Different classification systems provide us with a variety of perspectives to grow our knowledge base. However, hoping to find ‘the best classification system’ to apply in every situation in clinical practice is like searching for the Holy Grail – it cannot be found. We are unique people trying to help other unique people. We need to re-evaluate how we value the ‘levels of evidence’ and the role of science in directing clinical practice, and develop a more balanced view that values the insight that is
uniquely derived from clinical practice. The clinical ‘lab’ plays a key role in new knowledge generation through the development of innovative techniques for assessment and treatment, which can then be tested by science. Knowledge gained from clinical experience is not more important than science, but it certainly is no less important. Overall, maintaining an open mind and broad perspective will assist both scientists and clinicians to discover how best to work together and learn from each other in the common goal of providing best care for our patients.

IT’S ABOUT MORE THAN PAIN – INTEGRATED SYSTEMS FOR OPTIMAL HEALTH

It has been long recognized that simply relieving a patient’s pain does not necessarily result in a full return to all functional activities. Furthermore, there are subgroups of patients, such as high-level athletes, whose functional goals and measures (race time, power delivery in a stroke for example) are just as, if not more, meaningful to them than the relief of pain. Indeed, there is an increasing market in helping people without pain to optimize performance as well as prevent injury by facilitating strategies for better posture and movement. Pain is not a problem for these people, but an inability to meet their functional goals is. Non-painful impairments are also recognized as a potential contributor to the development of pain, both in sites distal to the impaired area and in the area itself. Furthermore, if we take the broader view that ‘pain is an opinion on the organism’s state of health rather than a mere reflexive response to injury (Ramachandran in Doidge 2007),’ we need to alter our focus and consider what it means to be ‘in health’ and not only what it means to be ‘in pain’. The World Health Assembly has defined health as ‘a state of complete physical, mental, and social well-being and not merely the absence of disease or infirmity (WHO Constitution).’ Speaking at the 1985 annual conference of the American Medical Association (Seattle, USA), Dr. Paul Brenner defined health even more broadly as ‘the full acceptance and appreciation of life.’ Restoring health is about more than removing disease; creating optimal strategies for function and performance is about more than removing pain.

What it means to be ‘in health’ is individually defined. Therefore, changing our focus from removing pain to restoring optimal health and optimal strategies for function and performance is intrinsically linked to the patient’s values and goals. Our role as clinicians is to best facilitate and empower patients on their journey to achieve their personal optimal health and function. To do this effectively, we need to not only understand their pain, but need to also understand them as a person. Jones and Rivett (2004) refer to this as ‘understanding both the problem and the person.’

To understand and manage patients and their problems successfully, manual therapists must consider not only the physical diagnostic possibilities (including the structures involved and the associated pathobiology) but also the full range of factors that can contribute to a person’s health, particularly the
effects these problems may have on patients’ lives, and the understanding patients (and significant others) have of these problems and their management (Jones & Rivett 2004).

This paradigm requires that clinicians broaden their perspectives and skill sets, and also opens up a wider range of potential and possibility for effecting change.

The Integrated Model of Function was developed from anatomical and biomechanical studies of the pelvis, as well as from the clinical experience of treating patients with lumbopelvic pain (Lee 2004, Lee & Vleeming 1998, 2004, 2007). From its inception, the Integrated Model of Function focused on the evaluation of the function of the pelvis, and how the pelvis effectively transfers loads across tasks with varying characteristics. The model addresses why the pelvis is painful by identifying the underlying impairments in four specific components: form closure, force closure, motor control and emotions. This is in opposition to pathoanatomical models that seek to only identify pain-generating structures.

This model has continued to evolve with the publication of anatomical, biomechanical and neurophysiological research as well as the clinical expertise gained through collaborative efforts worldwide and remains a useful framework to understand the pelvis in function and in dysfunction.

The Integrated Systems Model for Disability and Pain evolved from working with the Integrated Model of Function and was first introduced in 2007 as the System-Based Classification for Failed Load Transfer (Lee & Lee 2007, Lee et al 2008, Lee & Lee 2008a,b). We have since recognized that using the word ‘classification’ is limiting for this model because its primary purpose is not to place patients into homogeneous subgroups. In contrast, it is a framework to understand and interpret the unique picture of each individual patient in the clinical context to facilitate decision-making and treatment planning. The model provides a context to organize all the different types of knowledge needed (scientific, theoretical, professional craft, procedural, and personal) and provides for the development and testing of multiple hypotheses as the multidimensional picture of the patient emerges. A multimodal treatment plan can then be designed based on the complete picture of the person and their presenting problem(s).

The Integrated Systems Model for Disability and Pain (LJ Lee, Diane Lee) allows clinicians to characterize all the components that contribute to what Melzack terms the ‘message that represents the whole body’ as a ‘flow of awareness’ (Melzack 2005).’ It is an integrated, evidence-based model that considers disability and pain as defined and directed by the patient’s values and goals. The model relates impairments found in systems, underlying pain mechanisms, and the impact of these impairments on their current whole body strategies for function and performance. Thus the model analyzes the patient’s current whole body strategies, determines the underlying reasons for those strategies, and relates these to current knowledge about the necessary state required in all systems to provide optimal strategies for function and performance, and ultimately, for health. As a systems-based model, it has
inherent flexibility to evaluate and integrate new evidence from research and innovative clinical approaches as they emerge. As a patient-centered model, it can continually adapt to changing goals and values of the patient. As the model applies to the whole person, rather than to a specific type of pain presentation or body region, it can be used across pain and disease populations and is not only applied to patients with lumbopelvic or pelvic girdle pain. However, for the context of this book, specific examples for patients with problems in the low back, pelvic girdle and hip regions will be used to illustrate the model. In the context of the LPH complex, the Integrated Model of Function fits within, and is encompassed by, The Integrated Systems Model for Disability and Pain. The Integrated Model of Function provides a way to subgroup patients with failed load transfer (FLT) in the LPH complex; those with a primary form closure, force closure, motor control or emotional deficit (Ch. 5). The broader Integrated Systems Model for Disability and Pain also considers how a patient could be subgrouped according to the primary system impairment of patients and also considers the role of the rest of the body and mind to the observed failed load transfer (FLT) in the LPH complex. For example, is the primary impairment causing the FLT intrinsic to the pelvic girdle itself (SIJ laxity → pelvic-driven pelvic girdle pain – see case report part 1 of Julie G – Ch. 9) or extrinsic to the pelvic girdle (foot-driven pelvic girdle pain – see case report Louise or thorax-driven pelvic girdle pain (first proposed by LJ Lee) – see case reports part 2 of Julie G and part 2 of Louise) or due to a negative cognitive/emotional state (see case report Julie K – editor to add online link). The Integrated Systems Model for Disability and Pain also considers the interaction and contribution of multiple systems (articular, myofascial, neural, visceral, hormonal, neuroendocrine etc.).

Therefore, while The Integrated Systems Model is based on the identification of the multi-system impairments that are the key drivers behind the problems facing the whole person, which could then be used to subgroup patients, the primary purpose of the model is to provide a framework for building a unique tapestry that tells the patient’s story. It also facilitates clinical reasoning ‘on the fly’ as the patient’s story unfolds and the clinician begins to understand the significant pieces of their tapestry. When used reflectively, it is our goal that The Integrated Systems Model will facilitate, foster, and promote the development of clinical expertise.

THE INTEGRATED SYSTEMS MODEL FOR DISABILITY & PAIN - A FRAMEWORK FOR UNDERSTANDING THE WHOLE PERSON AND THEIR PROBLEM

Underlying constructs of the model
Before we can describe the components or systems of The Integrated Systems Model, it is important to define its underlying constructs. These include the definitions of key terms and are as follows:
The terms ‘body’, ‘function/functioning’, ‘disability’, ‘impairment’, and ‘health condition’ are taken from the International Classification of Functioning, Disability and Health (ICF) definitions (p.189-190, 2001):

Body functions are the physiological functions of body systems, including psychological functions. ‘Body’ refers to the human organisms as a whole, and thus includes the brain. Hence, mental (or psychological) functions are subsumed under body functions.

Functioning is an umbrella term for body functions, body structures, activities and participation. It denotes the positive aspects of the interaction between an individual (with a health condition/ [perceived problem(s)]) and that individual’s contextual factors (environmental and personal factors).

Disability is an umbrella term for impairments, activity limitations and participation restrictions. It denotes the negative aspects of the interaction between an individual (with a health condition) and that individual’s contextual factors (environmental and personal factors).

Impairment is a loss or abnormality in body structure or physiological function (including mental functions).

Health condition is an umbrella term for disease, disorder, injury, or trauma. A health condition may also include other circumstances such as pregnancy, ageing, stress, congenital abnormality, or genetic predisposition.

2) In a state of optimal health, an individual will have the option to choose from a wide variety of strategies that provide for optimal function and performance during any meaningful task (movement, activity, or role in a desired context and environment). Determining whether a task is meaningful requires understanding the person and their values and goals.

3) By definition, optimum function and performance occurs in a state of health, and will be a state free from undesired pain experiences. Given the definitions of health above, optimum function and performance is individually defined, and attainable in the presence of any health condition, although it may be influenced by specific features of the health condition.
4) Pain is not the only reason that people become disabled. Disability, or the inability to do what the person wants to do, can exist without pain.

5) Optimum function and performance for any task requires the synergistic, integrated operation of multiple systems in the body. ‘Synergy’ is defined as a ‘combined or cooperative action or force’ (Webster’s New World College Dictionary) and ‘simply defined, it means that the whole is greater than the sum of its parts (Wikipedia).’ To ‘integrate’ is to ‘form, coordinate, or blend into functioning or unified whole (Merriam-Webster’s Online Dictionary).’ Synergy and integration require that each system, and thus the components of each system down to the cellular level, is functioning, and that the many complex feedback and feedforward mechanisms that control each system are working optimally. Then, the systems must work together to produce desired outputs in the body. Congruence of information received from feedback and sensory systems is also important. Not all the underlying mechanisms that produce the integrated, synergistic operation of body systems are fully understood, although science is continuing to reveal the connectedness and interdependence of body systems. Melzack’s concept of the body-self neuromatrix highlights this need for synergy and integration.

6) Impairment(s) in any one or combination of systems can give rise to undesired outputs in one or more systems. These outputs include painful states, non-optimal posture and movement (inefficient, loss of desired performance or output), loss of function, overactive and/or sustained stress response, and negative emotional states.

7) Designing and implementing the most effective treatment plan for restoring health depends on identifying the relevant impairments in the key systems that are barriers to healing and that need to be addressed in order to restore function and health. The relevance of each impairment is determined through a clinical reasoning process that uses a combination of different types of reasoning (Ch. 9). Each impairment is evaluated in the context of meaningful tasks to determine how much the impairment contributes to the non-optimal strategies for function and performance, and the pain experience. The impairments/systems/regions with high contribution values are called the key ‘driver(s)’ in this model. The term ‘pain driver’ is used to refer to the underlying cause(s) of the pain experience, which could be the pain mechanism itself or a multitude of combined impairments that collectively increase physical and psychological stress and perpetuate the pain experience by exceeding the adaptive/coping
mechanisms of specific tissues and the person as a whole. Note that since the human body is dynamic, that is a changing entity, the key drivers for disability and/or pain at different points in time can change. Furthermore, the driver(s) of disability may be different than the driver(s) of pain.

8) *The Integrated Systems Model* is applicable to disability and/or pain of any duration; that is, from acute onset to chronic, persistent, or recurrent problems.

9) Every person is unique genetically, emotionally, cognitively, culturally, and socially; the activities and roles that have meaning for them and their pain experience will be uniquely their own. In this way, the specific combination of impairments and systems that contribute to output experiences will be different for each patient. However, taken together, science and clinical expertise provide us with the necessary information to allow us to identify common patterns and parameters for normal and abnormal functioning of systems, as well as how subgroups of patients with certain common features (determined in research by inclusion and exclusion criteria of the study) respond to different treatment approaches. This information is invaluable and indispensable, and the continued pursuit of furthering our knowledge base (both propositional and non-propositional knowledge) in research and in the clinic creates a continually refined understanding of what allows us to enjoy health. However, knowledge gained from either the clinic or the research lab has limitations. Clinicians must constantly examine their emerging hypotheses for multiple types of bias. While clinical practice guidelines derived from research can be helpful and provide new insight, they may also be inappropriate and incorrect for certain patients. Therefore, caution is always necessary when developing general treatment protocols based on ‘homogeneous populations’ since homogeneous populations are an illusion and do not truly exist outside of research constructs.

10) Each person is a dynamic entity and can change from moment to moment and day to day. Science continues to find more evidence of this. Clinically, this implies that continual reassessment is essential for revising hypotheses about the drivers of the patient’s problem.
Components of the model – The Clinical Puzzle – A tool for clinical reasoning and developing clinical expertise

The Clinical Puzzle is a graphic that conceptualizes The Integrated Systems Model for Disability and Pain. It represents the person and their problem(s), and the systems that support optimal strategies for function and performance. The puzzle is used clinically and in teaching as a tool for clinical reasoning and decision-making.

The person in the middle of the puzzle

At the centre of the model is the patient, the person in the middle of the clinical puzzle. Seeking to understand the unique makeup of the person (the color, shape and content of their tapestry), without judgment, is the goal of listening to the patient’s story. It is essential that during the subjective examination the therapist create a supportive, compassionate environment that allows the patient to tell their story freely. Open ended questions, such as ‘what can I do for you today?’ or ‘please tell me your story’ create an invitation for the patient to share the things about their current experience that are most meaningful and relevant to them, along with their goals and values. This is in contrast to the therapist who has a checklist of questions to obtain answers to, and who strongly directs the subjective examination along a path that the therapist deems (in their wisdom) to be the best. This checklist format of subjective examination is more likely to miss out on essential information from the patient.

Understanding the person in the middle of the clinical puzzle also incorporates information about the sensorial, cognitive, and emotional dimensions to their experience of their problem(s). Problems may be disability and/or pain. The sensorial dimension includes the location and behaviour of the problem(s), the cognitive dimension includes their beliefs and attitudes about their current experience, and the emotional dimension encompasses both positive and negative feelings about the experience. Problems such as incontinence (stress and/or urge), symptoms of pelvic organ prolapse, difficulty with breathing, and effortful movement, are all examples of problems that the patient may not talk about if they are only asked about their pain, but that are important to identify when present. From these multiple dimensions, the therapist can glean potential barriers to, and potential facilitators of, recovery.

How the patient perceives their body and their current experience of their body constitutes the current state of their virtual body. The virtual body is made up of both conscious and unconscious
components. As the objective examination proceeds, discrepancies between the actual body and the virtual body will become evident. An example of this would be when a patient perceives that they are standing with equal weight bearing on both feet, but postural examination reveals that the centre of mass is shifted to load one extremity to a greater degree than the other.

Meaningful tasks are postures and/or activities that are determined by aggravating activities, relieving activities, activities associated with negative beliefs and emotions (e.g. movements the patient is fearful of), activities in specific environments or contexts, and the patient’s goals (e.g. what would you really like to do that you are not currently able to do due to this problem?). All characteristics of meaningful tasks, including biomechanical requirements, environmental, social, and emotional context must be considered during the objective examination in order to most accurately analyze the strategies used by the patient during the meaningful task analysis.

The centre of the puzzle (the person in the middle of the clinical puzzle) also represents their genetic makeup and systemic health status, including the nutritional, neuroendocrine, autonomic, and homeostatic/stress/immune systems. Past experiences, social background, and other psychosocial features are also a part of the centre of the puzzle.

The process of the subjective examination and understanding the patient’s story is further discussed in chapter 8 (assessment) and chapter 9 (case reports). The experienced clinician will start to link information in the patient’s story and form initial hypotheses that direct the priorities of the objective examination to follow.

**Strategies for function & performance**

The meaningful tasks identified from the patient’s story direct the tasks chosen for analysis of strategies for function and performance, and are noted in the outside ring of the puzzle. These tasks, or the relevant component movements of the task, must be assessed to determine if the patient is using an optimal or non-optimal strategy for the meaningful task. Since the strategies that people use for whole body function are a result of, and depend on, the integrated function of all systems in the body, including all the systems represented by the person in the middle of the puzzle, the ‘strategies ring’ encircles the entire puzzle. If a non-optimal strategy is observed, the objective findings characterizing how the strategy is non-optimal are written beside the task listed in the outer circle of the puzzle. Strategy analysis is further discussed in chapters 8, 9 and 12.
Articular, myofascial, neural, visceral systems

The four other pieces of the puzzle represent the systems that are assessed during the clinical examination. Specific impairments, as well as information gained from diagnostic tests (e.g. X-rays, MRI etc.) and other sources, are charted within the relevant system in the puzzle. The therapist also considers and reflects upon the relationship of these impairments to the person in the middle of the puzzle (e.g. sensorial, cognitive, and emotional dimensions of the problem(s)) and the relationship these impairments have to the non-optimal strategies for function and performance during meaningful tasks.

As the examination proceeds, the therapist evaluates whether or not the observed non-optimal strategies for function and performance for each meaningful task are appropriate or inappropriate given all the information available (beliefs about the task, state of tissue healing/integrity of tissue, characteristics of the task and the task context including load requirements, mobility requirements, level of predictability, threat value, availability of accurate proprioceptive input). Note that for some tasks, the patient may have appropriate strategies (side bent lumbar spine posture due to acute radicular pain), while for other tasks the patient may have inappropriate strategies (fear of moving in any direction in the lumbar spine due to pain that only occurs in one direction of movement). If the therapist has reason to believe that a strategy is inappropriate, determining the reasons a patient chooses a particular strategy is essential for identifying the driver(s) of the problem and planning the most effective treatment program.

Specific impairments in the articular, myofascial, neural, and visceral systems for the lumbopelvic-hip complex are listed in Tables a-d. The articular system includes the bones and joints (passive structures) in the musculoskeletal system. The myofascial system includes muscle, and its tendinous and fascial connections, as well as the multiple layers of fascia throughout the body. The neural system includes all components of the central and peripheral nervous system. It also includes the neural drive to muscles, which is reflected in the resting tone and activity or control of the muscle system. The visceral system includes all the viscera of the body.

An impairment in any piece(s) of the puzzle within the outer circle (the ‘systems’), or loss of congruence and synergy between the pieces of the puzzle, can ‘drive’ non-optimal strategies for function and performance. Conversely, non-optimal strategies for function and performance can drive or create impairments within any of the systems inside the puzzle (the person in the middle of the puzzle, the neural, myofascial, articular and visceral systems). Thus, the entire puzzle is connected,
linked and interdependent and visually represents the integrated systems required for optimal health. All clinical puzzles are unique since no two individuals have the same life experiences.

If one considers all of the possible combinations of impairments and the associated findings that can lead to disability and/or pain, the LPH complex can seem complicated. In reality, when reflective critical thinking and a thorough examination are used, the primary cause and initial treatment plan emerges. The Clinical Puzzle for The Integrated Systems Model is a useful tool for understanding the whole person and their problem(s). It allows for organization of key information gained through the examination process, comparing and contrasting this information to current propositional knowledge and personal knowledge of the clinician, and for reflection and interpretive reasoning of the findings. This facilitates the formation of hypotheses to explain the relationships between physical impairments, pain mechanisms, psychosocial features, disability, health conditions, and the patient’s values and goals. The goal of the clinical reasoning process, facilitated by the puzzle, is to determine which hypothesis provides the ‘most likely and most lovely’ explanation of the patient’s whole experience, from which an integrated multimodal treatment plan is formulated and implemented. As treatment evolves over several sessions, the focus often changes as the patient’s journey towards function and better health occurs.

SUMMARY
Together with the theoretical and scientific information presented in part one, this chapter has laid the foundation for the rest of this text. Now it is time to put The Integrated Systems Model for Disability and Pain to work and show you how we use it in clinical practice to facilitate the resolution of the patient’s unique Clinical Puzzle. By now, you should understand that we do not follow recipes or clinical guidelines; we read the research thoroughly and rely on our clinical expertise and sound reasoning skills and logic to guide our clinical practice. We believe that our job is to empower patients to know more about themselves and their state of health so that they can become aware of how their faulty posture, movement, thinking and/or lifestyle habits can drive their problem(s) and prevent them from attaining the level of function and performance they desire. Ultimately, we hope to motivate them to make the changes necessary to feel better, move better and be better; it’s up to them.
Articular

- capsular sprain or tear
- ligament sprain or tear (grades I-III)
- labral or intra-articular meniscal tear
- intervertebral disc strain/tear/herniation/prolapse
- fracture
- joint subluxation or dislocation
- periosteal contusion
- stress fracture,
- osteitis, periostitis, apophysitis
- osteochondral/ chondral fractures, minor osteochondral injury
- chondropathy (softening, fibrillation, fissuring, chondromalacia)
- synovitis
- apophysitis
- fibrosis/osteophytosis of the zygapophyseal and intervertebral joints, sacroiliac joint, hip joint

Table a The conditions associated with the articular system of the Clinical Puzzle
Myofascial

- intramuscular strain/tear (grades I-III)
- muscle contusion
- musculotendinous strain/tear
- complete or partial tendon rupture or tear
- fascial strain/tear
- tendon pathology – tendon rupture, partial tendon tears, tendinopathy (acute or chronic), paratendinopathy, pantendinopathy
- skin lacerations/abrasions/puncture wounds
- bursa – bursitis
- muscular or fascial scarring or adhesions
- loss of fascial integrity of the anterior abdominal wall including
  - altered integrity of the linea alba
  - sports hernia (tear of transversalis fascia)
  - hockey hernia (tear of the EO),
  - inguinal hernia, or
  - loss of fascial integrity of the endopelvic fascia leading to cystocele, enterocoele and / or rectocele

Table b The conditions associated with the myofascial system of the Clinical Puzzle
Neural

- peripheral nerve trunk or nerve injury (neuropraxia, neurotmesis, axonotemesis)
- central nervous system injury
- altered motor control
- absence of recruitment, inappropriate timing (early or late) of muscle recruitment,
- inappropriate amount (increased or decreased) of muscle activity (all relative to demands of task)
- hypertonicity or hypotonicity of muscles at rest
- sensitization of the peripheral or central nervous system,
- altered central nervous system processing
- sensitization of the sympathetic nervous system

Table c The conditions associated with the neural system of the Clinical Puzzle
### Visceral

- inflammatory organ disease or pathology (e.g., appendicitis, cystitis, acute ulcerative gastritis, pleuritis, endometriosis)
- infective disorders of the pelvic organs
- organ disease

Table d The conditions associated with the neural system of the Clinical Puzzle