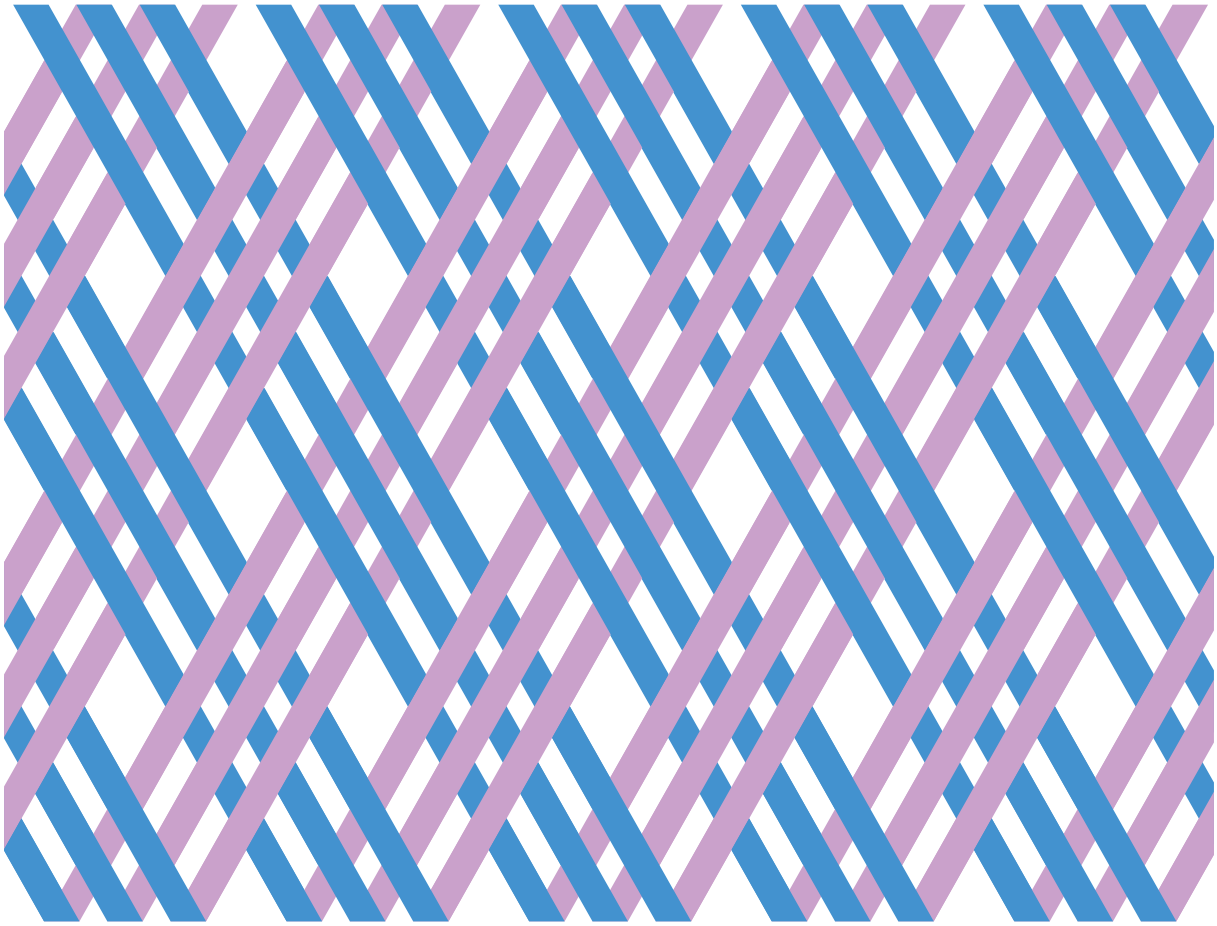
V3, February 2024

Te Kete

Diagnosis and explanations for patients with 'persistent somatic symptoms' and chronic pain

A basket of knowledge, skills and resources for students and health professionals



Written and edited by Hamish Wilson, with contributions from Martyn Williamson, Brett Mann, John Dunbar, Tony Dowell, Maria Kleinstaeuber and Jim Ross.

Foreword

By John Dunbar

Human beings are extraordinarily complex creatures. Medical school introduces us to the array of clinical conditions which afflict us, but it still doesn't prepare us fully for the complexities of patient presentations we experience.

As an Orthopaedic surgeon, I relied heavily on a biomedical model in the early part of my career and treated patients according to what their investigations suggested. I often wondered why some patients did so much better than others. Sometimes there were patients with pain or neurological symptoms for which there was no satisfactory explanation on the basis of my investigations and then I really struggled to help the patient. This was one of my greatest frustrations.

Fortunately, we are now in a time of burgeoning research into neuroscience which is helping to unravel the connections between mind and body. Where we once talked about medically unexplained symptoms, we now are beginning to explain these symptoms. Such explanations require consideration of influences within the realm of the biopsychosocial / spiritual model of health and wellbeing. Our increasing ability now to understand how those symptoms can occur opens the door to new explanations that can help to resolve them.

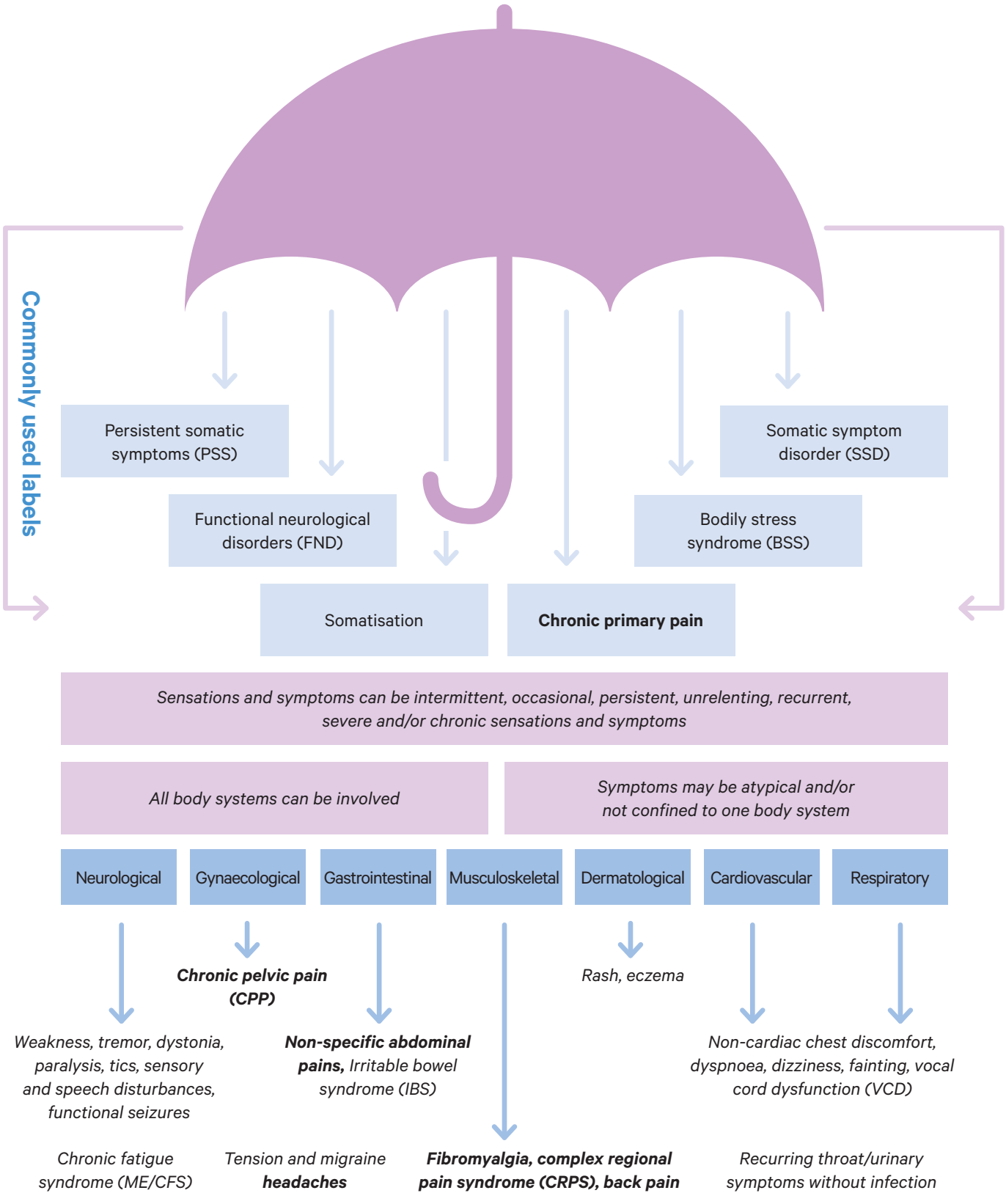
Te Kete has been compiled to bring together some of the latest neuroscience concepts with practical ways in which these concepts can be applied. It provides both doctors and patients with an understanding of these common conditions, all of which demonstrate the inseparability of mind and body. This requires a different mindset to that of the biomedical approach as well as lots of practice to convey these concepts well.

This is an exciting opportunity to begin your journey towards being able to help patients with symptoms and clinical signs that were once considered to be 'mysterious' or 'unexplained'. In contrast now, those symptoms and signs are useful clues towards more accurate understandings of how we humans function in the world.

John Dunbar
Orthopaedic Surgeon, Dunedin
January 2024.

Contents

The umbrella of persistent somatic symptoms and chronic pain



The umbrella

The ‘Umbrella of PSS’ is our attempt to make sense of so many confusing and seemingly disparate conditions. It includes some of the many labels in use, as well as examples of symptoms under various body systems. Regardless of the particular symptom however, the underlying physiological processes are similar. Understanding these processes can lead to better recognition and clinical management.

Notes

- Symptoms and syndromes can occur throughout the body, often without regard for medical understanding of body systems. Many symptoms and syndromes involve pain and are **in bold.**
- Symptom severity ranges from fleeting & mild to persistent & severe (eg CRPS, CPP).
- Diagnostic criteria for many of the recurring syndromes are listed in the Appendix.
- Patients may experience different PSS at different times or have several PSS simultaneously.
- Symptoms can co-exist alongside recognized biomedical problems such as arthritis, heart disease, Parkinsons, epilepsy and so on.
- Severity of PSS symptoms may correlate poorly with any concurrent tissue damage.
- Symptoms may involve CNS sensitization and nociplastic mechanisms of pain.
- The wide range of labels illustrates little consensus on cause or how to explain the illness to patients
- Our preference is to avoid labels that include negative or inaccurate connotations (medically unexplained symptoms, psychosomatic, ‘non-epileptic’, ‘psychogenic’ or ‘pseudo’
- seizures, ‘hysteria,’ ‘conversion’ disorder).
- The label is less important than how it is being used by clinicians, which may illustrate unconscious bias, inaccurate etiology or social and medical stigma.
- Explanations in day-to-day language are preferable, rather than using labels.
- Explanations need to vary, depending on the particular illness and the specific patient.

We have included many useful phrases for each of the explanatory models, followed by space for your own notes. Practice these phrases til they run off the tongue easily and/or modify them for your own use.

Introduction

There are many patients with symptoms that may seem mysterious or are not able to be explained under a medical framework. We are not referring to patients with rare diseases who initially elude diagnosis, or where there is a diagnostic puzzle to be solved. Instead, this Kete (a 'basket of knowledge') is about a wide spectrum of patients that can be readily recognised, once you know how to look.

Collectively, these illnesses are now referred to as 'persistent somatic symptoms'; examples include irritable bowel syndrome (IBS), chest discomfort and/or palpitations without heart disease, tension headaches, chronic back and other pains, or atypical symptoms that don't fit the usual patterns (see the Umbrella, page 4–5). There are usually diagnostic clues within the history and on examination that help to identify the illness.

> Up to 50% of patients in gastroenterology clinics have persistent somatic symptoms and 30-50% of patients in neurology have functional neurological disorders (FND).

Te Kete provides a modern perspective on these symptoms and syndromes, including many practical ideas and explanatory models that will be helpful in developing your own clinical approaches to diagnosis and how to provide specific explanations for a wide range of patients. There will be many future opportunities as junior doctors to make the correct diagnosis and to provide those patients with helpful advice.

Our first motivation for producing these resources is the suffering and healthcare costs of so many patients with symptoms that fall outside the usual biomedical framework. We believe there is much more that we can do as health professionals for these patients. This goal starts with recognising 'atypical' symptoms, making a firm diagnosis, then providing good explanations. Our second motivation is to help doctors feel more confident and competent in their interactions with these patients, thus minimising their own sense of frustration or helplessness, and improving their sense of competence and well-being.

Te Kete is founded on the following assumptions:

1. These illnesses are just as valid or 'real' as other diseases like heart or lung disease or cancer.
2. They have been associated with stigma and negative bias, partly because modern medicine did not have the tools to explain them.
3. However, 'medically unexplained symptoms' (MUS) are now adequately explained within contemporary neuroscience.

Background: Educational research

As part of an educational study funded by the Otago Medical School (OMS) (2019-2023), Hamish Wilson and Cassie Withey-Rila asked Trainee Interns about their observations of clinical practice and patients with persistent symptoms, 'somatisation' or 'functional illness' (eg chronic abdominal pain or other health issues, sometimes no diagnosis). Students commented on patients' often harrowing experiences of such illnesses who at times have quite negative interactions with health providers, as well as their own responses and ideas.

Briefly, student learning at OMS about these illnesses is patchy and inconsistent. Apart from some initial material in ELM, there is very little direct teaching on these illnesses in ALM. Unfortunately, there is highly variable role modelling from clinicians in terms of recognising these illnesses and/or how to engage with and manage such patients. Examples of definitive explanations being provided to patients were uncommon. Students were clear that they want more focused training on how to interact more effectively with these patients. These findings are consistent internationally with other educational research projects.

This Kete now accompanies several communication skills workshops at OMS. This is our attempt to rectify these current gaps in clinical training, noting that as PGY1 doctors, you will meet many of these patients in hospital practice (in wards, Specialist Outpatients, and in Emergency Depts) and in primary care. Explanatory models for these patients will be very helpful, as many of your colleagues simply don't know what to do. Learning objectives are listed on page 12.

This is the third version of Te Kete, building on student feedback so far. Te Kete includes some background concepts as well as a package of resources. You will need to translate the key concepts here into your own phrases, words and/or explanatory diagrams; ie to develop and practice your own 'patter' that explains the underlying ideas in patient-centred language. Patients will be appreciative, and this area of practice can become enjoyable and rewarding.

Useful phrases are listed on the following pages:

> The 'paradox' of PSS – symptoms are involuntary (similar perhaps to blushing), but may respond well to a simple explanation.

Common symptoms and presentations

Here is a list of common symptoms and presentations where 'persistent symptoms' disorders need to be considered in the differential diagnosis:

- Chronic or recurrent abdominal pain, nausea, vomiting. Possibilities include irritable bowel

syndrome (IBS) (patients tend to present to General Practice or Gastroenterology)
- Non-cardiac chest pain, collapse, fainting attacks (GP, Cardiology)
- Chronic headache, non-epileptic seizures, paraesthesiae, light-headedness, dizziness, walking difficulties, persistent symptoms after traumatic brain injury (GP or Neurology)
- Chronic pelvic pain, recurring urinary symptoms, sexual difficulties, impotence etc (GP or Gynaecology)
- Chronic pain e.g. back pain, fibromyalgia, CRPS (GP or Orthopaedics)
- Dizziness, recurrent sore throat (GP or ENT)
- Chronic fatigue syndrome and 'long-Covid' (GP and many others)
- Fibromyalgia (GP, Orthopaedics, Rheumatology)
- Vocal cord dysfunction (ENT, Respiratory)

In brief, all specialties have a set of symptoms where these illnesses need to be identified. The good news for most patients is recovery is possible, especially if they are provided with good explanations.

'Atypical' symptoms in a particular organ system can be a clue for considering PSS.

Terminology: why so many labels?

One of the main barriers to curriculum development in this area has been the history of confusing nomenclature. Labels in use (each with their advocates and opponents) have been somatisation, bodily stress syndrome (BSS), functional disorders and 'medically unexplained symptoms.'

We suggest that the label itself is less important than how it is being used. For example, a patient's difficulties can be described using the same term in either an affirmative or a derogative way, illustrating perhaps the speaker's underlying views. Clinically, our preference with patients is to offer explanations in simple language, rather than to use labels.

Having said that, our preferred term is 'persistent somatic symptoms' (PSS), which pragmatically carries less implications about underlying aetiology. We also use 'bodily stress', which may trigger useful discussion about physiological complexity in response to a wide range of environmental triggers and stressors. In neurology research and clinical practice, the recognized term now is Functional Neurological Disorders (FND), the metaphor being 'problems with the software, rather than the hardware'. Our preference is not to use 'medically unexplained symptoms', as PSS is now well understood and explained within contemporary neuroscience.

Regardless of labelling, there are now many comprehensive physiological models and treatment protocols which offer considerable hope and optimism for clinicians and their patient. The goal of educational initiatives and Te Kete is to enable future doctors to have more competence and confidence with these illnesses, while also addressing current clinical uncertainties in relation to diagnosis and treatment.

What these illnesses are not

Patients with these sorts of symptoms and presentations have sometimes been met with disbelief by health professionals, who are simply unable to understand what is going on. Some patients

have even been labelled with psychiatric diagnoses such as conversion disorder or 'factitious' illnesses. Unfortunately, such feelings of dissonance (where the patient's symptoms don't match clinical expectations or standard norms) has led at times to blaming the patient ('its all in their head', the patient is 'making it up', 'seeking attention', 'wanting drugs', and so on). All these attributions are inaccurate, as they are based on the idea that patients have control over bodily processes, or that psychological problems can *directly* cause PSS.

Such comments also illustrate the stigma that often accompanies these illnesses. In general, social stigma is 'the disapproval of, or discrimination against, an individual or group based on perceived characteristics' that are different. The difference in patients with PSS is that they don't appear to have a biomedical diagnosis that readily explains their symptoms. Clinical bias is to be prejudiced against that group.[1]

In our research project, students recalled many examples of stigma and bias, such as clinicians not wanting to see the patient or rolling their eyes, referring to other providers without engaging with the patient, or simply not being as attentive or compassionate as usual. Faculty interviewees also noticed that junior doctors' capacity for empathy with patients who had chronic pain appeared to reduce over time.

One of the origins of bias is an etiological one. While PSS can have an association with psychological problems and/or adverse childhood experiences (ACE), such association does not necessarily imply causation. The implication that the patient is 'psychiatric' is often offensive and counter-productive. One common outcome is that both the medical clinician and the pyschiatrist are saying the problem is in the other specialist's field, but not their own. Patients then feel invalidated and lost. These problems prevented earlier recognition of CFS/ME as a legitimate illness in its own right, and have confounded more effective approaches to treatment.

These comments are not to be critical of previous generations of doctors, as such inferences arise from misunderstandings about how body and CNS are intricately related, as outlined later in several sections. The implication of these points on stigma and bias is that each of us needs to recognise any reticence or barriers that inhibit our engagement with a particular patient; if so, the task is try and recognise the origin of those barriers.

A further manifestation of cultural stigma is that it can be internalized by patient's themselves, somewhat similar to the stigma associated with mental health issues. Patients may think they are 'weak' or 'making it up' or that they are not coping with life's pressures. Directly addressing this unhelpful (and inaccurate) self-blame can be a important initial step, prior to offering your explanation.

The absence of dedicated clinics in New Zealand

Unfortunately, the New Zealand health service does not offer dedicated clinics for many of the syndromes under consideration here. For example, and in contrast to the UK and Australia, funded chronic fatigue clinics are largely absent, as are dedicated clinics for functional neurological disorders. Many private specialists and other health professionals have developed their own treatment protocols, but the ongoing absence of state funding and legitimation of these illnesses is a further barrier to recognition, research and better management.

1 See for example: MacDuffie KE et al. Stigma and functional neurological disorder: a research agenda targeting the clinical encounter. CNS spectrums. 2021;26(6):587-92; Perugino F et al. Stigma and chronic pain. Pain and Therapy. 2022;11(4):1085-94.

This situation may reflect the low status of these illnesses within the 'disease hierarchy'[1] in modern medicine and its ongoing influence on research funding. Long-Covid clinics have been considered in New Zealand which may stimulate further generic development in this field especially with respect to Chronic Fatigue Syndrome.

In Recommended Resources, there are on-line sites and health professionals who currently offer specific treatment for these conditions.

Foundational Clinical Principles

Our research also involved interviews with leading clinicians in persistent pain, neurology and general practice who are clinically effective. Their principles and clinical approaches are summarised as follows.

Labels and physiology

1. 'Medically unexplained symptoms' (MUS) is one of many older and inaccurate labels for symptoms that are poorly conceptualised within biomedicine. However, most of these conditions are now adequately explained by contemporary neurosciences, sentinel examples being Chronic Primary Pain and Functional Neurological Disorders (FND).
2. The common underlying mechanism/physiology is 'Disorders of Perception'. This covers chronic pain and other recurring sensations in PSS, as well as disorders of movement in FND.
3. Our preferred labels include:
    - Persistent Somatic Symptoms (PSS) as the overarching description
    - 'Persistent Pain' or 'Chronic Primary Pain' for recurrent nociplastic pain
    - 'Functional Neurological Disorders' (FND) in Neurology.

Medico-social legitimacy and stigma

4. Stigma can arise from an absence of conceptual understanding and lack of clinical knowledge (often as a default to unexamined biomedical assumptions).
5. Recognition of PSS may be improved if these are considered as legitimate problems and disorders in their own right.
6. Legitimation of these problems/disorders may help to reduce current stigma and bias.

Clinical implications

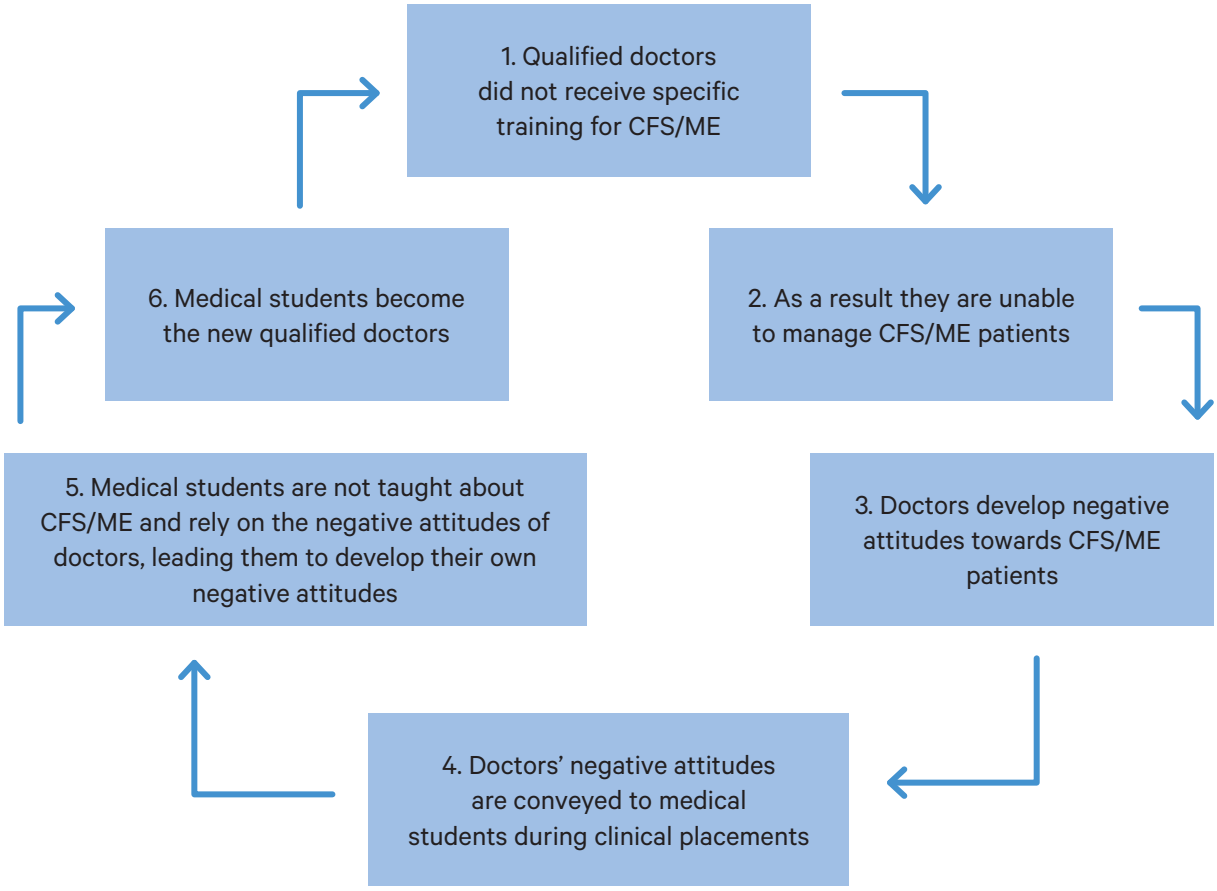
7. The possibility of PSS needs to be included in the usual differential diagnosis.
8. A 'positive diagnosis' can often be made on clinical grounds (pattern of illness, clinical signs, absence of 'red flags', negative tests).
9. A clear and positive diagnosis may help to reduce unnecessary investigations, referrals and inappropriate treatments such as use of opioids.
10. Treatment is based on providing patients with understandable explanations about the underlying mechanisms, and if necessary, addressing internalised- or 'self-stigma'.
11. Many clinicians within multi-disciplinary teams are now using these approaches: outcomes are often positive, including reduced healthcare utilization.
12. There is a 'paradox' in relation to PSS: patients' symptoms are involuntary (eg blushing), but may respond well to understandable explanations within a coherent management plan.

These principles directly address the intergenerational cycle of 'non-learning' about PSS and chronic pain in medical training, as illustrated on the next page.

1. Album, D, et al. Stability and change in disease prestige: A comparative analysis of three surveys spanning a quarter of a century. Soc Sci Med. 2017;180:45-51

To explain, the absence of coherent curricula for a specific medical topic means that student learning becomes dependent on role-modeling by clinicians. Our research confirmed the generational cycle of 'non-learning' and the development of medical bias in relation to PSS, as shown below.[1] This sort of dysfunctional learning cycle in medical training and practice can be addressed by specific teaching and learning about PSS and chronic pain.

Figure 1. Dysfunctional learning cycles in medical training



Implications for current training

We are now attempting to translate the emerging clinical principles into student learning, using the following guidelines.

1. Modern neurosciences and disorders of perception are relatively new concepts in medical and health professional practice and training.
1. New knowledge, skills and attitudes will be required for undergraduate students and for doctors in specialty training; specific learning will be required.
2. Existing tutorials and lectures related to PSS need to be integrated within a coherent structure.
3. Pilot communication skills tutorials (2022-3) in GPRH/RMIP demonstrate that teaching students about PSS is readily achievable and well received. Student learning is based on workshops and resources such as Te Kete.

1 Adapted from Stenhoff et al. Understanding medical students' views of chronic fatigue syndrome. J Health Psychol. 2015;20(2):198-209.

Communication skills workshops

Students at Otago Medical School are now attending training workshops[1] where they can apply the ideas in Te Kete into clinical work. Workshops are run on a 'flipped classroom' model, starting with students' clinical stories. Case scenarios help with clinical reasoning, then role plays enable students to practice various communication and explanatory skills. Student feedback is generally positive about this educational approach. Learning objectives and overarching principles that underpin this training are listed below.

Learning objectives

By the end of their medical training, students and junior doctors should be able to:

1. Describe and explain the physiological mechanisms underlying chronic pain and other persistent somatic symptoms such as Irritable Bowel Syndrome and Functional Neurological Disorders.
2. Within the differential diagnostic process, consider PSS and/or chronic pain as potential and legitimate diagnoses based on pattern of illness, presence and/or absence of relevant clinical signs, and relevant diagnostic criteria.
3. Engage effectively with patients in a first presentation or within a longstanding illness.
4. Use empathic communication and an interpersonal attitude that is destigmatizing, positive and supportive.
5. Use a range of explanations to help patients understand their illness.
6. Collaborate effectively with other health professionals to facilitate patient care.
7. Coach patients towards better self-understanding and self-care as part of the overall management.

Student feedback: *"Really useful role playing the scenarios and working on your own explanatory style; practice, practice, practice!*

"Alternating patient and doctor roles was very helpful, as it was great to trial ideas and observe others' approaches"

The next section provides more clinical and physiological details and introduces several explanatory models.

1 Workshop guidelines and case scenarios are available on request.

Clinical concepts
Mana-enhancing care

Recognising these illnesses

These disorders can be readily recognised using normal clinical reasoning, but you have to remember to think of them and to acknowledge them as being valid options. Here are some key diagnostic clues for the presence of persistent somatic symptoms:

1. Symptoms are unusual or do not relate well to that organ system.
2. Absence of expected symptoms in history or signs on examination for that organ system
3. There are no 'red flags' that point to major underlying disease (eg blood in bowel or anaemia).
4. Pain is out of proportion or inconsistent with initial injury (eg regional symptoms rather than anatomic, unusual associated symptoms such as tingling, swelling, redness (eg in CRPS)).
5. Presence of confirmatory clinical signs (eg Hoover's Sign in some functional neurological disorders such as gait difficulties).[1]
6. Previous investigations are negative.
7. Common pain medications have been ineffective.
8. Psychosocial factors present, that may contribute as triggers or as perpetuating factors.
9. Symptoms and signs match with relevant diagnostic criteria for common syndromes (see Appendix).

For many doctors, diagnostic questions can arise because these illnesses lie outside of our usual medical assumptions. To explain, the foundations of traditional biomedicine include biological plausibility (ie 'that all diseases must be explained by organ-based pathologies') and 'mind-body' split ('that personal and social factors do not influence body symptoms'). For much of medical practice, these ongoing 'rules of thumb' are often quite helpful; however, as they are often taken for granted, they can prevent doctors from thinking about the patient-as-a-person with wide ranging psychosocial influences that may contribute to symptoms.

Those assumptions are also now somewhat outdated, as they do not incorporate recent insights into neurophysiology, complexity and homeostasis, and the inadvertent role of the CNS in unconsciously contributing to sensations and symptoms. Many of the symptoms of PSS are simply exaggerated examples of normal physiology.

> By 'mind', we mean the central nervous system including the brain, which largely work below our usual levels of consciousness.
>
> The 'mind' includes all the automatic internal body physiology and controls that are designed to keep us functioning and safe.

1 See the wide range of FND positive clinical signs in Espay, Aybek et al. Current concepts in diagnosis and treatment of functional neurological disorders. JAMA Neurol. Doi: 10.1001/jamaneurol.2018.1264

Areas of clinical uncertainty in relation to these patients

As noted, these patients can trigger considerable uncertainty in doctors. Here are some of the clinical questions that initially can seem quite challenging, especially if these issues have not been addressed directly within undergraduate teaching.

1. The patient doesn't seem to fit the 'usual' list of diseases: the uncertainty is not being unable to find a biomedical diagnosis.
2. Worry about missing a serious disease; not knowing what one does not know (i.e. some rare diagnosis, or that the presentation was atypical for a serious disease like cancer or heart attack etc, or if there has been a false negative test that has thrown everyone off track).[1]
3. Uncertainty about the legitimacy of making a firm and confident diagnosis of 'persistent symptoms', and/or when stop investigating. What if the patient does not appear to have identifiable triggers in their history?
4. Lack of familiarity with clinical diagnostic criteria for the chronic syndromes in this field: e.g. IBS, migraine headache, fibromyalgia or complex regional pain syndrome.
5. How to initially broach the diagnosis with the patient: not knowing what words to use or specific strategies for how patients might respond.
6. How to explain the diagnosis to the patient; this uncertainty also arises because students are usually not well trained to offer explanations to patients about their 'normal' diseases, let alone these more complex illnesses.
7. What to put in the clinical notes: how to describe the illness, the diagnosis and the treatment, and not knowing how colleagues might respond to those notes.

Unless students receive specific training and have learnt about the underlying mechanisms of PSS, then various combinations of these uncertainties can add up to quite challenging consultations with such patients. Some of these uncertainties can be resolved by recognising that these illnesses are both common and legitimate, and that the underlying physiological pathways are now well established.

Emerging insights into human functioning and the physiology of PSS

Several underlying mechanisms can contribute to these intermittent symptoms which at times, can become recurrent and disabling. For example, almost everyone (including medical students and doctors) will experience various bodily sensations when acutely afraid; racing heart, sweaty palms, breathlessness, difficulty in breathing and thinking and so on. These sensations are examples of *unconscious physiological responses* to perceived danger, illustrating how central and peripheral components of the nervous system are intended to protect us from harm.

In the modern world however, the pressures and tensions of daily life often mean that the sympathetic nervous system (SNS) remains 'switched on', while the parasympathetic nervous system (PNS) remains down-regulated. Typically in chronic SNS activation, our muscles, hearts and lungs remain overstimulated, while 'non-essential' organ systems such as the skin, digestive and immune systems remain partially 'on hold.' Symptoms can thus arise from either persistent over- or under-stimulation of those organ systems (for example, palpitations, muscle tension and tendency to infections when under chronic pressure). All this occurs at an unconscious level (for more details, see the SNS explanatory model, page 26).

1 For further exploration of the 'differential diagnosis of no diagnosis', see Illness without Disease, in Wilson H and Cunningham W. *Being a Doctor: Understanding Medical Practice,* U of Otago Press, 2013.

> The clinical implications of persistent SNS activation have stayed largely below the medical radar.

A further insight from modern neuroscience research is that underlying neurophysiological pathways can become sensitized and easily triggered. A good example is migraine headache, which in response to any number of minor stimuli, is a well-defined set of physiological pathways causing headache and vomiting, without an underlying 'structural' abnormality. The problem here is not so much the trigger itself, but the recurring physiology. In brief then with migraine, triggers in the physical and psychosocial environment can set off a developing or well-developed pathway of symptoms. This is a useful example for persistent pain and many other recurring symptoms (see the Pain Neuroscience Education (PNE) section, page 37).

An emerging and potentially unifying theory for PSS and chronic pain is known as the 'Predictive Coding Model of Perception.' This will be explained further in the FND section on page 59.

Principles of clinical assessment and management for PSS

Assessment and explanation take time, but is a worthwhile investment of your energy as it may reduce the patient's need for further consultations.[1] These general principles can be used for all patients, regardless of which particular illness they have, or which particular explanatory model you use. For all patients presenting with either new or long-standing persistent symptoms, the following points need to be considered.

Developing relationship, holding and respect

- Use whakawhānaungatanga and empathy. This is doctor-patient engagement and commitment to ongoing relationship.
- Who is the patient as a person: What is their 'back-story'? (see tips below).
- Look for underlying anxiety and patterns of sympathetic nervous system (SNS) arousal.
- Validate symptoms and experiences as being real as part of normal or exaggerated physiology.

Making a diagnosis and offering this to the patient

- Check for organ-based pathology, using the usual clinical reasoning process.
- Consider unusual medical diagnoses such as rare diseases or false negative tests.
- Share your decision-making with your colleagues, especially if you have ongoing doubt, concerns about the diagnosis or about missing something.
- If the diagnosis is PSS, provide a 'positive' diagnosis and explain that to the patient.
- Use a non-blaming approach (that these symptoms and syndromes are common and valid).
- Identify and address any internalized stigma (see page 22–23).

Providing explanations

- Choose and then provide an understandable conceptual explanation for what is happening; links between life pressures and onset of symptoms, mind-body connections, SNS affecting

1 Lagrand et al. Health care utilization in functional neurologic disorders: impact of explaining the diagnosis of functional seizures on health care costs. Neurology: Clinical Practice. 2023;13(1).

various parts of the body, involuntary neurophysiological pathways, insights from pain neuroscience, lifestyle pillars, etc).
- Explain how to reduce SNS excitation and increase parasympathetic activation; use mind-body approaches such as daily relaxation, mindfulness, yoga, work-play balance and so on.
- Encourage the patient to do their own research, perhaps providing various links listed in Te Kete.

Ongoing management

- Offer to provide ongoing clinical relationship and follow up.
- Limit further investigations as much as possible, unless there is a change in pattern.
- Engage partners, family and whanau in your explanations and ongoing support.
- For almost all patients, discuss the benefits of physical activity (eg 150 min/week) and good nutrition (more whole food, less highly processed food (HPF)).
- Never initiate narcotics, or if already being used, reduce their current use. They are not helpful for these conditions and usually make management more complicated.
- Encourage self-management: exercise, sleep, taking holidays, reducing stressors, talking therapy, CBT, mindfulness meditation, group education/support, and so on.

Choosing investigations more wisely[1]

Doctors may feel some pressure to keep investigating, just in case they have 'missed' a diagnosis. With clinical experience however, many doctors become confident enough to stop investigating or referring for other opinions, as the overall illness narrative is often clear. This is especially true when the pattern of illness is distinctive for PSS and relevant tests have already been done. Here are some questions to ask of yourself in those situations:

1. If I explain my reasoning and reassure the patient, will this be sufficient without further testing?
2. Do I really need to order this test, suggest this treatment or refer for another opinion?
3. What are the risks to the patient if I do so? Are there potential adverse outcomes from procedures or medications?
4. What is the plan when the tests are negative?
5. Will ordering a test increase the patient's anxiety that I still think it is something serious?
6. Are there simpler, safer options than my powerful treatments or referrals? For example, the therapeutic use of time and organised follow-up, engaging the patient's family, general exploration of life, and so on.
7. Who can I talk to about these clinical questions: do I have colleagues who know about PSS in relation to contemporary neuroscience and/or a supportive peer group?

Tips for understanding patients and their experience of illness (the 'back-story' or personal narrative)

Getting to know the patient 'as a person' is very helpful. Being in a 'naïve enquirer' role, without judgment, may help you to understand the patient's backstory and their beliefs about their body, their illness and their life. Unconsciously held beliefs (for example, see Case 1 on page 19) can contribute to the generation of bodily symptoms. Useful questions are:

1 See for example, Levinson W, Born K, Wolfson D. Choosing wisely campaigns: a work in progress. JAMA. 2018;319(19):1975-6.

1. *What do you think is causing your problem?*[1]
2. *Why do you think it started when it did?*
3. *What does your partner and/or whānau think of your illness?*
4. *What do you think your sickness does to you? How does it work?*
5. *How severe is your sickness? Will it have a short or long course?*
6. *What kind of treatment do you think you should receive?*

One of the goals in exploring the patient's experience of illness is to learn about their perceptions of healthcare so far. As noted already, it is unfortunate that many patients with various forms of PSS have not felt validated by their clinicians, so they may initially be quite wary. Listening to the patient's story in depth can be a start in developing trust.

Further questions can elicit the patient's own therapeutic goals and the psychosocial and cultural meaning of their illness, if these issues have not already been revealed in their answers above:

7. *What are the most important results you hope to receive from this treatment?*
8. *Have your symptoms affected your work, relationships, sleep and so on?*
9. *Have you ever experienced difficult times in your life, such as physical, sexual or emotional abuse?*
10. *What do you fear most about your sickness?*[1]

ACEs, health and illness

Question 10 above links to emerging understandings about the impact of adverse childhood experiences (ACEs) on health and illness in later life. ACEs include: abuse (physical, emotional, sexual); neglect (physical, emotional); and household dysfunction (mental illness, incarceration of relative, substance abuse, violence, parental separation). Patients with chronic pain often have several ACEs, which may increase their distrust of authority figures including health professionals. It may not be necessary to unpack or further discuss background trauma, other than to be aware of their potential impact on the present. In these situations, it may be useful to start with *'What can I do to make this consultation feel a bit easier for you?'*

We appreciate that a thorough exploration of the patient's illness experience alongside a comprehensive explanation may take 30 minutes or more, perhaps up to an hour. This means that clinical staff need to have some flexibility in how they allocate their time so that longer or dedicated consultations can be available. Some clinics and organisations have recognised how longer consultations can alter the illness narrative, and interestingly, medical students can sometimes help to take a fuller history.

In brief, the clinical approach to these patients starts with whakawhanaungatanga and exploration of the patient's illness experience. This is followed by a considered explanation suitable for that particular person. This overall method enables patients to feel safe and respected, enabling opportunities for openness and engagement with their doctor and with suggested strategies.

The next section outlines 5 explanatory models, followed by a clinical example.

1 Adapted from Kleinman A, Eisenberg L, Good B. Culture, illness, and care: clinical lessons from anthropologic and cross-cultural research. Ann Intern Med 1978; 88: 251–8.

Five explanatory models

Several explanatory models are now available for clinical practice. These models illustrate how recent research has revealed various features of the neurophysiology of PSS. Each of these models will be expanded further. Here is a brief overview:

01

Sympathetic Nervous System (SNS) Education

The SNS education model explains the 'fight-flight-freeze' response to daily stimuli, how this system can contribute to intermittent and recurring body sensations and symptoms, as well as how to reduce those responses through deliberate enhancement of the parasympathetic system. See page 26.

02

The somatisation model

Life issues, tensions and stressful events can trigger the onset and continuation of symptoms, through neurophysiological pathways. Patients are encouraged to identify links between daily life and the onset and continuation of symptoms. See page 32.

03

The Pain Neuroscience Education (PNE) model

This is the favoured model in persistent pain clinics, where educational sessions for patients describe the neurobiology and neurophysiology of pain and how it is created within the nervous system. It is based on neuroplasticity and central sensitization, where the 'brain' attempts to protect the organism from harm. In contrast to nociceptive pain associated with acute injury, 'nociplastic pain' is altered sensation and pain without peripheral tissue damage. This explanatory model often involves stories and metaphors to help patients 'reconceptualise' their experience of pain, and it has become very helpful for patients, doctors and other health professionals such as physiotherapists. See page 37.

04

Explaining Functional Neurological Disorders (FND)

FND are increasingly common in all age groups and include motor disorders (weakness, tremor etc) and 'non-epileptic' or 'functional' seizures. With the recent insights into physiology, explanations are now based on the neuroscience of sensation and movement, helping many patients to recover well. See page 59.

05

Addressing lifestyle factors model

The six pillars of lifestyle medicine are whole-food based nutrition, physical activity, sleep, positive social connections, managing stress, and avoidance of harmful substances. As lifestyle factors are estimated to contribute to 80% of non-communicable disease, almost all patients may benefit through addressing one or more of these pillars of lifestyle, depending on the patient's choice. See page 71.

Explanations take time, but the investment can be well worth it.

The crucial role of explanations in the overall management

Specific explanations can help each patient reduce their worry about the reasons for their symptoms. If patients can understand the underlying mechanism of their symptoms, they can also be coached toward actions that counteract those mechanisms and processes.

'Understanding is everything' helps to reduce the common vicious cycle of symptoms that increase worry and anxiety, which can then lead to further symptoms. This is an unconscious process mediated through the SNS. A trusting and respectful interaction between doctor and patient will also start to reduce the SNS and enhance parasympathetic innervation.

Varying the explanation for each patient

Explanations need to be tailored to each patient and their pattern of illness. New illnesses may require a different approach compared to patients who have had chronic pain or symptoms for months or years. Some patients have more features of anxiety than others, and if so, directly addressing that can be helpful. We have suggested various explanations for some of the syndromes listed in the Appendix.

This means that you will need to practice a number of different approaches: for example, using a bodily stress or somatisation model for patients with a recent onset of symptoms or for chronic issues such as flare-ups of IBS. Patients with palpitations or hyperventilation can also benefit from explanations about the SNS and how it selectively affects different body systems causing specific symptoms.

Other problems such as functional neurological disorders, chronic fatigue or complex regional pain syndrome can also be initiated by factors such as infection or injury; the links to psychosocial concerns may be less clear. In these situations, pain neuroscience education (PNE) can be helpful, especially when symptoms (eg. chronic pain) become well-embedded.

Case 1 below illustrates some of these approaches.

Case 1: Life with IBS

Case 1 below is a recent representative patient history of PSS[1], followed by an explanation that draws on elements from several of the models.

A 25 year old man presented to his GP with a further bout of abdominal pain and loose bowel motions over the last 24 hours. These episodes occurred once every 1-2 months, seemingly out of nowhere. It starts with central stomach pain and he has minor relief after each bowel motion. His usual bowel habit is regular with no constipation and there is no blood. This is his final year of study at university. His grandfather died of stomach cancer. He has had no other medical problems. Examination is unremarkable.

On further history, these problems started age 5 when he first started school and were more frequent at that time. Every fortnight or so, he would spend most of the day on the toilet, eventually being labelled by his teacher as 'lazy.' Symptoms persisted in lesser forms for the next 20 years. The episodes increased in frequency last year, so he was referred for colonoscopy. This

1 All case histories are used with permission.

was normal and his symptoms were attributed to 'mental strain.' However, the general pattern did not change.

The explanation today for this patient was based on a positive diagnosis of IBS (pattern of symptoms, no red flags, normal exam and colonoscopy). It was pointed out that as 99% of the time his bowels work normally, his symptoms were not being caused by a structural or pathological process. It was more that they tended to 'overwork' at times. This observation was intended to counteract his long held belief that there was 'something wrong with him' and/or that he is 'likely to get cancer' at some point in his life. This belief was addressed directly.

The explanation included suggestions that IBS is very common and does not lead to further problems like cancer. While these points were probably also offered after the colonoscopy, it is likely they hadn't 'landed' with him as he did not see how there could be links between 'mental strain' and stomach pain.

Those sorts of links were then provided within this consultation, based on 'increased bowel peristalsis' when the SNS is activated. This required an explanation of the role of the SNS and how it can differentially affect various parts of the body, especially in low grade and/or chronic stress.

Although he was not able to recollect any particular triggers to recent bouts, his homework was to keep a diary of the episodes, noting any prior or forthcoming events or even thoughts that might be triggers. He was reassured that the intensity of further bouts will reduce if he can accept that they do not indicate a diagnosis that has been missed. He was given some reading about the SNS and how it can cause all sorts of symptoms in the body such as bowel problems, headache, dizziness and so on. He seemed to be open to all these ideas, and was asked to be in touch in a month or so to talk further.

There are possible parallels with the explanations and diagrams for chronic musculoskeletal pain on pages 48–50; instead of a 'pain control centre' inappropriately initiating swelling, erythema or weakness as an outcome of central sensitisation, in this situation the relevant 'bowel control centre' initiates peristalsis, mucous production and increased motility leading to pain and loose bowel motions. Once sensitised over time, the patterns can become well established and the pathways require very little input to fire off, leading to 24 hours of discomfort and diarrhoea.

Combining motivational interviewing with explanations for PSS[1]

A minority of patients can develop quite fixed ideas about what is causing their persistent symptoms. Such ideas can especially arise if they have not been provided with a well-considered medical explanation. One example is pain after an injury that persists long after the injury itself has healed (eg chronic regional pain syndrome). Not surprisingly, patients who are involved in ongoing battles with the Accident Compensation Commission (ACC) may be in this group.

For some patients then, it can be strategic to ascertain if they are open to your 'new' explanation. This situation is somewhat similar to personal beliefs about smoking or eating, where the preferred clinical approach now is to use motivational interviewing. This particular consultation model is respectful of patients' beliefs and personal agency, only offering information that matches the patient's readiness for change. In PSS, 'readiness for change' implies being open to

1 For more details, see Nijs et al. Integrating Motivational Interviewing in Pain Neuroscience Education for people with chronic pain: a practical guide for clinicians. Physical Therapy. 2020;100(5):846-59.

new explanations about their symptoms and why they are persisting, as well as making changes in personal habits and routines.

Combining your explanations within a motivational interviewing style makes a lot of sense, as it enables clinicians to be both supportive for the patient, while remaining realistic about their own efficacy. Here are some useful steps and phrases to consider. Firstly, elicit the patient's current beliefs about their symptoms:

Useful phrases to practice and use	Purpose
"What do you think is causing your symptoms at the moment?" *"Have your doctors/physiotherapists/clinicians offered you reasons for why your symptoms are persisting? What do you think of those ideas so far?"*	Explores the patient's ideas about cause of symptoms.

From their responses, you may be able to assess how open the patient is to different ideas and explanations. The equivalent motivational stages would be pre-contemplation, contemplation and preparation/action.

Pre-contemplation stage: Patients who have firm beliefs about the nature of their problem that are inconsistent with modern understandings of PSS. Useful responses might be:

Useful phrases	Purpose
"So from your perspective, it sounds like [summarise their ideas and beliefs] is causing your ongoing problems. I can understand that idea, as it sounds intuitively correct."	Respect for their perspectives.
"On the other hand, many people with your sorts of symptoms have recovered quite well and there are several explanations now for how improvements can happen. Would you like to talk about this further at some point?"	Explores their openness to different or fresh ideas.
"I can understand why you are doubtful about other explanations and/or that you have too much else on at present to consider those."	Respect for their current situation.
"I am happy to see you again at any time to keep talking about these problems."	Commitment to ongoing clinical relationship.
"There are other clinicians and clinics who have had success with your sorts of medical issues. Let me know when you are ready and I can offer you some names."	Sows the seeds of possibilities.

Contemplation stage: Patients who are willing to consider other reasons for their symptoms, but who have yet to put those ideas into practice.

Useful phrases	Purpose
"I am happy to provide you with an explanation for your symptoms; would you like me to go through that now?"	Helps the patient to clarify various ideas.
"Would you like me to provide you with the names of other clinicians and clinics who have had success with your sorts of medical issues?"	Illustrates there are other health providers with expertise for these sorts of problems.
"Are you open to making some changes in your life that might help with your symptoms?	Explore readiness for change.

Preparation stage/action: Patients who changing their ideas about PSS and are starting to make changes in their life.

Useful phrases	Purpose
"What do you understand now about how your symptoms are generated? I can help you to clarify those ideas if you wish"	Doctor's stance is to stand alongside the patient's journey.
"Which online materials have you found most useful so far?"	Explores how much independent work or study the patient is doing.
"From your talks with other clinicians who have been helpful, what has been the most useful thing so far?"	Explores their learning and shifts in ideas to date.
"What changes are you making in your daily life?"	Assumes that changes will be required.
"Have you got the support of family and friends in making those changes?"	Important that whānau is involved.
"How can we help to support you at the moment?'	Commitment to ongoing support and relationship.

Addressing internalised stigma

Patients with long-standing symptoms may also require an extra step – this is to address their own internalised doubts and fears about their illness (eg, that they are 'not coping', have a 'mental illness' and so on). Such misconceptions can arise in part from interactions with doctors or other health professionals who had not taken their illness seriously, or who had actively dismissed them. This lack of validation can be extraordinarily demoralising.

To address this, it can be helpful in some consultations to include some 'pre-emptive' comments. For example, *'Your symptoms are not your fault, you are not making them up, they are involuntary responses out of your control'* and so on. Useful phrases might then be:

Useful phrases	Purpose
"I know some other patients who feel a bit bad about themselves if they have these sorts of symptoms, as if they are somehow to blame or they are not coping or something. Does that feel like you at all?"	Demonstrates is open to talking about more certain issues, ie does not avoid sensitive topics.
"I want to reassure you that having symptoms like yours does not mean that you are not coping"	Reassurance.
They are simply part of your subconscious nervous system working overtime"	Explanation.
"These sorts of symptoms are not under our voluntary control; things like blushing or stammering are similar, in that try as we might, we can't easily turn them off. I can explain more if you like"	Useful day to day common examples.
"Almost everyone can get these sorts of symptoms at times in their life. They do not mean that you are weak or have a mental illness or anything. Has anyone suggested to you that that might be the case?"	Normalises, ie something that happens to everyone. Explores influence of others.

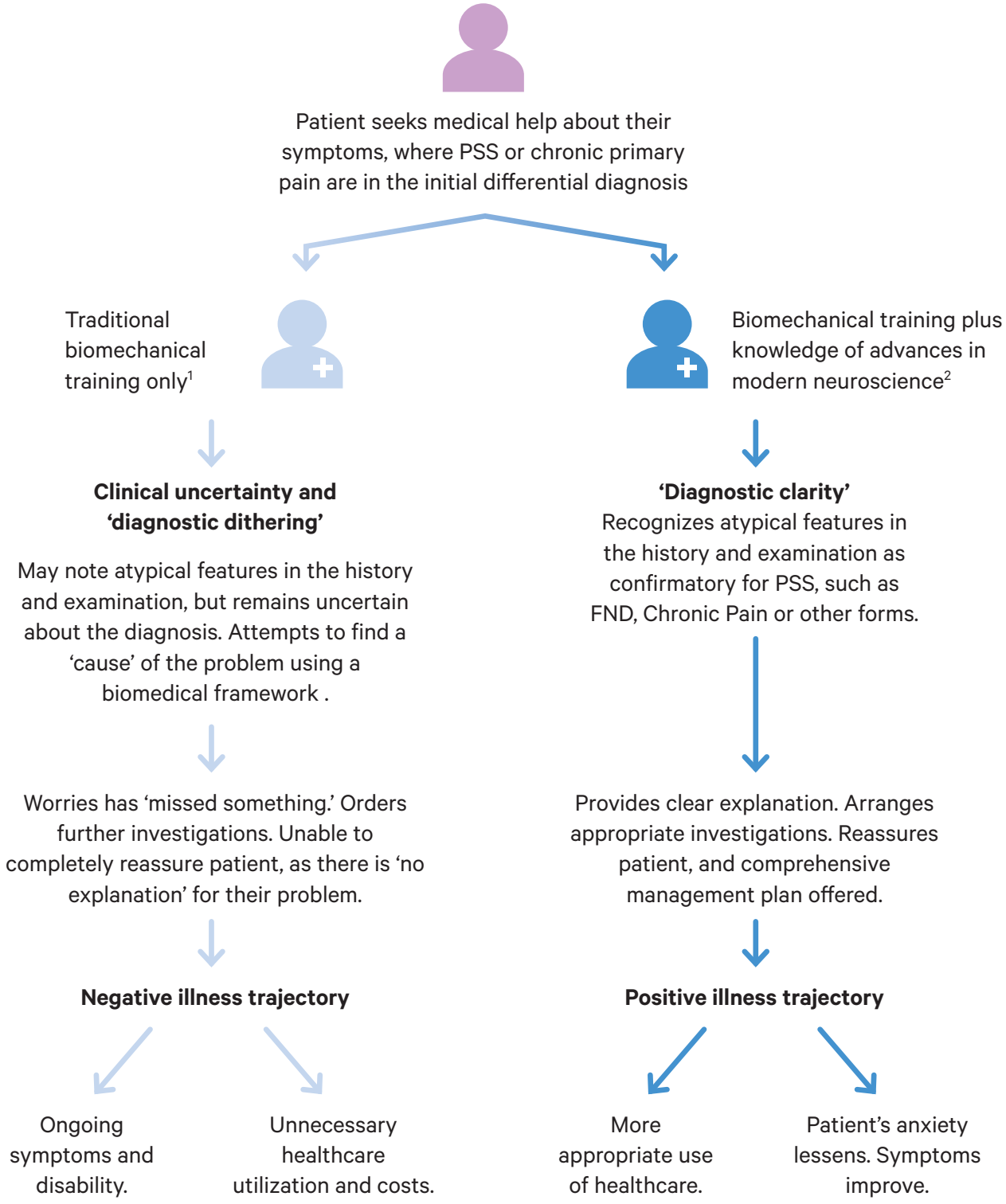
During this sort of discussion, some patients might ask: Are you saying this is 'all in my head?' It will be important to respond carefully. Examples might be:

Useful phrases	Purpose
"Well, that is a common fear, but it is in fact inaccurate. Symptoms are caused by the unconscious part of your central nervous system, not by your conscious brain. None of us can turn pain on at will, for example, nor can we stop these sorts of symptoms just by willing ourselves to do so."	Provides a biological explanation.
I guess you might be worried that other people will be thinking that sort of thing about you? Have other people implied that to you at all, and how did you handle that?"	Addresses social stigma and effect on the patient.
What are your own thoughts about these symptoms and how they started?	Provides space to hear about their own ideas.
And so on...	Keep open to more discussion.

Practicing all these sorts of phrases above is essential preparation for seeing patients with persistent symptoms. The underlying clinical attitude is respect for the patient in their situation, while being aware of the possibility of recovery. Figure 2 summarises the impact of more effective

clinical approaches to PSS (just as we found in our research). It illustrates how a good explanation can dramatically change the illness trajectory of patients with PSS or chronic pain, depending on the core beliefs of the attending practitioner.

Figure 2. The illness trajectory of patients with PSS or Chronic Pain



1 Core beliefs: 'Symptoms and/or pain are due to structural damage or disease of organ systems. Pain and other symptoms are generated at tissue level (nociceptive, uni-directional). Severity of pain is proportional to severity of damage. Treating an injury or other pathological cause should relieve the symptoms and pain.'

2 Core beliefs: 'The CNS creates bodily sensations (including pain) in order to adapt to changes in the internal and external environment and to prevent further harm'. Sometimes this occurs without ongoing tissue damage or structural or pathological changes to organ systems.

Space for notes and phrases

Further learning

Learning to help patients with persistent symptoms will require further study and considerable practice. Use these notes to reflect on your clinical experiences. Websites and further resources are also listed on page 91. There is space for confidential notes on your patients with PSS and/or chronic pain on page 94.

Summary

This section has outlined the general principles of clinical assessment and management for patients with PSS. The next sections explore each of the explanatory models in more detail, including lists of useful phrases that you can modify to suit your own clinical style.

Explanatory model 1: The ever-present Sympathetic Nervous System (SNS)

Unfortunately in medical practice, the role of the sympathetic nervous system in initiating and maintaining symptoms is often not acknowledged. However the SNS affects all body systems as part of the 'fight-flight-freeze' response to external and internal stressors. There is now better understanding of how these autonomic responses to perceived danger or stressors can trigger a wide range of symptoms, not only systemically but also regionally within the body, even when the underlying organs are structurally normal. Quite often, people can start to believe they have a serious illness; such beliefs and thinking patterns can then trigger further rounds of symptoms.

Patients with a lot of anxiety need an understandable explanation for why they often feel so terrible, or why they have particular symptoms that seem to puzzle their doctors. Explaining the links between SNS and body symptoms can be very helpful for these patients. Examples of useful phrases are listed overleaf.

Biological or evolutionary background[1]

Briefly, the stress response in humans is biologically driven as a necessary and unconscious response to danger, in order to keep us safe from harm. The sympathetic nervous system is hard-wired for an immediate and effective physical response such as fighting or fleeing. In this state, the cardiovascular, respiratory and musculoskeletal systems receive more innervation and blood supply, while the digestive, reproductive, skin and immune systems are temporarily shut down. When the danger is over, all systems are supposed to return to normal.

However in modern society with its ongoing pressures, the SNS may not get turned off entirely, a mechanism that is usually mediated by the Parasympathetic Nervous system (PNS). This means that muscles remain tense and tight, the heart and lungs continue to be overstimulated, while the gut, skin and immune systems do not function optimally. This differential activation and/or ongoing deactivation can explain symptoms in various body systems such as raised blood

1 See Porges SW. The polyvagal theory: Neurophysiological foundations of emotions, attachment, communication, and self-regulation (Norton Series on Interpersonal Neurobiology): WW Norton & Company; 2011.

pressure, ectopic heart beats, muscle pain and tension, respiratory problems including asthma and hyperventilation, irritable bowel, autoimmune disorders and chronic pain.

Secondly, a vicious cycle can develop of pressures or worry activating the SNS which then causes symptoms, which then trigger more anxiety, which further increases SNS activation, and around we go. Explaining how all this works can help the patient relax about their symptoms as they realise they do not have a terrible underlying disease, which is breaking the cycle.

This explanation also leads to the rationale for recommending various activities that lower the SNS and activate the PNS. Most patients are unaware of the biological effects of taking a break, relaxing, hot baths, mindfulness, yoga, exercise and so on, all of which can effectively help the PNS to down-regulate the SNS.

Panic attack and chest pain

The advantage of these concepts is that you will be able to identify and help patients with PSS that are affecting the chest or causing respiratory symptoms. Common examples are non-cardiac chest pain and unexplained shortness of breath. The background here is that anxiety states can range from a low level all the way up to a panic attack, however such background fears are often not recognised clinically.

There can be considerable overlap of symptoms in a full-on panic attack and acute cardiac symptoms, and there are useful diagnostic and comparative tables available (see Non-cardiac chest pain in the Appendix, page 84, for more details).

Interestingly, many people experiencing their first panic attack will seek medical, rather than psychological help. They present themselves to ED or to primary care. However, around 25% of those attending ED for chest pain will have a panic disorder, but significantly, most of them are not diagnosed at the time. This observation illustrates the common default to search for a biomedical diagnosis, rather than being clinically aware of how the SNS can create physical symptoms.[1]

The implications here are that anxiety and panic disorder need to be considered within the differential diagnosis of chest discomfort or pain, either when patients present to ED or later on return to their GP. Exploring the patient's background and current situation needs to be part of the normal clinical process in assessment. Explaining the links from their personal context to SNS arousal and then on to various symptoms may help to reduce the usual cycle, which may eventually improve the patient's health and prevent further healthcare costs. See next page for useful phrases.

Hyperventilation syndrome

It is important for all doctors to become familiar with disrupted breathing patterns (eg "un-explained dyspnoea") and chronic hyperventilation syndrome, which often complicate chronic functional illnesses. There needs to be a low threshold for enquiring about symptoms such as dizziness, light-headedness, paraesthesia, feeling one cannot get a deep enough breath, shallow breathing, holding one's breath, frequent sighing, yawning, 'atypical' chest pain, and so on. It may only take 3-4 'extra' breaths per minute to trigger these symptoms. Acute hyperventilation can be recognised readily, but chronic hyperventilation may be less obvious. They are only spotted if you are remain aware of the possibility.

1 Foldes-Busque G, Denis I, et al. *A closer look at the relationships between panic attacks, emergency department visits and non-cardiac chest pain.* J Health Psychol. 2019;24(6):717-25.

If you suspect chronic hyperventilation (eg in patients with dizziness, light-headedness, or 'having difficulty taking a deep breath'), it can be very useful to use the hyperventilation test. Ask the patient to deliberately hyperventilate (show them how) for 1-2 minutes during the consultation and see if this exacerbates their recurring feelings of dizziness/light-headedness, pins and needles, shortness of breath, or whether it causes a qualitatively different sensation.

Once again, a simple explanation to these patients can be sufficient, or you can refer to an appropriate physiotherapist for breathing exercises. As with most other patients with PSS, you could also recommend some of the resources listed overleaf or at the end of Te Kete.

Useful phrases to practice and use	**Purpose**
"It sounds like there is quite a lot going on in your life, which usually triggers our fight-flight-freeze response. This is called the sympathetic nervous system or SNS; do you know how this system works?"	These phrases introduce the idea of a possible explanation for their symptoms.
"Would you like me to explain what I think might be happening in your body?"	This phrase offers an invitation to learn about the body. If the patient is not willing at the moment, then offer the chance to return another time.
"Before I explain, it is important to realise that because you have symptoms, it does not mean you are not coping; it is just your body's unconscious reactions to what is going at present."	Pre-emptive comment to address any beliefs that symptoms mean they are not coping etc. You could also come back to this a bit later.
"OK then. We all have an alarm system in our bodies that help us adapt to events and to keep us from the harm. The common metaphor is running away from the tiger. So if there is danger, the sympathetic nervous system is hard-wired for an immediate and effective physical response such as fighting or fleeing. So our heart, lungs and muscles will temporarily get more blood so can we fight or run, while all the 'non-essential for immediate survival' organs get partially shut down, for example, the stomach, skin, and the reproductive and immune systems"	You could put more emphasis on the body system the patient is having problems with. Could also add a diagram of the body and various body systems. Patients love to take away your diagrams to read later and show their whānau.
"That's all ok in the short term, but usually, modern life doesn't go away like tigers do, so our SNS stays slightly switched on. It only takes another car pushing into our lane or a deadline at work, and the SNS will start up. This means our muscles can stay a bit tense, our breathing and heart rates can stay up, while all the other organs don't get back to normal either."	Once again, use the body system in question as an illustration.

"Does this make sense to you?"	Check understanding so far; provide plenty of space for their comments and questions
"So while your SNS is remains partially switched on, it can easily cause your current symptoms. It doesn't mean there is a problem with [your heart, lungs, skin, bowel, etc], as most symptoms tend to reduce a bit when life settles down again."	This is the explanation bit, so you need to check that they getting it so far.
"In your situation, it sounds like the fire-alarm keeps going off, even though there is no fire."	The 'faulty fire alarm' metaphor is a good one.
You could also use clinical illustrations of the SNS in other body systems, eg medical students getting ectopic heart beats prior to their exams, businessmen or rugby players getting loose bowel motions before a board meeting or a big game, getting breathless when thinking about a family member with a serious illness, feeling dizzy when there is simply too much going on, and so on. Having a 'bank' of these stories at the back of your mind is very useful.	
"Another part of the SNS is the Parasympathetic Nervous System (PNS). This is the system that tones down the SNS after the danger has gone. This is another built-in unconscious system, but usefully, there are many sorts of activities that stimulate the PNS to do its job."	Introduces the second physiological component of the fight-flight-freeze alarm and warning system.
"We can use these activities to help calm our bodies down, not only after a sudden issue, but also on a regular basis so that our SNS is not firing away too much in the background. What sort of things do you find help relax you, or when do you find your symptoms are the least bothersome"?	Use their own experiences of daily life to illustrate the workings of the PNS. If you are comfortable in doing so, offer what you do yourself to wind down and relax, and how that reduces any of your own sensations.
"PNS enhancing activities are going for a walk, hot baths, mindfulness, yoga, exercise, turning off the computer, putting the cell phones aside for a while, talking with a friend, sharing a joke, deliberately relaxing muscles, slow breathing, feeling gratitude, and so on. Would you consider doing one or more of these on a regular basis, as this may help to reduce your symptoms?"	Suggestions in a respectful way, so they remain in control of what they are going to do.
"To recap, do you understand what I have tried to explain?"	Helps to see if your explanation has 'landed' so far.
"Are you a person who uses Google to look up things? Would you like some tips as to what to look for?"	This draws attention to their own tasks and personal agency in getting better.
"Would you like to come and see me again to chat further?"	Emphasis on ongoing relationship, rather than expecting a quick cure from this consultation.

Further tips

While you are providing these sorts of explanations, many patients will reveal their own thoughts and ideas about their symptoms. Some of these ideas will be quite correct about body processes, while others will be inaccurate. For example, some patients may think their symptoms are related to a previous injury, that they have had a 'weak stomach' or similar since childhood, or that their symptoms 'run in the family'. In a respectful way, it can be useful to notice those ideas and address them directly as they can act as ongoing triggers for various symptoms.

- It is helpful to ask patients to focus more on the times they are feeling well than when they have symptoms; keeping a diary of feeling good is a useful change in orientation (see also the sections on neuroplasticity).
- Always be optimistic that symptoms can improve. As noted throughout, symptoms are caused, not by pathological processes, but by normal physiology that is on overdrive for some reason. By definition then, symptoms can be reversible, even long-standing ones. So never suggest or imply to patients that there is no cure.
- Some patients may require more focused work on their personal situation or stress. Start by asking how they resolved previous issues in their life and sometimes a referral for counselling is helpful.
- Although not the usual emphasis in medical school training, there is a large amount of research now into the biological and social sciences that underpin personal wellbeing and resilience. It would be useful to explore this, not only for yourself at the start of a medical career, but also for discussion points with your patients. Similarly, stories of people who have recovered can be very inspiring for patients.

Resources for clinicians and their patients

The Centre for Clinical Interventions is based in Perth. It is a specialist psychological service that has been very proactive in developing patient education resources. The link for clinicians has a wide variety of down-loadable resources. For example, under Anxiety, the following handouts are listed, which explain various connections between anxiety and body symptoms (through the SNS) and how to address those issues. We recommend that you use these or similar resources to develop your own explanatory model for SNS activation.
cci.health.wa.gov.au/Resources/For-Clinicians

- What is Anxiety? And, The Vicious Cycle of Anxiety
- Unhelpful Thinking Styles
- Breathing Retraining
- Progressive Muscle Relaxation
- Stress and Anxiety, Coping with Stress, Anxiety and Exercise.

Empower Therapies. Mel Abbott is an Auckland based provider who runs 4-day group workshops based on education about SNS. She has helped many patients with chronic illnesses such as persistent fatigue, anxiety, long-Covid and autoimmune disorders. See empowertherapies.co.nz

Porges SW. The polyvagal theory: Neurophysiological foundations of emotions, attachment, communication, and self-regulation. WW Norton & Company; 2011.

Space for notes and phrases

Summary of SNS model

To summarise this explanatory model, we humans are hard-wired to respond quickly to perceived danger in the environment. This 'evolutionary' perspective can be used to explain many of the symptoms of PSS. Most patients are aware of the SNS, but have not been provided with its clinical implications or how to consciously enhance the PNS. Learning how to explain the SNS/PNS needs to become part of your day to day clinical skills.

Explanatory model 2: The Somatisation model

Many patients present with symptoms that can be unique to each person and often do not fit the usual organ-based patterns. Many of these symptoms are short-lived, while other patients can develop a recognised PSS syndrome, identified by some doctors as 'somatisation.'

Somatisation is defined as the *'bodily representation of personal or pyschological distress.'*[1] Symptoms can be triggered by intercurrent concerns such as unresolved work or relationship issues.

The approaches listed here are generic and can be usefully incorporated into the usual differential diagnostic process of identifying organ-based disease. As always, this includes identifying red flags for serious problems while also noticing life-related symptoms as part of the initial work-up. Considering the possibility of PSS within the first consultation is preferable, as it is better to consider this on available clues in the history and examination, than by making than a retrospective diagnosis by exclusion after various tests have come back negative.

Making a positive diagnosis of PSS is often possible during the first consultation. A clear diagnosis requires experience and clinical confidence, but if you approach your consulting with this possibility in mind, then quite quickly you will gain confidence in identifying and managing these patients.

Clues and patterns

The following patterns can enable you to consider and at times make a positive diagnosis of PSS as part of the differential diagnosis within the first consultation:

- Time correlation of symptoms with triggering life events and issues, pressures, stressors.
- The nature of the symptoms: several body systems involved, symptoms may not correspond to peripheral nerve distributions, autonomic symptoms (eg in CRPS, there is often swelling, redness, heat, and limb weakness).
- Absence of other expected symptoms or signs for that body system (ie do a thorough history & examination).
- Previous history of other or similar unexplained symptoms that did not progress to major disease.

1 Broom B. Somatic illness and the patient's other story: A practical integrative mind/body approach to disease for doctors and psychotherapists: Free Assn Books; 1997

- Pain out of proportion to contributing event, or does not improve in the usual way with time (often nociplastic features).
- Confirmatory clinical signs present (eg in FND).
- Common pain medications ineffective.
- Maladaptive psychosocial factors (e.g. negative emotions, poor self-efficacy, maladaptive beliefs and pain behaviours).
- Multiple negative investigations in the past, or so far with this particular illness.

Key diagnostic questions when taking a history:

The usual diagnostic concern is 'What if I miss a major biomedical diagnosis?' The steps below can help to identify the possibility of PSS within your usual history, which can then be confirmed on examination (absence of symptoms and signs of organ-based disease, no red flags, presence of positive signs of PSS).

1. *"What was going on in your life around the time the symptom started?"* (temporal relationship)
2. *"Are there times of the day or week when your symptoms are less likely to be present?"*
3. *"Are there times when your symptoms are more likely or always present?"*
4. *"Is your symptom ever related to pressure, 'stuff on your plate', busyness, work responsibilities, or relationship challenges?"*

If you incorporate these questions in all clinical interviews, you will be more likely to consider and be more confident about a PSS diagnosis, whether it is the first presentation of a new illness, or a recurring or chronic symptom.

Tip

Try not to use the word 'stress' unless the patient does so first. Instead, notice and use their own phrasing and/or employ other phrases that normalize modern life (eg. *pressure, too much on plate, life-stuff,* and so on).

Using a non-blaming approach (the 'pre-emptive strike')

One of the key components of the somatisation or PSS model is to explain that having symptoms does not necessarily mean the patient is not coping. This is especially true for driven individuals who find it hard to admit to 'stress'; this may indicate underlying perfectionism, stigma or shame about having symptoms that are triggered by personal challenges in their lives.

Remember that these symptoms are never under voluntary control. Symptoms do NOT mean or imply psychological illness or weakness, a 'low pain threshold', that their pain is 'in the head', that they are 'putting it on or making it up', are seeking 'secondary gain,' that it is patient's fault in some way, or that they have mental illness.

None of these ideas or attributions about patients are accurate. They usually arise within the

doctor as part of the doctor's frustration of either not being able to make a disease-based diagnosis or feeling powerless to help that patient. In other words, these responses are known as counter-transferences[1]; they illustrate how easy it is to blame the patient, instead of learning more about that patient as a person and why their body is behaving that way.

In brief, the somatisation approach to these patients can be summed up as involving empathy, normalisation and judicious self-disclosure. This approach enables patients to feel safe and respected, offering them an opportunity for openness and engagement with their doctor and with suggested strategies.

The communication skills below are useful to read carefully and incorporate into your own consulting repertoire. For example, while some patients recognize that their particular symptoms can be triggered by personal life pressures (and who feel quite OK about that), other patients may incorrectly believe that getting bodily symptoms means that aren't coping or 'are not tough enough.' If you follow these guidelines or steps carefully, you can often pre-empt any defensiveness about underlying links. (See also the suggetions on 'internalised stigma' on page 22.)

How do I suggest PSS without 'offending' the patient?

The key overall strategy:

Empathy	+	Non-blaming comment	+	Normalisation

1. Empathy: You must acknowledge and validate the symptoms

- *"I can see that you are in quite a bit of pain/discomfort."*
- *"These symptoms are quite worrying/distressing/concerning for you, and/or..."*
- *"I would feel XX too in this sort of situation, or similar..."*

And

- *"I/we will do our best to help you sort out what is going on and to help you with them."*

2. Non-blaming comment or pre-emptive strike:

- *"These sorts of symptoms are often connected to what is going in a person's life, and..."*
- *"If there is a connection, this doesn't necessarily mean you are not coping."*

3. Normalise (and use a little self-disclosure):

- *"We can all get physical symptoms when we are under pressure or have too much on our plate."*
- *"I get ... eg tummy pain, headaches, when I was sitting exams/before giving a talk/when there are issues at home"* (ie choose something real for you that you are OK to share).

Tip

It is useful to practice these non-blaming phrases until they are completely automatic.

1 Stone, L. Blame, shame and hopelessness: medically unexplained symptoms and the 'heartsink' experience. Austral Fam Phys. 2014;43(4):191.

How do I *explain* links between life pressures and symptoms?

Explanations reduce anxiety, and once patients understand how body and mind work together, most of them don't want further investigations or referrals. Use the key phrases below and/or gradually develop your own ones. Metaphors are useful. Practice saying these phrases so they become instinctive.

Useful phrases to practice and use	Purpose
"These sorts of symptoms are often connected to what's going on in a person's life."	Introduces concept of symptoms being connected to daily life, rather than being an internal body problem.
"Mind and body are linked together. When we are under pressure or have life-tensions or issues, we can easily get feelings in the body such as nausea, pain, dizziness or [the patient's symptom]."	Could interlock fingers of both hands together to illustrate mind and body connections.
"By 'mind', I am referring to our subconscious mind, not our conscious one. The subconscious bit keeps going all the time, even when we are asleep as it has to keep our digestion and heart and breathing going automatically."	Check their understanding of automatic body functions that are below awareness and not under voluntary control.
"This part of the body can be quite sensitive to what is going in your life."	Normalizing body responses.
"It's a bit like a dam overflowing when there is too much water in it; when we have too much on our plate, our bodies can react. In those situations, most of us get something in the body, such as headache or tummy pain."	Dam metaphor.
"Have you ever felt your heart racing, or get a bit sweaty before an exam or giving a speech? These sensations illustrate how the body works to warn us of danger or harm."	Using some of the SNS explanations.
"However, sometimes it does that too well, even when we get used to having lots on our plate. These body symptoms are often just the fight-flight response being left in the 'on' position."	

You then need to see how the patient responds to your 'offer' of an explanation. Listen carefully and respond with empathy.

Key strategies

Almost all patients want an explanation for what is happening to them, whether that is due to a heart attack, cancer or bodily symptoms from the pressures of life. Some patients are already thinking that personal tensions or issues in their life may be contributing to their symptoms, while others might need to go away and discuss it with their whānau or family.

The sequence of Empathy + Non-blaming comment + Normalisation will help with trust, but may need to be repeated a number of times.

Getting the patient to return for another discussion is a better strategy than debating the point.

Address any specific fears in relation to the symptom; you may need to ask directly.

For further elaboration of the principles outlined here, see the podcast: www.goodfellowunit.org/events/somatisation-15-minute-consultation

Space for notes and phrases

Summary of the somatisation model

The somatisation model is commonly used by general practitioners and other health providers. GP registrars are routinely taught how to use this model during their training. It is especially useful for first presentations of new symptoms, which are often triggered by intercurrent personal issues and stressors. In these situations, a simple explanation can pre-empt unnecessary investigations and further use of healthcare care resources.

Some patients with long standing symptoms may not remember the physical or psychosocial trigger at the beginning. Similarly, some patients have recurring symptoms that can be set off by very minor triggers that are below the patient's awareness. So while the somatisation model is often effective, it can be helpful to have further explanations at your fingertips. Explanatory model 3 is 'Pain neuroscience education' which is based on modern neuroscience. This will be outlined in detail in the next section. PNE is particularly useful for chronic pain and other persistent symptoms.

Explanatory model 3: Pain Neuroscience Education for chronic pain

Persistent or chronic pain is one of the more striking examples of persistent somatic symptoms; its effect on life can be severe. It is one of largest health burdens in NZ (over 1 in 5 people are affected), causing more healthcare costs than cancer and diabetes combined. Curiously though, money for research into chronic pain has lagged far behind that for other diseases and students receive little specific training. The outcome is that many doctors simply don't know what to offer their patients.

Before reading on, it will be useful to try and identify your own ideas or current concepts about pain. Box 1 is a short quiz: please take your time to answer as honestly as possible by putting a circle around True or False for each question.

Box 1: Identifying one's own pain concepts

Statement	True/False	
Persistent pain means there is ongoing damage to a body structure	T	F
The body tells the brain when it is in pain	T	F
The more the pain, the greater the damage or injury	T	F
Descending neurons are always inhibitory	T	F
Pain means you are best not to move or exercise	T	F
The central nervous system decides when you will experience pain	T	F

Answers overleaf

The lived experience of patients with persistent pain is often very difficult. They may look 'normal' to others, so they often feel misunderstood, or at worse, not believed. Chronic pain is also commonly associated with stigma. This can be felt from their own families, their doctors (who may not know what to do), or it can be internalised as self-criticism and shame. Patients are usually offered a wide range of well-intended but ineffective treatments, and there are long waits to be seen at the underfunded pain clinics. In short, chronic pain has often been a mystery to both patients and doctors, further contributing to significant suffering and healthcare costs.

The good news

However, researchers and clinicians are now realising that similar to other examples of PSS, persistent pain is a potentially reversible illness. This assertion arises from recent insights through modern neuroscience and neurophysiology. However, those insights have yet to be translated into much of clinical practice or into medical school training.

> The 'science of pain' is now becoming one of the great stories of scientific progress.

Early medical ideas were that 'pain is felt as a direct response to tissue damage', so that 'more damage causes more pain.' However, we now know that *pain is created by the central nervous system as a method of protecting us from danger,* whether there is tissue damage or not. If your training so far has not included this concept, then you may find this section hard to follow, at least initially.

The sections that follow will outline why all of the statements in Box 1 are *false,* except for the last one. If that comes as a surprise, then you will need to revise your basic knowledge of pain. If your learning preferences are more towards on-line resources than reading the text that follows, on the next page are some useful links that outline the modern science of pain.

> The current definition of pain is: "An unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage" (IASP, 2023)

Nociplastic pain and central sensitization

- Neuroplasticity: www.youtube.com/watch?v=1EQ3kAPzVVI 5 min. This is from the Perth Brain Centre, which treats people with a wide range of disorders.
- "Tame the Beast". This is the leading 5-minute video on the contemporary science of pain and how we might better understand it. It features Professor Lorimer Moseley from Adelaide, one of the leading pain researchers: www.tamethebeast.org
- "The Mysterious Science of Pain", 5 min. Dr Joshua Pate, Sydney: www.youtube.com/watch?v=eakyDiXX6Uc
- "When it comes to pain, everything matters." Brit J Sports Medicine Blog; again with Lorimer Moseley. https://blogs.bmj.com/bjsm/2019/11/30/when-it-comes-to-pain-everything-matters-with-prof-lorimer-moseley/ 30 min. Very useful interactive discussion on contemporary management of low back pain.
- "Don't mislabel nociceptors as pain fibres." Brit J Sports Medicine Blog. Lorimer Moseley on teaching pain science in medical and physiotherapy schools. With a medical student, it discusses what medical students learn (and don't). 25 min. https://soundcloud.com/bmjpodcasts/dont-mislabel-nociceptors-as-pain-fibres-lorimer-moseley-on-teaching-pain-science-ep339
- Dr Ben Darlow (WSM): Pain in Primary Care. This is Ben's Prezi about the nature of pain. https://prezi.com/view/H5SFSAiqEteR1eORWhq2/
- 2023 ELM2 Lecture: Introduction to Pain. Dr Jerin Mathew. This was a new pain science lecture for 2023: https://echo360.net.au/lesson/G_a2c53e7e-869b-4375-8142-86411df1362b_b02d5cd0-8bc5-4396-8f20-2f7c1834c8a2_2023-05-04T09:59:00.000_2023-05-04T10:54:00.000/classroom (You may need to self-enroll; scroll down to 20230504: MSK_Mathew_Pain.)

What do patients say about their recovery from chronic pain?

A useful entry point into a better understanding of chronic pain is to ask patients what helped them in their recovery. Australian researchers did just this with 97 people who had had months or even years of recurring problems.[1] This group included a wide range of diagnoses and all sorts of treatments been tried to no avail, including medications, surgery, opioids, complementary therapies and so on. Thematically, their key learning insights were:

- *"Pain does not mean my body is damaged"*
- *"My thoughts, emotions and experiences will affect my pain"*
- *"I can retrain my overprotective pain system."*

So rather than some miraculous new drug or special surgery, it was the patients' own learning that had unlocked their pain cycles, and normal life was able to be resumed. Patients such as these illustrate one of the foundations of clinical management for chronic pain, which is simply teaching them about the nature of pain. However, such a learning journey may require considerable effort, as it is not always easy to relinquish our previous ideas.

1 Leake HB, Moseley GL, et al. What do patients value learning about pain? A mixed-methods survey on the relevance of target concepts after pain science education. Pain. 2021;162(10):2558-68.

Box 2: Modern neuroscience: Key concepts and definitions

Neuroplasticity: The ability of the brain to form and reorganize synaptic connections, especially in response to learning or experience or following injury.

Central sensitization: An increased responsiveness of nociceptors in the central nervous system to either normal or sub-threshold afferent input.

Peripheral sensitization: A reduction in the threshold and/or an increase in magnitude of responsiveness at the peripheral ends of sensory nerve fibres.

Nociceptive pain: Damage to tissues, e.g. acute trauma or injury, post-operative.

Neurogenic pain: Damage to nerves, e.g. diabetic neuropathy, shingles, carpal tunnel.

Nociplastic pain: Pain arising from altered nociception, despite no evidence of tissue damage. This pain arises from activation of the peripheral and central nervous system causing increased sensitivity.

Hyperalgesia: Increased pain from a stimulus that usually causes pain.

Allodynia: Pain due to a stimulus that does not usually cause pain.

Key insights from pain neuroscience: All pain is real. Pain and other sensations are created by the CNS, which is then felt in a particular region of the body. Nociception is neither necessary nor sufficient to create the sensation of pain. Generally however, the greater the rate of change at tissue level, the greater the sensation of pain. Neuroplasticity can increase sensitivity of signals; this means sensitivity can also be reduced, so recovery from persistent pain is possible.

Pain neuroscience education (PNE) is teaching patients about the modern science of pain. This is the foundational approach in pain clinics and interdisciplinary teams. In order to provide PNE yourself however, you will need to learn and understand modern pain science, including the key concepts in Box 2. Please note that these sciences may contradict some of what you have been taught in medical school, and/or some of what may seem 'obvious' from your own experience.

Contemporary pain science lets us know that recovery from persistent pain is possible.

Here are the current 'cornerstone' concepts of modern pain science:

1. Pain protects us and promotes healing.
2. Persisting pain overprotects us and prevents recovery; persisting pain can occur without peripheral damage.
3. Many factors will be influencing pain; there is no such thing as a pain that has a single cause.
4. There are many ways to retrain an over-protective pain system.
5. Treatment of chronic pain is based on patient education about modern pain science. This helps patients understand why they still hurt, and what they can do about it.

The counter-intuitive nature of pain is explained further in Box 3.

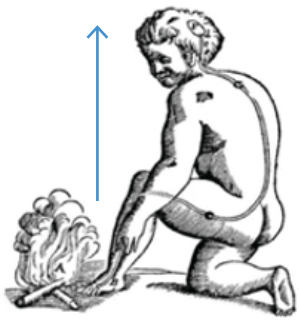
Box 3: A brief introduction to modern pain science

It seems obvious that when I hurt my foot, 'pain fibres' will send messages up to my brain to say it is injured, right? However, while this pathway is logical at first glance, the real nature of pain is more nuanced. Consider the following observations:

- After a leg has been removed, some people get 'phantom limb' pain, which can be severe.
- Expecting pain will increase the pain, such as in dental work. In another example, a builder jumped onto a nail sticking up from a plank. He looked down, and to his horror, saw a large nail coming out through the top of his boot. In severe pain, he was rushed to ED, where his boot was removed, revealing how the nail had neatly passed between his first and second toes, without even scratching the skin. The pain settled immediately.
- Pain can be deferred or reduced. In sport or in battle, people may not feel pain from quite severe injuries such as broken limbs or abdominal wounds; pain may come later.
- Pain levels change. People with chronic pain can report times when their pain was low or absent.
- Context affects the level of pain: in lab experiments, painful stimuli are routinely reported to be higher if a red light is shown at the same time, compared to pain levels associated with a blue light.
- The meaning of pain affects its intensity: pain that could signify cancer may reduce if a more benign cause is found.
- Validation of pain: People have more pain if they are not believed, as they don't feel safe to be honest about their pain.
- Routine MRI studies of the spine of normal patients will reveal many abnormal findings, but only some of those people have pain.
- The occurrence and intensity of chronic pelvic pain has very poor correlation with the presence or not of endometriosis.

Box 3 continued

In brief then, pain does *not* arise from a 'straight-through' or uni-directional flow of sensation from the periphery to the brain (as in Descartes' model from 1662). A more nuanced understanding is that there are no such things as 'pain' sensors. Instead, peripheral sensors send signals to the spinal cord, based on changes in temperature, pressure, biochemical levels and so on. If the changes are very rapid, then *pain is created by the CNS* at an instantaneous unconscious level. In the example above, pain will then be felt *in the foot,* so it can be removed from further harm. While much more complex, this system has the advantage of being extremely flexible in order to respond to a wide range of situations.



Descartes; On Man, 1662

Understanding pain in this way is counter-intuitive; it has been described by various researchers as (wait for it)… a *"mindfuck"* - it turns our old ideas upside down. In fact, every sensation or feeling in the body is produced by the CNS to strategically help us adapt to our inner and outer worlds and to keep us from harm.

It can take a while to get your head around these concepts. However, patients who grasp these ideas tend to do better in their recovery from chronic pain, have less pain after surgery, and so on. Those clinicians who 'get it' also seem to be more effective in helping patients to manage their pain. Their conceptual basis incorporates the complexity of human existence within social contexts; in other words, a biopsychosocial appreciation of personal experience and multiplicity of relevant factors.

In brief, the task of various tissue nociceptors is to report changes in temperature, pressure, chemical balance and so on. If the rate of change is very rapid, the spinal cord and CNS will interpret those changes as pain, causing you to do something. In contrast to what has often been taught to students, there is no such thing as 'pain receptors' or 'pain nerves'.

If these concepts seem challenging, then our suggestion is to find a way to sit alongside them for a while. Perhaps you could do your own research on pain such as listening to podcasts, asking pain specialists what they actually do, and even better, asking if you can watch them explain pain to a patient. You could also ask patients who have recovered; what worked for them or what made a difference? Keep a journal of patients you have seen with pain, and so on. There is also a wealth of laboratory and clinical research now available through Google Scholar that backs all that we are suggesting here.

At present however, there is a very large conceptual gap between those clinicians who 'get' the modern pain sciences and those who remain unaware of it. The latter group tends to request

unnecessary X-rays for patients with recurrent low back pain, repeat another laparoscopy looking for endometriosis, or provide ineffective opioids. So, a word of caution here: if you do start to explore neuroplasticity and modern pain science and start to re-think how the body works, it can feel a bit disconcerting – many of your colleagues may still be thinking 'uni-directionally' or anatomically, as if the body is a sort of predictable machine. Their underlying model is largely biomedical.

After many thousands of years of evolution, however, it would be surprising if the human organism had not developed an amazingly complex and adaptable system for responding to danger in the environment. While acute pain looks as if it starts in the damaged tissue, the brain is in fact simultaneously interpreting the rapid changes notified by the nociceptors and is creating the sensation of pain in the foot, so you can withdraw your leg from danger. Pain levels are always calculated strategically by the CNS, in your best interests, but at times, the CNS can come up with some inaccurate calculations.

What is the physiological basis of chronic, persistent or recurring pain?

The CNS takes its role in protecting the organism very seriously. And unfortunately, it can continue to create pain when such protection is no longer necessary. A good example is Complex Regional Pain Syndrome (CRPS). After a minor injury, the person's pain, swelling and disability can persist, well after the injury has in fact healed. Those initial responses to injury were helpful, as the automatic CNS adaptations cause swelling, discomfort and tenderness which forces rest to prevent further damage, allowing the spontaneous process of healing to occur. The immediate tissue and nerve sensitization are normal and beneficial. However, if for some reasons the CNS gets stuck in a protective mode, the nociceptors and sensory systems get fired up and remain on high alert.

This is a bit like a faulty fire alarm going off when there is no fire. Even worse, the fire-alarm is automatically linked to the local fire-brigade who now come rushing in with sirens on and much excitement. This over-protective CNS process activates peripheral and central sensitization, which are the major pathophysiological features within chronic pain. Before going further, however, the concept of neuroplasticity needs to be further explained.

Neuroplasticity and nociplastic pain

Learning a new skill such as riding a bike requires new neural connections to be made within the brain. Particular neuromuscular pathways will develop in order to coordinate vision, proprioception and muscle contraction. As you get better at riding, the 'bike-riding connections' or 'biking centre' of the brain expands as do those neurophysiological pathways. Expert riders would have much larger biking centres than occasional cyclists. This is neuroplasticity, an immediate and day-to-day response of the CNS in relation to what we do. It happens below our level of awareness and is not under our conscious control.

As noted before, pain is generated by the CNS in response to particular inputs. Its purpose is to prevent further harm. In chronic pain, the 'pain generating centre' of the brain becomes larger and more efficient at what it does; i.e. to create pain, reduce movement, increase inflammation and so on.

These activities of the pain generating centre can lead to 'hyperalgesia', which is more pain than expected for a particular stimulus. 'Allodynia' is pain caused from minor stimuli that don't usually

cause any pain. The underlying CNS mechanism here is peripheral sensitization; nociceptors and peripheral nerve endings become more sensitized, increasing their upwards input to the spinal cord and brain.

Allodynia and hyperalgesia are now well recognised clinical clues that CNS sensitization is occurring. In addition, symptoms and signs may be unusual or atypical for that organ system, or regional rather than strictly anatomical (e.g. the whole lower leg is involved rather than just the ankle joint). Asking the patient to fill in a pain body-map may reveal the widespread areas that are affected, while pain or sensory changes may be inconsistent with known dermatomes.

It is interesting that during the majority of medical training, symptoms and signs that don't fit the usual biomedical diagnoses are sort of discarded as *'irrelevant'* or *'noise'* compared to the usual confirmatory clinical signs of pathology (e.g., finding rebound tenderness in suspected appendicitis). However, those 'atypical' findings on history or examination have considerable significance, as they actively illustrate the CNS processes at the heart of the ongoing problem. Pain in these situations is now known as *'nociplastic'* pain, differentiating it from the other main mechanisms.

> Nociceptive and nociplastic terms stem from the Latin verb *noceō* – to harm or injure. Nociceptors inform us of changes in the tissues and potential harm, while the term nociplastic helpfully includes the idea of plasticity and potential reversibility.

Many current clinicians are unaware of these concepts or the significance of nociplastic pain, so do not realise that central sensitization is present. The clinical outcome is that they may continue to investigate chronic pain patients as if they had an acute mechanical problem such as a fracture or an infection. Behavioural examples would be to repeat all the previous tests for patients with recurring abdominal or chest pain. These management decisions are unhelpful as they are based on inaccurate assessment of the underlying problem. And without getting an answer, patients fears are not reduced, increasing the CNS production of pain.

Arising from local research,[1] nociplastic awareness is defined as 'the clinical capacity to recognize features of nociplastic pain within a patient's history and examination.' To identify nociplastic pain, you must be aware of its possibility, then actively look for it within particular consultations. Here is a list of identifiable clues in the history and general background of the patient that raise the possibility of nociplastic pain.

- Atypical symptoms are present
- Nociceptive or neuropathic origins are pain seem unlikely or if present, do not fully account for all the pain or other symptoms
- On either history or examination, there is sensitivity to touch, pressure, movement, heat or cold (hyperalgesia or allodynia)
- The patient has increased sensitivity to sound, light or smell
- The patient has sleep disturbance or fatigue
- The patient has cognitive problems such as concentration or memory issues.

See also the diagnostic flow-diagram in the Appendix, on page 78.

Case 2 is a representative case history that illustrates how nociplastic pain and other signs of CNS sensitization were present but not identified, leading to a delay in diagnosis. Physiological interpretations are included in brackets.

Case 2: Life disruption: nociplastic pain and CRPS

After riding a motor bike for several hours, a 32 year old woman developed pain in both hands and arms. Rather than settling, the pain increased over the next week, spreading up both forearms *[atypical response to minor trauma, regional symptoms rather than anatomical]*, and was associated with weakness, patchy numbness and 'zinging' or tingling sensations *[signs of CNS sensitization]*. The illness became very challenging emotionally with sleep and general concentration also affected *[neuroplasticity and systemic signs of CNS sensitization]*. Past history included undiagnosed symptoms including a laparotomy for abdominal pain, which in retrospect was related to intercurrent life stressors *[life patterns of PSS triggered by intercurrent stressors]*.

Her family doctor referred her for investigation of 'compartment syndrome' or bilateral referred pain from her cervical spine *[search for biomechanical or nociceptive origins of pain]*. Standard medications, physiotherapy and massage were ineffective *[further clues to nociplastic mechanisms, as those treatments are less effective in nociplastic pain]*.

After 18 months of persisting pain *[illustration of the usual delay in reaching a definitive diagnosis]*, she was diagnosed with CRPS. This diagnosis was reached by reviewing the pattern of illness, the atypical nature of symptoms and normal investigations. She made a steady recovery after being provided with a comprehensive pain science explanation for her symptoms.

1 Wilson H, Dunbar J, Cadman E. Are there clues to nociplastic pain mechanisms within patients' stories of chronic musculoskeletal pain? A qualitative study. 2023. Current research.

Applying pain science to clinical practice

In brief then, acute pain is strategically produced by the CNS in response to various nociceptive input. The goal is to keep the organism from harm.[1] Persisting pain can occur without tissue damage. This occurs when the CNS has become over-protective, involving peripheral sensitization such as nociceptive sensitivity and down-regulation of the usual spinal cord inhibition. Pain levels are influenced by many factors in the individual patient, so addressing those factors will always require a comprehensive patient-centred model.

Pain neuroscience education (PNE) is now the mainstay of effective management of chronic pain. Whether provided by individual clinicians or in multidisciplinary pain centres, almost all patients can be readily taught the modern science of pain. Group education helps to validate the experience of pain by hearing other peoples' stories within a shared learning environment. Some patients may also require specific physical interventions, such as nerve blocks or focused physiotherapy, but such treatments will always be more effective if the patient understands the rationale. Usually, that their symptoms arise from neuroplasticity and brain sensitization, and that their treatments are designed to lower CNS sensitivity.

Pain specialists and other practitioners of PNE have usually completed specific training in order to develop skills in communication and explanation. They often use diagrams and metaphors to augment their coaching of each patient about the nature of pain.

Medical and other health professional students can readily learn how to do PNE, being a logical development of their consultation and communication skills. Whether in ED, hospital outpatients or in general practice, you will meet countless patients with persisting pain. There will be many opportunities to help inform them about the nature of pain and why it can continue. The necessary requisite is sufficient knowledge and understanding of modern pain science.

The next section provides some examples of communication skills and techniques that you can try with patients who have persistent pain. This approach includes various diagrams which may help you to develop your own explanatory style.

Principles for providing a 'pain talk'

Here are some general guidelines for setting up a consultation in order to talk about pain in more detail; ie giving a 'pain-talk'.

- Find a private room if possible and allocate sufficient time (eg 30-45 min); don't rush the patient or (ideally) have beepers going off
- Remember whakawhanaungatanga and mana-enhancing care
- Don't expect them to understand immediately as they may need to take time to consider and to revise their own ideas
- Being respectful of their current beliefs is the best way to help them recognize those beliefs, then they may perhaps be able to consider other ideas
- 'Lecturing' to patients may not be helpful; the juggle is providing them with new information, respecting their current pain and situation, and providing a setting in which they might become open to new learning
- The setting, context and attitude required to hold an educational consultation about persistent pain are somewhat similar to a 'breaking bad news' consultation.[2]

1 Taking an evolutionary perspective, all actions of the CNS are driven largely by what is known as the 'pleasure principle'; this is the instinctive avoidance of pain (whether physical or emotional) and the instinctive seeking of pleasure. These instincts help organisms to firstly survive and then to flourish.

2 See for example: Baile et al. SPIKES—a six-step protocol for delivering bad news: application to the patient with cancer. The Oncologist. 2000;5(4):302-11.

Micro-skills

- Explore their prior and current ideas about pain, what they had been told or what they have found on Dr Google
- Use plain language
- Use diagrams (see overleaf)
- Use metaphors (see page 53)
- Use other patients' stories of recovery (anonymously)
- Check patient understanding all the time
- Ask what they are taking away from your talk; one key idea they may now consider
- Ask what they will say now to their family
- If they are open to exploring, provide links to further resources (eg those on page 39)
- Arrange a second discussion time
- Let them know you are there for them, whether or not they agree with your suggestions.

General guidance about managing persistent pain also needs to include the following:

1. Provide a general steer about not having further investigations or surgery
2. Provide some general advice about not starting opioids, or if already on them, offer the idea of eventually reducing them
3. Recommend the patient finds someone who can further explain their pain and stand alongside them, while the patient does their own research and learning about persistent pain.

Diagrams and explanations

Given there is so much stigma around chronic pain, gaining your patient's trust is the first step. Many patients feel they have not been believed by their health professionals, but trust can grow if you take sufficient time to hear and acknowledge their experience of pain and what their general illness has been like so far. Validation of their experience may also help them consider and retain what you have to say. Pain is always real.

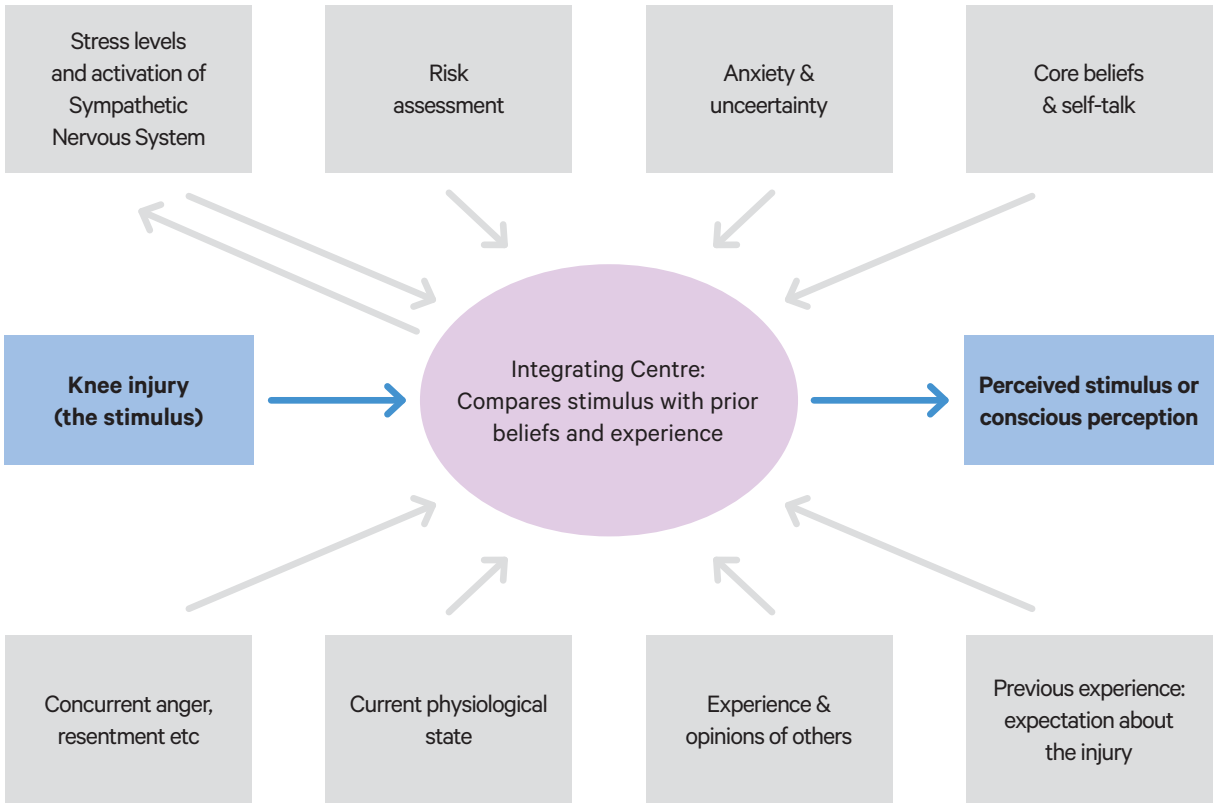
Diagrams and illustrations are useful explanatory tools. For example, Mr John Dunbar is an orthopaedic surgeon in Dunedin who uses simple drawings with patients to explain CRPS or other chronic musculoskeletal pains.[2] Although he doesn't use medical terms with patients, Diagrams 1-4 overleaf illustrate how it is possible to develop chronic pain from a minor injury. The mechanism is hypersensitized neurophysiological pathways contributing to nociplastic pain.

Mr Dunbar's explanations have helped to reduce or even resolve longstanding symptoms for many patients. The exact model is less important than providing a conceptual understanding of pain as a result of neuroplasticity and how the patient can learn to reverse that process back to more normal levels.

2 Adapted from Dunbar J, Wilson H. Emerging models for successful treatment of complex regional pain syndrome (CRPS) in children and young adults. J Prim Health Care. 2019;11(3):283-7.

Diagram 1: The role of the integrating centre in response to a stimulus

This illustrates how the CNS/brain will process information when there is a stimulus such as an injury to the knee. There are many contributing factors to the eventual modified or 'perceived' sensation.



Based on contemporary neuroscience, nociceptors register a rapid change in knee tissues, so they inform the spinal cord and CNS/brain that there is an acute problem. Incoming nociceptive data is then processed by the 'Integrating Centre'; as in Diagram 1. The Integrating Centre works below the level of conscious thought, rapidly comparing this data with previous experience of similar data (eg previous injuries) to see if there might be a serious problem. It also receives input from other centres, such as core beliefs about one's robustness, physical capacities, ability to recover, current physiological status at the moment, opinions from others, and so on.

The sum total of all these influences will now be a 'modified or perceived stimulus'. If there is sufficient concern, the CNS will instantaneously produce pain in order to remove the leg from further danger.

Background levels of stress also contribute to the integration. For example, if the person already feels unsafe, threatened or out of control, then all neurosystems will be on higher alert or sensitized, so pain levels will be modified. The Integrating Centre might also trigger a further stress response involving the sympathetic nervous system (SNS). This prepares the body to respond to this stimulus with 'fight, flight or freeze', depending on the situation. This process might increase pain, which increases SNS activation in a rapid feedback loop, shown by the double arrows.

Diagram 2: The conscious and unconscious brain

This diagram now locates the Integrating Centre (IC) in the subconscious part of the brain. Having compared the new stimulus against all other prior data, the IC informs the conscious brain that there is pain, which is felt in the leg. The IC also controls motor function, so the leg becomes weak and needs to be rested, while the Inflammation centre usefully initiates the tissue healing process. All this occurs unconsciously (at spinal cord level) and is a normal response to trauma or infection.

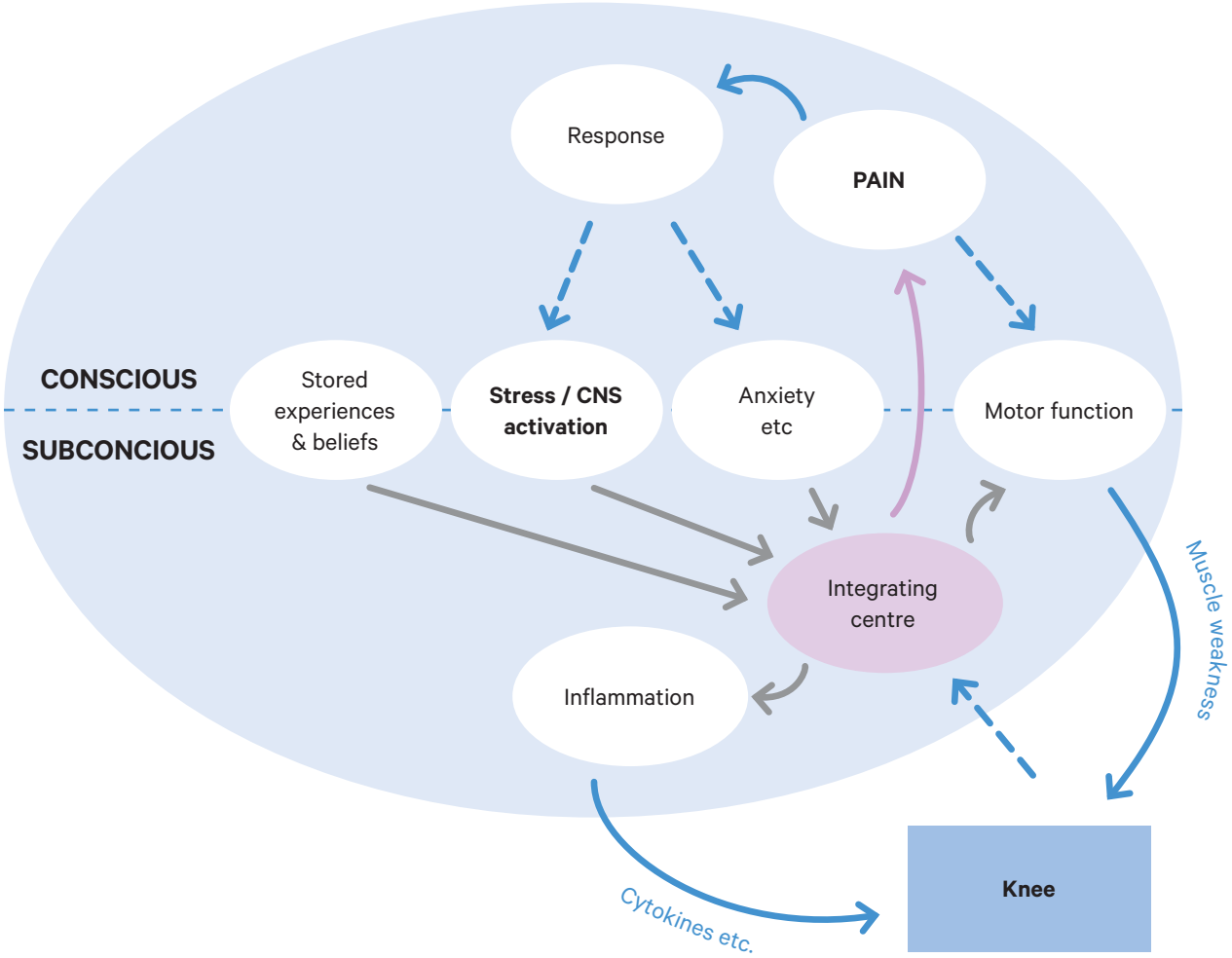
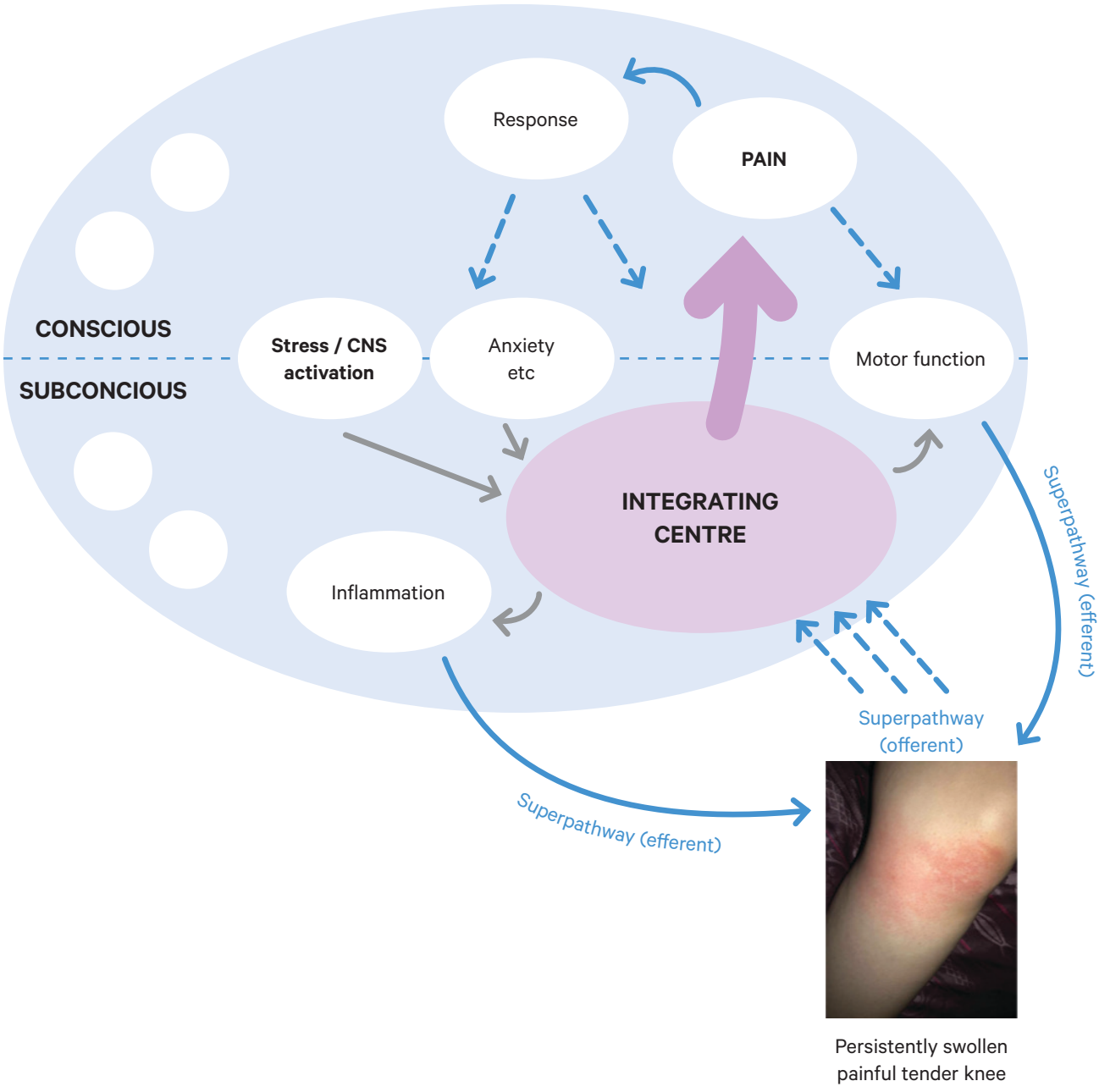


Diagram 3: Neuroplasticity and chronic pain

However, if there is continued input to, and output from the IC, then motor and inflammatory pathways continue to become more established, while pain continues to be reported to the conscious part of the brain. Neuroplasticity means that this particular integrating centre is now quite enlarged, with much more active nerve highways back down to the leg to reduce motor function and increase inflammation. The photo is the knee of a person with complex regional pain syndrome, showing swelling and inflammation. The smaller circles illustrate other neural centres that get crowded out; eg centres for sport, play and laughter.

The diagram also highlights the 'vicious loop' of pain to more stress responses, to more pain, all at an unconscious level. The second vicious loop is the efferent and afferent pathways which feed into each other; more inflammation causes more peripheral input from the knee, which increases the afferent pathway and subsequent CNS responses. Clinically, the lower leg may now show hyperalgesia and allodynia, both signs of nociplastic pain.



Persistently swollen painful tender knee

Explaining these processes takes time, but it is well worth it. The patient now understands why and how their knee still hurts. Because they understand the difference between 'stimulus' and 'perceived stimulus', they become less fearful of their pain. They know it doesn't mean there is ongoing knee or tissue damage. This insight helps to reduce anxiety, so there is less input to the IC, thus breaking the vicious cycle. In brief, their task is to try and shrink down the over-enlarged IC and reduce the sensory input from the tender and swollen knee.

Understanding the diagram provides the rationale for other components of modern pain management such as relaxation, doing enjoyable things, mindfulness and so on, as they help to reduce the background physiological sensitivity of the CNS and increase parasympathetic activation (which counters the SNS), all of which helps to shrink the IC. The role of CBT can now also be understood, as it addresses underlying core beliefs or 'priors'; assumptions about the body or the injury that can feed into the IC and get in the way of recovery.

Similarly, some patients also try to visualise some sort of 'block' in the afferent and efferent super-highways, such as a block to cars on a motorway or to the flow of water in a pipe. This sort of visualisation process can be quite helpful.

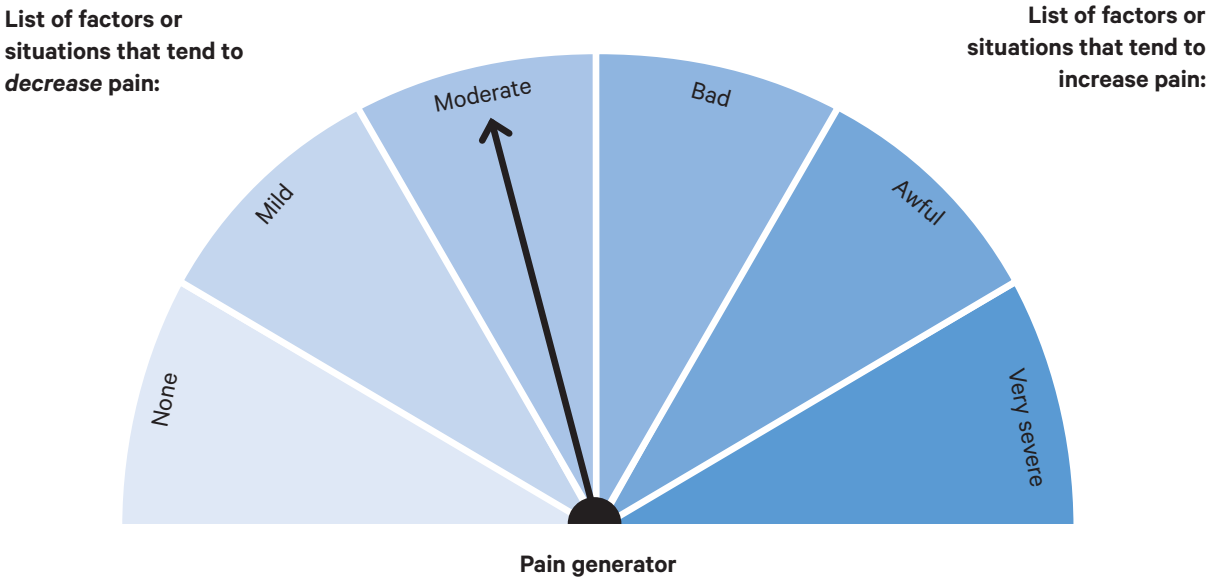
The patient realises there is no ongoing damage in their knee, so it is safe to start moving, which will also help muscles become stronger again. Using their leg won't cause any damage, even if their IC is worried about that. Although the leg will be a bit sore initially, that's not a sign of further damage, it's just those pathways still overdoing it.

All these explanations are thus based on neuroplasticity; the daily increase and decrease of CNS connections throughout the body depending on how much they are being used. This is why teaching patients about these processes requires a prior understanding of modern neuroscience.

Diagram 4: The 'pain generator'

A variation to the integrating control centre model is the 'pain generator', which is the end result of central and peripheral nervous systems contributing to the sensation of pain. Once the patient is on board with the modern science of pain and how it is generated by the CNS, then this diagram can help them identify contributing or relieving factors.

The arrow points to the sum total of all the factors that might increase the pain, versus all the factors that might decrease it. Ask the patient when their pain is least, versus when their pain is more present, adding their answers to the diagram.



Thinking about pain in these ways can be incredibly helpful, as illustrated by the patients we mentioned at the start of this section. As a reminder, personal ideas that helped their recovery were:

- *"Pain does not mean my body is damaged"*
- *"My thoughts, emotions and experiences will affect my pain"*
- *"I can retrain my overprotective pain system."*

Useful phrases and metaphors

Metaphors are useful when explaining any condition to patients, especially those in pain or those due to have surgery. Here are some common sayings.

Forest metaphor

- *Having persistent pain is a bit like being lost in a forest; its lonely, no-one knows where you are, and you don't know how to get out.*
- *However, there is a map that you can use; this map is called 'Learning more about Pain', what sort of factors increase it and what decrease it. We can guide you through that map.*

Railway metaphor

- *Persistent pain is a bit like moving into a house right next to the main railway line; every time a train goes past, it feels scary that it will come right into your living room! But over time, you do get used to it, and you don't even hear it.*
- *Persistent sensations from your [leg, stomach, head] can be a bit like that, as initially you think the sensations mean there is something seriously wrong or you are in danger. In your situation, we know the pain is coming from your pain system itself, as it has become very sensitive. It is possible to gradually learn how to 'turn-down' your pain centre or pain regulator or pain generator, and we can help you with that.*

Faulty fire-alarm metaphor

- *Persistent pain can be a bit like the body's alarm system going off unnecessarily, a bit like a faulty fire-alarm that goes off even when there is no smoke or fire.*
- *It is a glitch in the software, rather than in the hardware.* This is also one of the commonest and most useful metaphors for functional neurological symptoms, such as motor symptoms such as weakness, sensory symptoms or functional seizures. (See the section on FND in the Appendix).

Addressing self-blaming or internalized stigma

For people who feel they 'cope well' with life, developing mysterious symptoms can lead to self-doubt, even self-recrimination. For 'copers' then, it can be a terrible assault on one's sense of self and can trigger self-blame. These tendencies can be further exacerbated in chronic pain, given the stigma that is often attached to it. It is important to fully appreciate that pain can be triggered by any number of factors, not just psychological ones.

- *Anyone can develop this sort of pain in certain circumstances; it's nothing you have done that has caused it... "it's not your fault"* (this can be repeated several times)
- *Other people might imply that your pain is 'all in your head' as if you can somehow consciously create pain. This is not true, as we simply can't turn pain on or create other sensations in our body off and on at will. Try now to make your foot go all numb, or start to get chest pain!*
- *The brain and the spinal cord all work below our level of control or consciousness to create the sensation of pain.*

Exercise and re-training

- *It is important to realise that having pain does not mean there is damage there; you won't cause any further harm with movement or gradually exercise.*
- *"Motion is lotion"* (a great phrase to use about the benefits of movement and exercise)

Other useful phrases

- *Extra-sensitive alarms: Your nerves work like an alarm system to protect you from hurt, the alarm system in your [back, neck, leg etc] has become extra sensitive*
- *Surgery and hospital experiences can ramp up the alarm system, so tend to give you more pain*
- *Calming sensitive nerves: You can calm down the alarm system by understanding, knowledge and movement*
- *Hurt does not equal harm: You can be 'sore but safe'*
- *No freaking over flare-ups: The ups and downs of normal recovery*
- *Pain is normal: Pain after surgery is to be expected and normal and will settle in a few days.*

Case 3: Persistent back pain and the 'attention model'[1]

This is a recent GP narrative about their effective interaction with a patient who had recurrent upper back pain.

I developed this approach after reading about neuroplasticity, always remembering that *neurones that fire together, wire together.* The conceptual model I use is also informed by my understanding of human cognitive biases and how our brains function automatically below or 'sub' our consciousness and awareness.

The approach is based on the brain's function of keeping us safe allied with its ability to get stuff mixed up owing to how we handle the information overload of every moment of every day. The model is 'Increased attention leads to increased flight/fright response which increases attention' to the problem, i.e. more neurones are recruited and we notice it more frequently and more easily.

The treatment is to divert attention and associate with relaxation or calmness to reverse the vicious cycle. I'll describe it using a case example of a patient with chronic thoracic spine pain stemming from a relatively innocuous injury 18/12 ago. Innocuous that is in terms of damage, but painful at the time.

The patient is using codeine and has run out 6 weeks early as her pain has been getting worse.

Step 1. Determine the absence of pathology at the site of pain with a careful examination. This reassures the patient that you are taking them seriously. (At this point it's not useful to share your findings as this can typically produce an abreaction as the patient feels their legitimacy is being denied.) This particular patient has no tenderness to palpation either in spine or paraspinal muscles. She had an MRI

1 Kindly provided by Dr Martyn Williamson, Alexandra.

Case 3: The 'attention model' continued

organised by the back specialist which showed some narrowing of her disc spaces in line with degenerative change.

Step 2. Ask the patient questions like "What impact is this pain having on your life? What can't you do or enjoy that you could before?" These questions alert you to the patient's suffering and your careful listening helps validate their lived experience. Once you have done this, then they are much more likely to be willing to listen and take in what you have to say. Another useful question is "What do you think is going on with your body to cause this pain?" With this lady she had the idea that her discs were wearing away and that things would get worse. Quite a distressing situation for her to be in and raising the level of anxiety, which is an important contributing factor.

Step 3. The explanation:

> "What's happening to you is this. The brain's main job is to keep us safe and yours is trying its best to keep you safe. And it's doing all its stuff automatically without you being aware of it. When you twisted your back 18/12 ago and felt pain, the brain noted 2 kinds of things, the date the time and the circumstances of the injury plus the degree of risk. When a pain is severe, as acute back spasm can be, it senses our natural anxiety and labels the pain as dangerous for us. Once it's done that it keeps an eye out for further sensations from the area and overtime becomes an expert at spotting them. Your brain now has devoted more brain cells to task of monitoring your back than mine has for example. So, it's busy spotting signals to act as an early warning system. When it does notice something, it remembers the link to your flight/fight response and reinforces that. It magnifies the sensations in order for you to take the necessary action.
>
> The problem is that your brain is now paying too much attention to your back. There will also be other times when you barely notice your pain, perhaps because your brain's attention is on something else, especially when this something else does not involve the fight-flight-freeze pathway. Can you think of activities when you notice it less?" (These are virtually always present, and the patient gets a little 'aha' moment as they realise this.)

Now we can share our own findings and interpretation of the MRI, thus providing a way for the patient to reframe or reconceptualise their problem and to create a more useful model of understanding.

> "I've looked at your back thoroughly and there's no damage that we or your automatic brain need to be concerned about. In other words, you can SAFELY perform any activity within your usual range. Your back is as safe from damage as anyone else's. If we hauled in 100 people off the street at random, who are your age and did an MRI on them we would find multiple people with the same disc changes as you. They will be pain-free. Those changes on the scan are a

Case 3: The 'attention model' continued

normal part of getting older for many people."

At this point I would usually check that the patient is following the discussion. Assuming they have, and are accepting of the explanation:

> "So, what this means is that when you feel this back pain, it's just your brain warning you about danger when it doesn't need to. You can reduce the sensation by focussing the brain's attention elsewhere as in those activities you identified earlier. You can also thank your brain for the warning. 'Thank you, brain, for the warning, I'm safe, you can relax now'. When you associate that comment with relaxation techniques such as slow breathing with an out-breath up to twice the length of the in-breath, the brain will start to connect the pain with relaxation and calm and divert its resources somewhere more useful. In this way you can retrain your automatic brain and reduce or get rid of the problem, so it no longer impacts your life. How does that sound?"

The patient is grateful you've been able to paint a much more satisfying future than they had before. They will often be excited by the prospect of change.

> "So, I think that if you practice these two things over the next few weeks you won't need pain killers anymore. How does that sound?"

This patient made a very good recovery from her pain over the next fortnight. She also knows what to do now, if she gets further flare-ups of pain.

Using contemporary neurosciences for other forms of PSS

The PNE model can also be extrapolated to other persistent problems, such as recurring throat or bladder symptoms in the absence of infection or damage. The parallel is that just as there is a 'pain generator' within the CNS, there will also be a 'bladder control' generator, a 'hunger control' system, a 'fatigue system', and so on.

For example, if a patient has a throat infection, local inflammation will cause pain and swelling, not only to fight the bacteria but also to rid the body of the foreign organism. So just as pain is produced by the CNS in CRPS, a hypersensitive CNS may also learn to produce throat inflammation, even if there are very few or no bacteria present. This may explain why some patients have recurring sore throats without confirmed infection. Using PNE in this way for other symptoms is still an emerging concept which may lead to better management for many patients with these 'mysterious' symptoms. See case 4 overleaf.

Case 4: Mysterious urinary frequency and urgency

A 20 year old woman complained of sudden onset of urinary frequency, but without dysuria. She presented to a community pharmacy and was provided with antibiotics. As there was no improvement, she consulted her general practitioner, who provided a second course of antibiotics. A week later, a further GP gave her antispasmodics as her symptoms now included urinary urgency. Blood and urine tests were normal throughout (FBC, CRP, MSU, chlamydia and pregnancy tests, etc).

On her 4th consultation, she spoke of a previous urinary infection that included dysuria, and a gastrointestinal illness with lower abdominal pain. On that occassion, the consulting doctor said 'there was something wrong' in the pelvis, but then changed his mind, a comment that continued to worry her. Her brother had problems with urinary frequency and urgency in his early adolescence which remained undiagnosed and improved without specific treatment. She also recalled bouts of major fatigue as a child requiring time away from school.

In combination with the absence of clinical signs and normal investigations, the current and past history suggested some form of PSS, a diagnosis that was then offered to her to consider. The explanation was that her 'CNS bladder control centre' had somehow become sensitized as a result of incoming information and previous bodily experiences. She was reassured that she was 'normal' and that the symptoms would settle (which they did).

Summary of pain science and clinical approaches to chronic pain

To summarize the PNE section of Te Kete, contemporary neuroscience has changed our understanding of how the brain works and the sensation of pain. The main principles are:

1. Neuroplasticity enables flexibility and adaptation to demands of life and risk
2. Rather than arising solely from tissue damage or nociceptive input, pain is a centrally generated sensation from the CNS
3. Pain is always real, and is produced to protect us from potential harm
4. Understanding pain and the illness involves learning about the whole person (history and context); a biopsychosocial model is required
5. Clinical management of chronic pain now starts with patient education about the nature of pain, with encouragement to move and exercise
6. Recovery is possible, even from longstanding conditions.

The implications of these principles for professional practice are that:

- Clinical practice is only now just starting to use these insights to address the huge health and economic burden of chronic pain
- Most practicing doctors have not been taught modern pain science or attended communication workshops on how to explain pain

- Medical schools are only now just starting to teach this, so some cohorts of students will still be unaware of these advances
- This area of clinical practice has been difficult for everyone; doctors, patients and their whānau. The answer to such difficulties lies in better education for doctors and other health professionals who in turn, can help their patients to understand what is going on.

At present however, there appear to be three groups of practitioners. The first was trained many years ago and is unaware of the recent neurophysiological developments in pain science. The second group is aware of those concepts but is unsure how to translate them into clinical practice. The third group understands modern neuroscience and has been trained in relevant communication and consultation skills.

Around the world at the moment, medical schools are now gradually incorporating more lectures on the science of pain and more training of explanatory skills. We are hoping that by graduation, all medical students will now be joining this third group. It may take you some time and energy to learn such skills, but the rewards for you and your patients will be well worth it.

As shown in Figure 1, page 24, good explanations that are well accepted by patients can make a large difference in their subsequent illness trajectory and healthcare costs.

Further learning about PNE

Otago researchers have developed the Pain Library Guide for students and staff at Otago University: see otago.libguides.com/pain

Given that pain science and pain training is still at an early stage in medical education, postgraduate study in pain is also highly recommended. There are several high quality distance-taught papers on musculoskeletal conditions and pain from the University of Otago, Christchurch. These are multi-professional and endorsed by the International Association for the Study of Pain (IASP). See www.otago.ac.nz/christchurch/departments/department-of-orthopaedic-surgery-and-musculoskeletal-medicine/postgraduate

The NZ Pain Society holds an annual conference, which will be held in Dunedin in 2024. This conference includes an 'Open Day' where students are very welcome. It runs from Thursday 21st (the Open Day) to Sunday 24th March, 2024. Look for the reminder on Moodle in early March. See www.nzps2024.nz

Explanatory model 4: Functional Neurological Symptom Disorder (FND)

Functional neurological disorders are increasingly common in general practice and in neurology clinics (up to 30% of patients seen in OPD). These disorders include limb weakness or even 'paralysis' (as if the person has had a stroke), shaking or tremor, fixed muscle contraction (dystonia), tics, sensory and speech disturbances, and functional seizures (previously referred to 'psychogenic', 'dissociative' or 'non-epileptic' seizures). These problems can occur on their own or arise in conjunction with, or secondary to, other neurological disorders such as Parkinsons disease or other neurological disorders. Patients with epilepsy can also develop functional seizures.

Current best practice and treatment of FND is through providing a clear diagnosis which is based on the specific pattern of motor problems or seizures, and positive clinical signs such as Hoover's sign. Diagnosis is then followed by an understandable explanation (eg 'a software rather than hardware problem').

A positive diagnosis reduces patient anxiety and leads to more accurately focused treatment (eg FND-specific physiotherapy, patient education through on-line resources, focused CBT on personal beliefs, and clear arrangements for follow-up for further explanation and education. Australian researchers recently showed that if patients receive a 'satisfactory' explanation for their FND, their ensuing healthcare utilization and costs can reduce over the following two years, but continue or even increase after a 'poor' explanation.[1]

- "Functional neurological disorders (FND) are associated with considerable distress and disability. The symptoms are not faked.
- Diagnose FND positively on the basis of typical clinical features. It is not a diagnosis of exclusion.
- Psychological stressors are important risk factors, but are neither necessary nor sufficient for the diagnosis.
- FND can co-exist with other neurological disorders."[2]

1 Lagrand T et al. Health care utilization in functional neurologic disorders: impact of explaining the diagnosis of functional seizures on health care costs. Neurology: Clinical Practice. 2023;13(1).

2 Stone J, Burton C, Carson A. Recognising and explaining functional neurological disorder. BMJ. 2020;371(m3745).

Physiological mechanisms in FND

An emerging and potentially unifying theory for FND is known as the 'Predictive Coding Model of Perception.' Further to the physiology of pain as outlined in the PNE section, contemporary neuroscience has demonstrated that all sensations in the body are generated by the central nervous system (CNS) and subconscious parts of the brain. Incoming input to the CNS comes from both outside (ie hearing, vision, smell, taste and touch) and inside the body (nociceptors throughout the tissues). The CNS is monitoring literally thousands of incoming data points all the time in order to adapt to changes in the external environment and to maintain normal homeostasis.

The CNS makes many complex calculations between the incoming data compared to its prior experience and expectations, somewhat along the lines of Bayesian statistical analysis. If the CNS places more emphasis on its own expectations compared to incoming sensory data, physical sensations can arise which are inaccurate. A key point is that interoception (ie conscious awareness of sensations within the body) is not necessarily a 'true' reflection about what is going within body tissues.

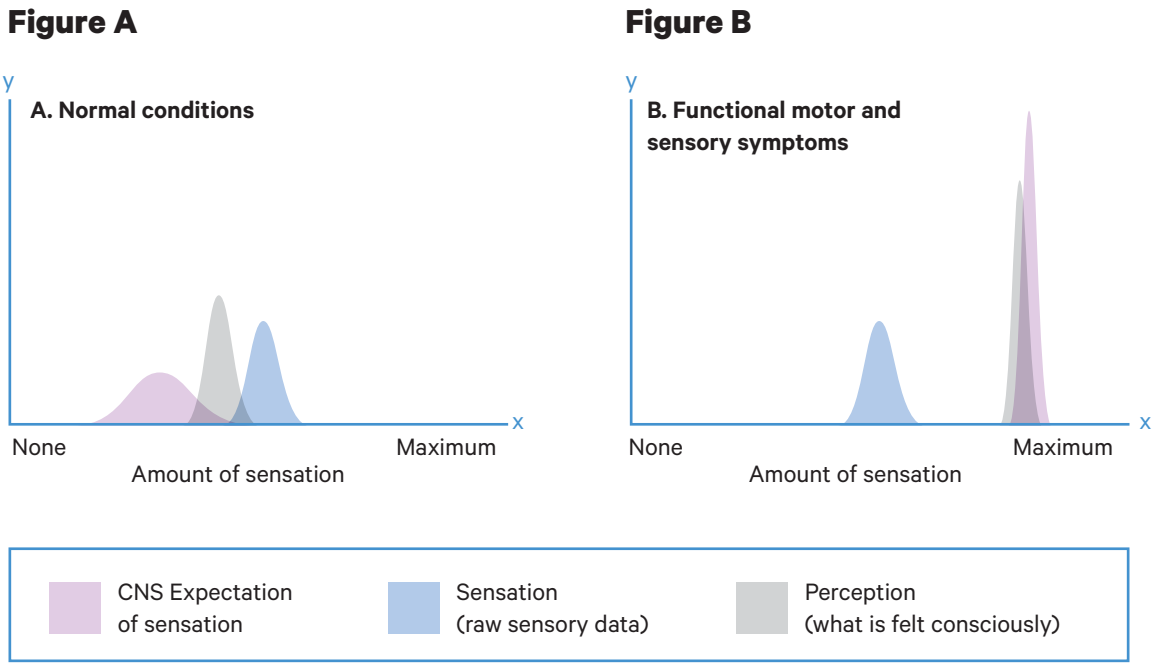
> Optical illusions are a good example of how the brain is always 'making a story' out of incoming data[2] (even when we 'know' how the illusion is being created).

Briefly then, symptoms in PSS can arise as a disorder of perception. Increased attention on the affected part of the body can also contribute to inaccurate sensation or motor control, where attention is influenced by recent 'evidence' of, or beliefs about, internal damage. See Figures A and B overleaf, which compares CNS processing in normal conditions and in functional neurological disorders (FND).

1 See Fobian AD, Elliott L. A review of functional neurological symptom disorder etiology and the integrated etiological summary model. J Psychiat Neurosc. 2019;44(1):8-18, or Van den Bergh O, et al. Symptoms and the body: taking the inferential leap. Neuroscience & Biobehavioral Reviews. 2017;74:185-203.

2 See for example; www.seevividly.com/blog/56/6_Cool_Optical_Illusions_That_Tell_You_About_How_Your_Brain_Works

Expectation, sensation and perception



The horizontal x-axis represents the range of probability of sensation from low to high, while the y-axis represents the relative magnitude or intensity. Starting with **Figure A,** the red curve is the existing or 'prior' expectation of sensation in the CNS/brain in relation to any particular stimulus. In normal conditions, the expectation is relatively weak and imprecise (ie there is not much prior expectation about an incoming stimulus). The blue curve is the incoming raw sensory data or unprocessed nociceptive input from tissues or senses.

Normally, the prior expectation does not dominate the sensory data and the person's perception (ie the grey curve, what is actually felt consciously) is somewhere between the two. For example, a patient's eventual perception of a new medication is equally modulated by the sensorial information and the expectation. This perception then becomes the new 'prior' for the next time the drug is taken.

Figure B attempts to represent these processes in functional neurological disorder (as an example of PSS). Here, the expectation in red of symptom recurrence is quite precise and high (ie a narrow curve on the x axis and high levels of magnitude on the y axis). This expectation overrides the incoming sensory data (blue) resulting in an eventual perception (grey) which is closer to the negative expectation. The 'prior' sensation in these situations may be influenced by beliefs such as 'my body is damaged or weak'.

Predictive coding processes illustrate basic tenets of contemporary neuroscience, that consciously felt sensations (ie perception) are created as the end-result of complex CNS calculations in order to adapt to the external environment.

Interestingly, there are now research links between three common clinical phenomena: persistent somatic symptoms such as functional neurological disorders; the well-recognised 'placebo response'; and its less well-known opposite, the nocebo response.[1]

In all RCTs for example, about 30% of patients report positive outcomes (placebo responses) to the inert agent used in the control arm. This is a phenomenon that has long been considered as 'normal', although not adequately explained within a biomedical framework. Non-medical examples of placebo effects are common throughout daily life, including getting drunk on placebo beer.[2] Consistent with predictive coding theory, the mechanism of action is the expectation of certain effects from the 'beer' and the influence of the social context.

Patients in the control arms of RCTS also report negative symptoms or nocebo responses. Recent examples of this occurred in 2022, when there was an increase in young patients presenting to ED in NZ with chest discomfort. This occurred just after the wide-spread publicity about a person who had died of myocarditis after their Covid Pfizer vaccination. However, very few of those patients had myocarditis or a rise in inflammatory markers.

In parallel in the UK, there was a sudden increase in patients presenting with headache after publicity about the risk of cerebral venous thrombosis related to the AstraZeneca ChAdOx1-S vaccine. These socio-medical phenomena could be considered as nocebo responses linked to high levels of societal fear during the pandemic, not only about the virus itself, but also about the vaccination programmes. Such widespread nocebo phenomena are more common than most people realise.

To summarise, predictive coding models of perception are now providing us with insights about the role of expectation and personal beliefs in how the CNS and brain function, especially how 'mind' and 'body' are artificial distinctions - instead, they are always working in unison.

> The physiology and the science are now 'in', so try not to use phrases to patients like *"We don't know what is going on here, but we know it is not serious."*

The nocebo hypothesis (NH) model for FND

The NH approach was developed by Dr Matt Richardson, a neuropsychologist at the Community Rehabilitation Centre, Wakari Hospital, Dunedin. Most people know about placebo responses, so the NH model firstly involves education about the mechanisms of both placebo and nocebo responses to various stimuli, then explaining and demonstrating the reversible neurophysiological mechanisms that underpin patients' symptoms.

This model builds on the idea that FND (and potentially other PSS as well) are triggered by abnormal beliefs about one's body. This is consistent with the observation that FND often occurs after another neurological diagnosis has been made, such as stroke or head injury. People believe their brain is damaged, so symptoms can emerge in a similar way to nocebo responses in relation

1 Fiorio et al. Functional neurological disorder and placebo and nocebo effects: shared mechanisms. Nature Reviews Neurology. 2022;18(10):624-35.
2 See www.youtube.com/watch?v=8EWqy6MGpxo

to perceived noxious events (eg medications, vaccinations, wind turbines and so on).

Explanation includes identifying those beliefs, then showing how they can be inaccurate. Hoover's sign, for example, demonstrates to the patient that they have normal muscle power of their leg, rather than their (damaged) brain is causing it to malfunction. In this way, the clinical examination can also be the start of treatment (see explanatory phrases overleaf).

For example, a patient with motor forms of FND can be shown how their walking on a treadmill can rapidly improve when they are listening intently to their favourite music (versus their usual attention on their 'weak' leg). Filming the improvement on the patient's cell phone can be used as a therapeutic challenge to their core beliefs and reinforces the idea that their symptoms are in fact reversible, which means that recovery is possible. Patient outcomes have been positive, consistent with other research based on similar explanatory models.[1] Emotion- or trauma-based components of CBT are not usually required, over and above the primary explanation.

Functional or 'dissociative' seizures: diagnosis and explanation

The incidence of patients with functional seizures appear to be increasing,[2] which may be due to the influences of social media where people film and post their own 'fits' or 'turns.' This sort of seizure can be differentiated quite readily from epileptic seizures on the following grounds:

> *The eyes may be tightly closed; the patient is crying or tearful; it lasts more than 5 minutes (best to time the event if possible); there is hyperventilation or side to side head shaking.*

None of these occur in an epileptic seizure which usually lasts less than a minute, is associated with loss of consciousness, incontinence, tonic-clonic limb shaking and post-ictal drowsiness. Sudden motionless unresponsiveness with eyes closed for more than 2 minutes is usually a functional seizure.

Family members may video the episode on their cell phone and bring it along. Most neurologists will now readily look at such a video which can help with diagnosis. Neurology referral is usually required to confirm and rule out other concurrent neurological disorders; the test of choice is simultaneous EEG monitoring and video recording which provides the definitive diagnosis.

It is preferable however, to start talking with the patient about the FND diagnosis and mechanisms while waiting for a referral to get through the system. The patient and their whanau require a clear and thorough explanation, as this helps to reduce anxiety and further ongoing health costs. Explanations can start on a positive note ('*You don't have a dangerous condition and it will usually get better once you understand what is going on'; 'it is not your fault*' etc, see below). Explore their ideas about their own body functioning and what their symptoms mean for them, including the patient's usual triggers to their seizures. Provide them with some resources to read, then see them again for further discussion.

Addressing stigma and shame

Historically, FND was incorrectly considered to be a psychological problem; patients with FND were diagnosed as having a 'conversion disorder' or even considered to be 'making up' their symptoms. These clinical responses to patients can add further distress – an iatrogenic contribution to patient suffering.

1 Nielsen et al. Randomised feasibility study of physiotherapy for patients with functional motor symptoms. J Neurol Neurosurg Psychiat. 2017;88(6):484-90.

2 Hull M, et al. Increased incidence of functional (psychogenic) movement disorders in children and adults amid the COVID-19 pandemic: a cross-sectional study. Neurol Clin Pract. 2021;11(5):e686-e90.

Fortunately, the conceptual advances in diagnosis and treatment outlined here are now helping to address the stigma and shame that have often accompanied these illnesses.

Just as with other PSS, previous or concurrent psychological stressors may act as triggers for the emerging symptoms of FND (see also the section on somatisation). However, not all patients with such stressors develop FND, and similarly, while treatment of psychological problems may be helpful, it may not resolve the symptoms. As Dr Jon Stone from Edinburgh suggests, while psychological problems or *'adverse [prior] events are more common in FND than in the general population... [they] are certainly not always present, and their presence is not useful diagnostically.'* This shift in concept is echoed in the revised diagnostic criteria for FND in the DSM-5 and ICD-11. It is important to be aware of the history of medical thinking and to interrogate the sources of those beliefs.

Useful phrases and communication skills

Here are some useful phrases, each of which has a specific purpose (adapted from Stone's benchmark article based on his own clinical experience[2]).

Useful phrases to practice and use	Purpose
"Would you like me to explain what I think is going on for you?	Ask permission to offer a more detailed explanation.
"You have functional weakness," and/or *"You have functional or dissociative seizures,"*	Explain what they *do* have.
For weakness: *"There is a problem with the way your brain is sending messages to your body—its a problem with the function of your nervous system, not its physical structure,"* For seizures: *"You are going into a trance-like state, it's a bit like someone being hypnotised."*	Emphasise the mechanism of the symptoms rather than their cause. Provide space and time to see how this diagnosis 'lands' with the patient, before moving on to more details.
Show the patient their Hoover's sign (see suggested wording below) or show a functional seizure video.	Explain how you made the diagnosis (ie pattern of symptoms and positive clinical signs).
"I do not think you are imagining/making up your symptoms/mad/going crazy/not coping."	Indicate that you believe them. Use words that match the patient's words and concerns.

1 See for example: MacDuffie KE et al. Stigma and functional neurological disorder: a research agenda targeting the clinical encounter. CNS Spectrums. 2021;26(6):587-92.
2 For more details, see Stone J. Functional neurological disorders: the neurological assessment as treatment. Practical Neurology. 2016;16(1):7-17.

"I see lots of patients with similar symptoms."	Emphasise that this is common
"Because there is no damage, you have the potential to get better."	Emphasise reversibility and a positive outlook.
"You don't have multiple sclerosis or epilepsy", etc	Explain what they *do not* have.
"This is not your fault, and there are things you can do to help it get better."	Emphasise that self-help is a key part of getting better.
"If you have been feeling stressed/low/worried, that will tend to make the symptoms even worse. Note however that that is not the cause of your problem."	Brings in the role of psychological factors without suggesting they are the cause of the symptoms.
Give them some written information; eg your own notes and diagrams and/or direct them to www.neurosymptom.org, or www.nonepilepticattacks.info	Use written information.
"I'll like to see you again. Please look at the information I have given you and come back with questions."	Commit to engagement and follow up.
"Other clinics have a lot of experience and interest in helping patients with functional movement disorder—they won't think you are 'crazy' either."	Consider a physiotherapy or psychological referral (preferably at a second visit), but only to health professionals with FND expertise.

Explanatory phrases	**Comments**
"Would you like some more detail on all this? This might take some time, so we could do that now or make another time."	Addresses the time constraints in the usual structure of medical practice. Is respectful.
"The problem is mostly about how the unconscious part of your brain and spinal cord is processing information and how it creates sensation and movement."	Introduces concept of how the CNS creates sensation and controls movement.
"Your CNS is sort of miscalculating the control of your movements at the moment."	This is a 'non-blaming' explanation, based on body physiology (compare the usual explanation for diabetes ie 'there are low levels of insulin').
"When you are not concentrating on your leg, often your movements will get better." *"It's a bit like trying to swallow a large tablet; the more you focus on that, the harder it gets, even though you can swallow food quite readily at dinner."*	Distraction techniques can be taught and learned.

Explaining Hoover's sign

Hoover's sign needs to be practiced on friends and colleagues until it becomes a normal part of your clinical examination for any patients with neurological symptoms. In weakness from FND, Hoover's is usually positive and provides a useful entry point into explaining what is going on. The following notes are adapted from Stone ('Neurological assessment as treatment', BMJ 2016). The patient is sitting in a chair; the doctor's hand is under the middle of the patient's thigh.

Doctor (testing weak extension of the left hip): *Try to keep your foot flat on the floor for me while I pull up on your leg.*

Patient (in a sitting position): I can't do it (ie left leg comes up off the floor).

Doctor (keeps hand under left thigh, while testing right hip flexion against resistance): *Now concentrate on lifting up your good right leg. Look at that right leg and focus on keeping it up in the air. [Pause] Now, can you feel that when you do that, the power in your left leg has come back to normal? I can't get that left foot off the floor now.*

Patient (and their partner): Wow, that's weird.

Doctor: This test is called Hoover's sign. It's a positive sign of a genuine problem called functional leg weakness. When you were just focusing on trying to keep your left foot on the floor, your left leg was weak. But the power comes back to normal when you focus on and move your other leg. This shows me that the weakness can't be due to damage anywhere in the nervous system.

Patient: (thinks about it a bit) Ok, so what's going on then?

Doctor: Your brain is having trouble sending a message to your leg to make it move, but when you are distracted by something else, the automatic movements can take place normally. This test shows me that there is a problem with how your nervous system is working, but not because there is any damage to it. It's basically a problem with the function of the nervous system—a bit like a software problem instead of a hardware problem. Shall I show you again? (Adapted from Stone).

This demonstration can be the start of further discussion.

Nocebo explanations	Comments
"Do you know about placebo effects? There is a quite useful explanation of your symptoms based on the cousin of placebo, which is called the nocebo effect."	Introduces nocebo hypothesis as another possible explanation.
"Nocebo responses can occur in relation to a negative belief about something; an example might be expecting more pain at the dentist, which often comes true."	Use several examples.
"Functional symptoms are similar, especially if you already feel there is something wrong with you."	Task is to elicit beliefs that might be relevant.
For seizures, enquire about the context in which they occur, which can help to identify triggers.	The fight-flight-freeze metaphor (see the SNS section) might be relevant for functional or 'dissociative' seizures .

Space for notes and phrases

Unhelpful phrases and approaches

In contrast to the above, here is a list of Don'ts; ie phrases that tend to be counter-productive for the patient in their journey towards understanding.

Unhelpful approaches and phrases	Possible outcomes
Making no diagnosis at all, or saying there is no 'neurological disease' found. This includes using the term 'non-organic'.	The patient is likely to go elsewhere to seek a diagnosis.
Suggesting the patient has an 'unexplained' diagnosis; ie using phrases like "These things are common in medicine and we don't really know why they happen".	As above, the patient is likely to go elsewhere to see what is going on. (We need to be familiar with functional disorders and to be able to make a positive clinical diagnosis alongside other positive diagnoses.)
Trying to explain that the problem is 'psychological' or explaining that these symptoms are 'stress-related'.	Likely to be rejected by most (80%) of patients. Often equated by patients as an accusation that the symptoms are 'made up' or 'imagined' or that they are not coping. Many patients with these symptoms do not have identifiable stress or psychiatric disorder.

Being realistic

With knowledge and skill development as above, many of these patients can be helped quite readily. As in other branches of medicine however, there will some patients for whom best treatment is still ineffective. Risk factors for not responding well to modern FND approaches are not being able to recall any of the ideas from the first consultation with you, having very fixed views about the cause of the problems, being involved in a legal dispute of some kind, or having very long standing or physically disabling symptoms.

There are parallels here for some patients with chronic nociplastic pain who do not improve after good PNE. However, unless you offer these modern treatments, you will not know how the patient will fare. Being committed to the possibility of recovery is perhaps the best clinical approach, as for all our patients.

Summary of explanations for FND

To summarise the modern explanation-based approach to FND, the following steps are required:

1. Inform the patient that their symptoms are genuine, common and potentially reversible
2. The diagnosis has been made on positive grounds (ie is not a diagnosis of exclusion)
3. Identify and address any beliefs about a 'damaged' body or similar
4. Offer a mechanism-based explanation involving neuroscience, predictive processing or nocebo-hypothesis (NH)
5. Provide straightforward advice about distraction techniques, self-help and further reading or websites
6. Organise further review to check understanding and progress so far
7. As required, refer for FND-specific physiotherapy and/or FND-specific psychological services.

Recommended FND reading and resources

- Stone J. Functional neurological disorders: the neurological assessment as treatment. Practical Neurology. 2016;16(1):7-17. The notes above are based on this article.
- Functional Neurological Disorder (FND): a patient's guide: www.neurosymptoms.org This is a very useful website for patients to explore and then come back and discuss.
- Espay, Aybek et al. Current concepts in diagnosis and treatment of functional neurological disorders. JAMA Neurol. Doi: 10.1001/jamaneurol.2018.1264 More details about diagnosis and treatment.
- Richardson M, Isbister G, Nicholson B. A novel treatment protocol (nocebo hypothesis cognitive behavioural therapy; NH-CBT) for functional neurological symptom disorder/ conversion disorder: a retrospective consecutive case series. Behav Cognit Pychotherapy. 2018;46(4):497-503.

thebmj Visual summary

Recognising functional neurological disorder

Looking for positive diagnostic signs in primary care

Functional neurological disorder (FND) describes a disorder of the voluntary motor or sensory system, which has been linked to corruption of pre-conscious phases of motor planning. It should usually be diagnosed by someone with specific expertise in the diagnosis of neurological disease, but it can be useful to recognise the signs and symptoms in primary care for appropriate and timely specialist referral

Person with motor or sensory symptoms such as:

- Paralysis
- Tremor
- Dystonia
- Speech symptoms
- Seizures
- Sensory disturbance — Including visual loss

Patient history

Symptoms

Patients with FND often have multiple symptoms. Ask about motor and sensory symptoms, fatigue, pain, sleep disturbance, memory, and dissociative symptoms

Ability

Ask patients to describe a typical day, to build a picture of how disabled they are. This can also help determine whether there may be comorbid depression or anxiety

Onset

Look particularly for physical triggering such as injury, migraine or syncope. Previous adverse experiences are a risk factor, but may not be present

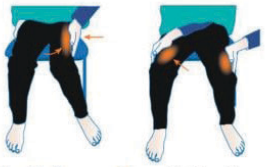
Examination

Functional neurological disorder is not a diagnosis of exclusion. It should be based on positive clinical features which demonstrate inconsistency between impaired voluntary movement and intact automatic movement, or in some cases, incongruency with pathophysiological disease

Positive diagnostic signs

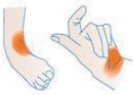
Functional limb weakness

Hoover's sign: Hip extension weakness that improves with contralateral hip flexion against resistance



Hip Abductor Sign: Abduction weakness that improves with contralateral hip abduction against resistance

Functional movement disorders



Functional dystonia typically presents with a fixed position, usually a clenched fist or inverted ankle

Functional facial dystonia presents with episodic contraction of platysma or orbicularis oculi

Left wrist tremor stops or entrains when copying examiner's movements with right hand

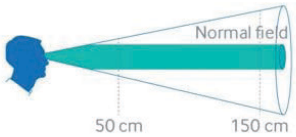
Functional or dissociative seizures

Should be diagnosed on the basis of finding characteristic features in the subjective account and observed description of the attacks, such as:

- Eyes tightly closed
- Tearfulness
- Longer than 5 minutes
- Side to side head shaking
- Hyperventilating

Ask patient if they would be willing to have their attacks video recorded by a family member

Functional visual signs



A tubular visual field defect at 50 cm which is the same width as at 150cm

Visual field spiralling caused by progressive constriction of vision during the test

Investigation and referral

While it may be reasonable for the diagnosis to be raised as a possibility with the patient in primary care, it is recommended to refer patients with a suspected diagnosis of FND to secondary care for specialist assessment

Common pitfalls

Common reasons for missing the diagnosis of FND include:

- An absence of psychological comorbidity
- No prior functional disorders
- Patients that dont fit a false stereotypical profile—such as male, older, working

Common reasons for making a wrong diagnosis of FND include:

- Placing too much emphasis on psychological comorbidity
- Relying on single signs rather than combinations
- Relying on normal tests
- Unusual or unexpected symptoms, but no positive diagnostic signs of FND

thebmj Read the full article online http://bit.ly/BMJfnd

See more visual summaries http://www.bmj.com/infographics

Explanatory model 5: Creating inspiration for lifestyle change

The six pillars of lifestyle medicine are 1) A whole-food, plant-predominant eating pattern, 2) Physical activity, 3) Restorative sleep, 4) Stress management, 5) Avoidance of risky substances, and 6) Positive social connections. As lifestyle factors are estimated to contribute to 80% of non-communicable disease, almost all patients can benefit through directly addressing these pillars of lifestyle.

The benefits for patients of addressing life-style factors are backed by substantial evidence. Current research is also exploring various methods of supporting patients in their lifestyle change. Briefly, patients need sufficient knowledge in order to consider change; they must also be enabled to make change in their own time and in their own way.

> Lifestyle medicine is based on matauranga me whakaute: providing patients with knowledge and respecting their choices.

Definition

> *"Lifestyle medicine is a medical specialty that uses therapeutic lifestyle interventions as a primary modality to treat chronic conditions..... Lifestyle Medicine Certified Clinicians are trained to apply evidence-based, whole-person, prescriptive lifestyle change to treat and, when used intensively, often reverse such conditions. The six pillars of lifestyle medicine are a whole-food, plant-predominant eating pattern, physical activity, restorative sleep, stress management, avoidance of risky substances and positive social connections."*
> (International Board of Lifestyle Medicine)

Ways of life and living habits that are prevalent in the Western world can impact negatively on all these domains. Less-than-healthy habits have been shown to induce epigenetic changes which

Definition

> *"Lifestyle medicine is a medical specialty that uses therapeutic lifestyle interventions as a primary modality to treat chronic conditions..... Lifestyle Medicine Certified Clinicians are trained to apply evidence-based, whole-person, prescriptive lifestyle change to treat and, when used intensively, often reverse such conditions. The six pillars of lifestyle medicine are a whole-food, plant-predominant eating pattern, physical activity, restorative sleep, stress management, avoidance of risky substances and positive social connections."*
> (International Board of Lifestyle Medicine)

Ways of life and living habits that are prevalent in the Western world can impact negatively on all these domains. Less-than-healthy habits have been shown to induce epigenetic changes which contribute to poor health as well as directly increasing the level of chronic inflammation in the body. Termed 'metaflammation', this ubiquitous phenomenon underlies much of chronic disease. Positive changes in any of the lifestyle pillars will result in healthy changes to metaflammation. At sufficient levels, these changes have the power to moderate, reverse and even 'cure' disease.

In the traditional medical model, patients are considered to be dependent on the advice of their doctors. With some allowance for personal preferences, they tend towards being 'passive recipients' of health care interventions. However, the lifestyle model of healthcare includes important conceptual differences. Firstly, it is *trans-diagnostic,* meaning that a given lifestyle change can have a positive impact on health regardless of the diagnosis.

Secondly in the lifestyle model, the *patient* is in charge of the decision-making, and is encouraged to take more active control of their choices that contribute to health. Failure to appreciate this difference is one of the reasons why many doctors struggle when they discuss lifestyle change with their patients. One tendency is to 'tell' patients what to do, rather than inspiring patients

1. Firstly, connect (whakawhānaungatanga) with the patient and help the patient connect with their own self. A simple question to someone with longstanding IBS, chronic fatigue or chronic pelvic pain might be *"What's it been like for you coping with this over the years?"* This curious but effective question can help the patient feel connected to you and put them in touch with what they've probably tried to suppress while dealing with their problem. It will often be the first time someone has been asked this of them and it can be quite an emotional and cathartic moment.

2. Connect the patient with future possibilities. A follow up question such as *"What would you be able to do that you want to do if you didn't have this problem",* connects a patient with a positive future which if their illness has been around for a while, they have often given up hope on. Pay close attention to this answer because within it lie the incentives and inspiration for change.

3. Let the patient know what's possible for them. *"Are you interested in making changes to your lifestyle which might cure you and which will certainly result in better health with an aim of being able to do...?"* (insert whatever it was that patient valued being able to do). This will usually be the first time a patient has heard that it is possible to achieve a reduction and even possibly a cure, simply through making changes themselves. This forms the basis of the 'inspiration' necessary to make significant enough changes to enact a quick and noticeable difference. This positive change is important to provide feedback which reinforces motivation to maintain the new habits. Frequently, the lifestyle changes recommended to patients are underdosed. They have minimal effect which is meaningful to the patient and how they feel and result in loss of motivation.

to make self-directed changes. Telling patients what to do has been consistently shown to have a low success rate. It can work but it does not work well. Doctors have also tended to motivate patients through fear i.e. "If you want to avoid these horrible complications of diabetes you had better....." The use of fear as a motivator has also been shown to have a low success rate.

When a person makes a lifestyle change, they are attempting to adjust ingrained habits. This can be hard to do because we do most things on automatic. This means willpower is not the answer. Willpower requires conscious effort and at some point, when a person relaxes, they will find themselves back in habitual actions.

There is no 'one way' to help a patient make change but the following steps are helpful.

There are some important points to bear in mind about lifestyle change:

- The more unwell the patient is, the greater the effect of any lifestyle change. People with serious problems benefit more than those with only minor problems. A person with higher blood pressure can gain a greater response than a person with blood pressure closer to the norm.
- The size or extent of lifestyle change is proportionate to the size of benefit. (Many patients who make minor change see only minimal if any benefits to their life and thus lose motivation). Thus it is better to start with a pillar which is amenable to major change quickly and one which will result in a noticeable benefit.
- Empower the patient through your inquiry so that they are in control. The patient chooses the level of change they wish to make, and they determine the 'pillar' they wish to tackle first.
- Once this decision is made, our role is then to support the patient making that change. We encourage the patient to set a date by which they'll make a specific change, we inquire into what they will need to do in preparation for the change and also help them derive solutions for potential obstacles or challenges they face.
- We agree on a review date that suits the patient.

The more the patient feels in charge of the process, the more likely they are to succeed. The implications of this are that we do not tell the patient what they should do and that we accept whatever decision they make whether this in keeping with our own views or not.

This process creates challenges for us as health professionals especially when patients do not appear to be 'co-operating' with their care. It is useful under these circumstances to hold certain presuppositions in mind which act as a brake on our own reactions. Examples include:

- At *any* point in time, everyone is doing the best they can with the (personal) resources available to them.
- All human beings are capable given the motivation and the resources.

These suppositions help us maintain our belief in the patient. Very often a patient can lose belief in themselves, and part of the therapeutic process involves restoring this belief.

Summary of steps for health professionals working with patients for lifestyle change:

1. Adopt helpful presuppositions prior to seeing patients.
2. Connect with the patient and help them to articulate their distress

3. Help the patient create a meaningful future possibility for themselves which will be realised as a result of making change.
4. Inspire change: what might be possible in their health, should the patient make a sufficient enough change?
5. Inquire into how and when would they like to start.
6. Explore what things might make their transition more difficult? Inquire with the patient how to mitigate these. (Patient is to come up with solutions which work for them)
7. Set a review date. When do they think it would be useful for them to be reviewed?

Useful lifestyle resources

IBLM is the major certifying body for health professionals who wish to become a Lifestyle Medicine Certified Clinician. International Board of Lifestyle Medicine: iblm.co

The following websites provide access to patient and clinician resources to support lifestyle change:

- American College of Lifestyle Medicine. The founding body of the discipline. It has supported the formation of similar bodies in other countries and also the International board of lifestyle medicine which oversees an internationally recognised qualification. lifestylemedicine.org
- British society of Lifestyle Medicine A national lifestyle organisation. bslm.org.uk
- Australasian Society of Lifestyle Medicine Regional organisation lifestylemedicine.org.au
- Doctors For Nutrition (DFN) A regional body set up to promote and support the use of whole food plant based predominant nutrition as a therapeutic and preventative option for chronic disease. www.doctorsfornutrition.org
- Plant Based Health Professionals UK. A similar organisation to DFN. plantbasedhealthprofessionals.com

Learning courses for health professionals

You will find access to short online courses on most of the above websites. For example, DFN have an online course to help medical students and clinicians discuss whole food plant-based nutrition (WFPB) with patients.[1] The following courses offer online certification in plant-based nutrition. These courses are open to all applicants and provide up to date knowledge on WFPB and associated health improvements. The Winchester course provides a more academic focus

- Winchester University UK www.winchester.ac.uk/study/further-study-options/short-courses/plant-based-nutrition
- Cornell University USA ecornell.cornell.edu/certificates/nutrition/plant-based-nutrition
- Guelph University Canada This course has only recently been established. courses.opened.uoguelph.ca/public/category/courseCategoryCertificateProfile.do?method=load&certificateId=29839235

1 Martyn Williamson (one of Te Kete authors) has contributed to this course.

Further reading

- Christ A, Latz E. *The Western lifestyle has lasting effects on metaflammation*. Nature Reviews Immunology. 2019;19(5):267-8.
- Hayes C, Naylor R, Egger G. *Understanding chronic pain in a lifestyle context: The emergence of a whole-person approach.* American Journal of Lifestyle Medicine. 2012;6(5):421-8.
- Egger G. *In search of a germ theory equivalent for chronic disease*. Prev Chronic Dis. 2012;9:110301.

Appendix: Diagnostic criteria in common persistent syndromes

There are now many well-developed diagnostic criteria for persistent somatic symptoms, bodily stress syndrome, or functional illness. These criteria can help doctors make a firm 'positive' diagnosis. Each of the syndromes listed below are followed by some suggested explanations, based on the 4 models described earlier.

While such syndromes are now well described, many other patients have unique symptoms or only partially fulfil the criteria. Clues that symptoms may be related to neurophysiological dysfunction (rather than structural damage) of body organs or regions are the inconsistency between symptoms and peripheral nerve innervation and/or local autonomic reactions (as for example in CRPS), the temporal relationship with life-stressors (exacerbations and improvements), and the absence of 'red flags' (symptoms that warrant further specific investigations such as melaena or night pain). In these situations, a diagnosis by exclusion is also helpful.

With experience however, these patient presentations often have recognisable patterns. Migraine headache is included below as an example of how to use diagnostic criteria. This syndrome is no longer considered to be 'medically unexplained', as the physiological process of cerebral arteries contracting (causing various aura) and opening (causing headache) are well theorised. However, it is a useful model of symptoms where there are no structural or organic underlying problems; instead, it is an example of complex neurophysiology in response to a range of initial triggers such as flashing lights, fatigue, foods and/or stress.

The following pages contain current diagnostic criteria for:

Nociplastic pain

Clinical criteria are listed below. Nociplastic pain underpins many of the PSS syndromes. As noted earlier, recognising features of nociplastic pain can be crucial within the differential diagnosis.

1. The pain is:

1a. Chronic (3 months +)
1b. Regional (rather than discrete) in distribution*;
1c. There is no evidence that nociceptive pain (a) is present or (b) if present, is entirely responsible for the pain
1d. There is no evidence that neuropathic pain (a) is present or (b) if present, is entirely responsible for the pain.

2. History. There is a history of pain hypersensitivity in the region of pain. Any one of the following:

2a. Sensitivity to touch
2b. Sensitivity to pressure
2c. Sensitivity to movement
2d. Sensitivity to heat or cold.

3. Presence of comorbidities. Any one of the following:

3a. Increased sensitivity to sound and/or light and/or odors (ie "cranial nerve central sensitisation"; ie Cr nerve V111 auditory, light Optic (11) nerve, odours (smell and taste (V and V11)
3b. Sleep disturbance with frequent nocturnal awakenings.
3c. Fatigue
3d. Cognitive problems such as difficulty to focus attention, memory disturbances, etc.

4. Examination. Evoked pain hypersensitivity phenomena can be elicited clinically in the region of pain. Any one of the following:

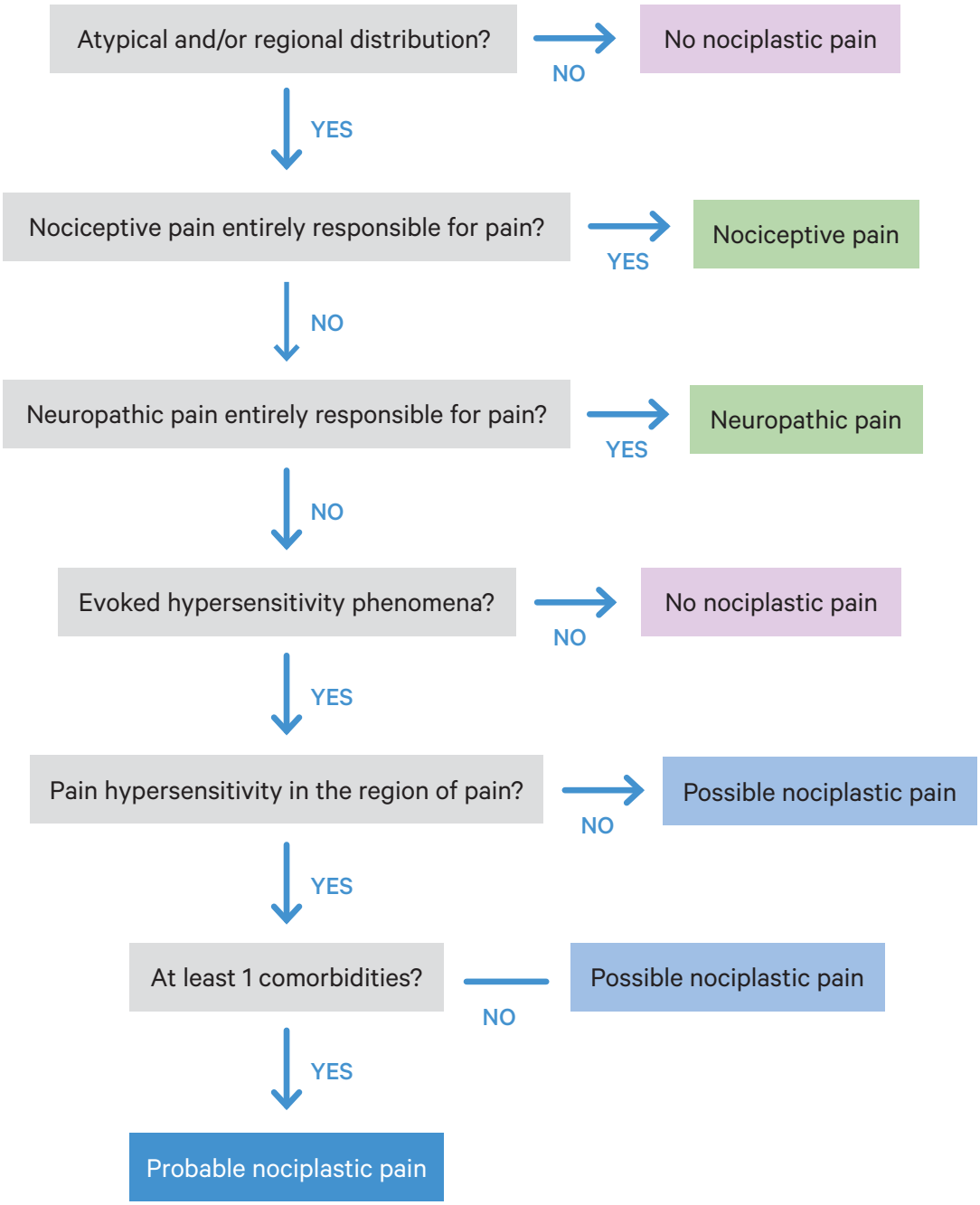
4a. Static mechanical allodynia
4b. Dynamic mechanical allodynia
4c. Heat or cold allodynia
4d. Painful after-sensations reported following the assessment of the above.

Grading of nociplastic pain:

Possible nociplastic pain: 1 and 4.
Probable nociplastic pain: All the above: 1, 2, 3, and 4.

See decision tree overleaf.

Figure 3. Clinical decision-making tree of the clinical criteria for nociplastic pain[1]



1 From Kosek E, Clauw D, Nijs J, et al. *Chronic nociplastic pain affecting the musculoskeletal system: Clinical criteria and grading system.* Pain. 2021;162(11):2629-34.

Irritable Bowel Syndrome (IBS): Rome Criteria

IBS and chronic abdominal pains comprise a significant proportion of patients at Gastroenterology Outpatients. In private specialist gastroenterology practice, these patients may be 60-75% of patients seen.

The patient must have 2 or more of the following symptoms associated with recurrent abdominal pain >1 day/week in the last 3 months (on average):

- Related to defecation (either increasing or improving pain)
- Associated with a change in stool frequency
- Associated with a change in stool form (appearance)

If the diagnostic result is negative – unlikely to be IBS.

Subtypes

1. IBS with constipation—hard or lumpy stools more than 25% of the time and loose or watery stools less than 25% of bowel movements
2. IBS with diarrhoea—loose or watery stools more than 25% and hard or lumpy stools less than 25% of bowel movements
3. Mixed IBS.

Suggested explanations

Abdominal pain in IBS is usually non-specific or general, rather than specific local tenderness as in appendicitis. Pain is probably due to higher pressure in the bowel wall as part of increased peristalsis. Note that before a big match some of the All Blacks will spend extra time on the toilet; getting the 'runs' or 'nervous diarrhoea' is very common.

The explanation and treatment for IBS can involve both dietary changes (usually more roughage, less highly processed food and sugar, add in bulking agents such as Metamucil), as well as being more aware of common triggers to symptoms such as unresolved work or family tensions. IBS lends itself well to a somatisation model of explanation including normalisation (eg 'it is normal for bowels to either slow up or speed up in response to life pressures; this is part of the sympathetic nervous system flight-fight-freeze that helps to keep us safe'), non-blaming comments ('people who cope well with lots on their plate can often get this') and/or metaphorical links between mind and body ('sounds like your boss is a pain in the stomach').

A neuroscience explanation may involve talking about the 'stomach and bowel control centre' in the CNS/brain that monitors and controls bowel absorption and transit time (speeding up in those patients with loose bowel motions and mucous, and slowing down for constipation etc). The factors that influence digestion will be similar to those factors listed in the PNE section, so similar diagrams could be used depending on the patient's story and belief systems. See also Case 1 on page 19 for an example.

Fibromyalgia

Diagnostic criteria:

A patient satisfies diagnostic criteria for fibromyalgia if the following 3 conditions are met:

1. Widespread pain index (WPI) 7 and symptom severity (SS) scale score 5 or WPI 3–6 and SS scale score 9.
2. Symptoms have been present at a similar level for at least 3 months.
3. The patient does not have a disorder that would otherwise explain the pain.

WPI is the number areas in which the patient has had pain over the last week, such as shoulder, arm, hip, leg etc. Score will be between 0 and 19. SS scale score includes fatigue, waking unrefreshed, and cognitive symptoms.

For more details see: Wolfe F, Clauw DJ, Fitzcharles MA, Goldenberg DL, Katz RS, Mease P, et al. The American College of Rheumatology preliminary diagnostic criteria for fibromyalgia and measurement of symptom severity. Arth Care Research 2010;62(5):600-10.

Suggested explanations

By the time the diagnosis of fibromyalgia is made, it is likely that central sensitisation and nociplastic pain pathways will be well established. By then, patients may believe there is no explanation and therefore no chance of recovery. Hearing about their illness experience and validating their pain is an important first step; ie that their pain is real, that they are not making it up or have a mental disorder. Once some trust is established, it may be possible to offer various explanations, noting that re-conceptualisation of their ideas about pain may be difficult for many people. Incorporating PNE into a motivational interviewing framework can be helpful, starting at the baseline of the patient's knowledge and understanding about their symptoms (see Nijs J et al. Integrating Motivational interviewing in pain neuroscience education for people with chronic pain: a practical guide for clinicians. Physical Therapy. 2020;100(5):846-59).

Tension headache

Headaches are extremely common. Non-pulsating headaches that are not aggravated by routine physical activity may help screen to patients for tension headache. Key features on history include:

- Bilateral location
- Non-pulsating quality
- Mild or moderate intensity
- Not aggravated by routine physical activity
- Mild nausea
- Photophobia (light sensitivity)
- Phonophobia (sound sensitivity)

On exam: *"Pericranial tenderness is easily detected and recorded by manual palpation. Small rotating movements with the index and middle fingers, and firm pressure (preferably aided by use of a palpometer), provide local tenderness scores of 0-3 for frontal, temporal, masseter, pterygoid, sternocleidomastoid, splenius and trapezius muscles. These can be summed to yield a total tenderness score for each patient. These measures are a useful guide for treatment, and add value and credibility to explanations given to the patient".*

Suggested explanations

Headaches occurring during the week versus weekend and/or holidays often establish links to intercurrent pressures. A useful explanation is the SNS fight-flight-freeze response, which with ongoing tensions, pressures and issues can remain in the 'on' position. This means that muscles remain slightly tense and ready for action; over time, this leads to trigger points and pain within the muscle. If patients can recognise the links and understand the underlying physiological basis, they can then actively try to reduce the SNS firing and increase their parasympathetic system (PSNS) activation. All the usual lifestyle recommendations such as taking breaks, relaxation, mindfulness training, swimming, exercise, yoga etc, will help the PSNS.

See more details at ichd-3.org/2-tension-type-headache

Migraine headache

(ICHD-3 Diagnostic criteria for migraine)

1.1. Migraine without aura

Recurrent headache disorder manifesting in attacks lasting 4-72 hours. Typical characteristics of the headache are unilateral location, pulsating quality, moderate or severe intensity, aggravation by routine physical activity and association with nausea and/or photophobia and phonophobia.

Diagnostic criteria:

A. At least five attacks fulfilling criteria B-D below
B. Headache attacks lasting 4-72 hr (untreated or unsuccessfully treated)2;3
C. Headache has at least two of the following four characteristics:
    1. unilateral location
    2. pulsating quality
    3. moderate or severe pain intensity
    4. aggravation by or causing avoidance of routine physical activity (eg, walking or climbing stairs)
D. During headache at least one of the following:
    1. nausea and/or vomiting
    2. photophobia and phonophobia
E. Not better accounted for by another ICHD-3 diagnosis.

Notes:

1. One or a few migraine attacks may be difficult to distinguish from symptomatic migraine-like attacks. Furthermore, the nature of a single or a few attacks may be difficult to understand. Therefore, at least five attacks are required. Individuals who otherwise meet criteria for 1.1 Migraine without aura but have had fewer than five attacks should be coded 1.5.1 Probable migraine without aura.
2. When the patient falls asleep during migraine and wakes up without it, duration of the attack is reckoned until the time of awakening.
3. In children and adolescents (aged under 18 years), attacks may last 2-72 hours (the evidence for untreated durations of less than two hours in children has not been substantiated).

1.2.1 Migraine with typical aura

Migraine with aura in which aura consists of visual and/or sensory and/or speech/language symptoms, but no motor weakness, and is characterized by gradual development, duration of each symptom no longer than one hour, a mix of positive and negative features and complete reversibility.

Diagnostic criteria:

A. Attacks fulfilling criteria for 1.2 Migraine with aura and criterion B below
B. Aura with both of the following:
    1. fully reversible visual, sensory and/or speech/language symptoms
    2. no motor, brainstem or retinal symptoms.

Migraine headache (continued)

Suggested explanations

Although the physiological pathways for migraine headache may involve more severe pain, photophobia and vomiting (which may need specific drug treatment), a similar approach to explanation for tension headaches can also be used to reduce the number of attacks. A pain neuroscience explanation can also be adapted quite readily for severe headache (see Minen MT et al. Neuroscience education as therapy for migraine and overlapping pain conditions: a scoping review. Pain Medicine. 2021;22(10):2366-83).

Non-Cardiac Chest Pain (NCCP)

Some patients who present to their GP or to ED with chest discomfort and/or pain may have coronary artery disease (CAD) causing angina or a myocardial infarction. It is important that CAD is considered, so in most EDs, the protocols correctly include ECGs, cardiac enzymes, CRP and so on.

However, many patients do not have CAD. Once this has been established through negative tests, they are usually labelled as 'Chest-pain not otherwise specified', reassured it is not their heart, and then discharged. This necessarily pragmatic approach rules out potential life-threatening illness, but it does not make a positive diagnosis of PSS or bodily stress, if that is the underlying problem. One outcome is that these patients are no better off in terms of understanding what caused their pain. They may also worry the doctors have missed something important. It is important then to identify patients with underlying anxiety and/or bouts of acute panic. See the table below.

Acute cardiac symptoms	Acute anxiety and/or panic attack symptoms
Age: ≥ 45 yrs (men), ≥ 55 yrs (women)	Age: peak onset late 20's
Palpitations, rapid, slow or irregular heart beat Chest pain, tightness, crushing or radiating Sweating Short of breath Cold peripheries Tingling, numbness Nausea, vomiting Faint/dizzy Anxiety	More than one presentation of chest pain not related to exertion Atypical pains for heart problems, body-wide symptoms, more distress present, pain is not the major feature Palpitations, pounding heart, or accelerated heart rate Sweating, trembling or shaking Shortness of breath, smothering, feelings of choking, fear of dying Chest pain or discomfort Nausea or abdominal distress Dizzy, light-headed, or faint Chills or heat sensations Numbness or tingling sensations De-realization or depersonalization Fear of losing control or "going crazy"

Suggested explanations

Reassurance that the pains are not from their heart is the first step, but further explanations are usually required if the patient is to manage further bouts. Chest pain of non-cardiac origin is often attributed to muscle tension, intercostal joint tenderness, or thoracic spine dysfunction. Sometimes clinicians such as physiotherapists, osteopaths or chiropractors who are trained in muscle and joint problems can be very helpful.

At other times, non-cardiac chest pain seems to be associated with anxiety or tension as outlined above. An initial explanation then would use a Sympathetic Nervous System (SNS) model, where

muscle tension, increased heart rate and other symptoms are part of the fight-flight-freeze response to conscious or unconsciously perceived danger of some kind. Taking a very careful history may identify relevant triggers to the bouts of pain. This can lead to further discussion about how those triggers can create low grade anxiety or tension, then finding techniques to calm the SNS down.

Some patients are less aware of their underlying feelings, so a pain neuroscience model might be useful where ‘chest pain’ is created by the CNS, even if that pain seems to be less than adaptive at the time.

Complex Regional Pain Syndrome (CRPS)

IASP-proposed revised CRPS clinical diagnostic criteria.[13]

A clinical diagnosis of CRPS can be made when the following criteria are met:

- Continuing pain that is disproportionate to any inciting event.
- At least 1 symptom reported in at least 3 of the following categories:
  - Sensory: Hyperesthesia or allodynia
  - Vasomotor: Temperature asymmetry, skin colour changes, skin colour asymmetry
  - Sudomotor/edema: Edema, sweating changes, or sweating asymmetry
- Motor/trophic: Decreased range of motion, motor dysfunction (eg, weakness, tremor, dystonia), or trophic changes (eg, hair, nail, skin).
- At least 1 sign at time of evaluation in at least 2 of the following categories:
  - Sensory: Evidence of hyperalgesia (to pinprick), allodynia (to light touch, temperature sensation, deep somatic pressure, or joint movement)
  - Vasomotor: Evidence of temperature asymmetry (>1°C), skin color changes or asymmetry
  - Sudomotor/edema: Evidence of edema, sweating changes, or sweating asymmetry
  - Motor/trophic: Evidence of decreased range of motion, motor dysfunction (eg, weakness, tremor, dystonia), or trophic changes (eg, hair, nail, skin).
- No other diagnosis better explaining the signs and symptoms.

Suggested explanations

CRPS is one of the most severe and debilitating of the pain syndromes within PSS. The explanatory model in the PNE section (page 37) may take some time, but they can turn such an illness around. It will be useful to offer some initial information, provide some resources for the patient and family to explore, then see them again for another consultation. These are useful first steps if there are long delays to be seen at a persistent pain or orthopaedic clinic that is effective with CRPS.

Chronic Fatigue Syndrome/Myalgic Encephalitis (CFS/ME)

The National Institute for Health and Care Excellence (2007) (NICE) state that doctors should consider diagnosing CFS if a person has **fatigue** and that all of the following apply:

- it is new or had a clear starting point (i.e. it has not been a lifelong problem)
- it is persistent and/or recurrent
- it is unexplained by other conditions
- it substantially reduces the amount of activity someone can do
- it feels worse after physical activity.

The person should also have one or more of these symptoms:

- difficulty sleeping, or insomnia
- muscle or joint pain without inflammation
- headaches
- painful lymph nodes that are not enlarged
- sore throat
- poor mental function, such as difficulty thinking
- symptoms getting worse after physical or mental exertion
- feeling unwell or having flu-like symptoms
- dizziness or nausea
- heart palpitations, without heart disease.

This diagnosis should be confirmed by a clinician after other conditions have been ruled out, and the above symptoms have persisted for at least four months in an adult and three months in a child or young person.

Suggested explanations

CFS/ME can be a very debilitating disease. Fatigue is, however, a normal sensation that helps to restore the organism as part of essential homeostasis. Similar to pain, the purpose of the sensation of fatigue is to prevent further damage to the body if the CNS deems that to be likely. Looking at this illness then through a neuroscience lens, the 'fatigue centre' of the CNS is perhaps misreading the incoming signals and making errors in its calculations, so the sensation of fatigue is being turned on almost all the time. These ideas may lead to an effective explanation for CFS/ME, with or without touching on the role of the SNS as well. Almost all PSS involve negative feedback loops, and recovery often begins when the individual's experience of such cycles are identified and addressed.

Some patients develop chronic fatigue after a head injury or traumatic brain injury, while other patients develop fatigue after a bout of Covid. Regardless of the initial trigger, the list of symptoms above indicate that central sensitization can be one of the key components of CFS/ME. This needs to be explained to patients, including how neuroplasticity can also be a positive, as over-active pathways can be turned down again.

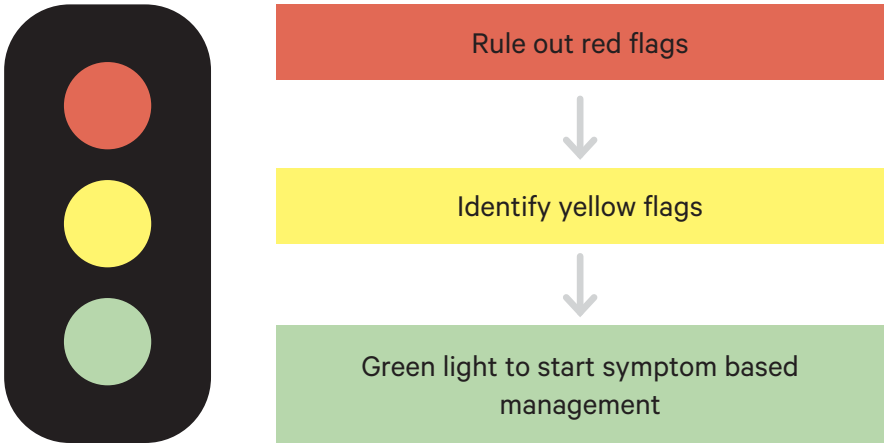
Looking at CFS/ME as a functional neurological disorder can also be useful. As noted in the FND section, "Functional neurological disorders (FND) are associated with considerable distress and disability. The symptoms are not faked. Diagnose FND positively on the basis of typical clinical features. It is not a diagnosis of exclusion. Psychological stressors are important risk factors, but are neither necessary nor sufficient for the diagnosis." The leading website for both patients and doctors is www.neurosymptoms.org from Dr Jon Stone in Edinburgh. There are also several community-based clinics for patients with CFS/ME listed in the Resources section.

13 Harden RN, Oaklander AL, Burton AW, Perez RS, Richardson K, Swan M, et al. *Complex regional pain syndrome: practical diagnostic and treatment guidelines,* 4th edition. Pain Med. 2013 Feb. 14 (2):180-229.

Pelvic pain and chronic pelvic pain (CPP)

As a junior doctor, you will encounter patients with pelvic pain in emergency departments, as an inpatient, at outpatients or in primary care. Remember that for most patients, pelvic pain can be a common and normal feature of menstruation and there is no sinister cause. Note also that correlations between pelvic pain and the presence or absence of endometriosis is quite poor; some patients with this have no pain, while other patients have pain but there is no endometriosis. As outlined throughout Te Kete, such observations are not surprising, given that pain is an outcome of complex interactions between the periphery and the CNS.

When assessing younger patients during one of their early episodes of pelvic pain, the following traffic light system is very helpful (with thanks to Dr Karen Joseph and Dr Emma McFarlane).



Rule out red flags: look for pregnancy such as ectopic pregnancy, STI, IBD, malignancy, weight loss, growth slowing in adolescence, GI bleeding, vomiting, fever, persistent diarrhoea, or any abnormalities on examination. Unless there are specific abnormal findings on history or exam, then limit investigations to simple tests (eg CBC, CRP, MSU, Ptest, STI screen and ultrasound of pelvis). Avoid 'specialty ping-pong' (a succession of referrals to various specialties) as this can reinforce the message that pain means there is some sort of ongoing problem. The absence of red flags is sufficient guidance for immediate management.

Identify yellow flags: Assess background factors. Explore their experience of their symptoms and what they think about it. For adolescents with pain do a HEeADSSS[1] assessment and enquire about Adverse Childhood Experiences (ACES). Psychosocial yellow flags are more predictive of long term prognosis than presence or absence of endometriosis.

Green light to start symptom based management: Explanation needs to start with what they understand about their own anatomy of the pelvis and their ideas about what is wrong. This can lead to education about pelvic anatomy and how pain with menstruation is common and normal. Listening to their experience of illness may be a good start in gaining their trust. There are lots of useful online explanatory videos about physiology and simple education can be sufficient. If the pain has become persistent and you have time, offer some ideas from Te Kete or provide them with further resources.

1 See for example starship.org.nz/guidelines/adolescent-consultation Home; education; eating and exercise; activities and peer relationships; drug use, cigarettes and alcohol; sexuality; suicide/self-harm/mood/depression; safety/risk taking behaviours.

Recurring or chronic pain in the pelvis can be one of the more distressing persistent pain syndromes. Pelvic pain can be triggered by a wide number of factors but once established it shows all the hallmarks of other chronic primary pain conditions such as CRPS, where the underlying problem appears to be central sensitisation causing nociplastic pain. Further searches for nociceptive origins of pain (ie 'lesion-focused' management) such as repeated endoscopies looking for endometriosis are likely to be unsuccessful.

Patients may have had a challenging illness so far with many unfruitful investigations. Their treatment may also have included opioids which can relieve pain in the short term, but can become counterproductive in the long term, ie more than a week.

Treatment outcomes are improved if management is provided by interdisciplinary teams who are all well-versed in contemporary neuroscience. Health professionals in these teams usually include pain specialists, pelvic floor physiotherapists, occupational therapists and psychologists, each of which can target various components of the underlying neurophysiological processes. Just as a door with multiple locks will need many keys, patients with CPP may need wide ranging input. Addressing the stigma and shame of having a chronic condition may be important; this is done through empathic engagement, respect and addressing core beliefs.

CPP could be considered as a 'disease of the pain system', but unfortunately in New Zealand, public funding for persistent pain clinics is often insufficient, while gynaecology services in main towns may not cater specifically for patients with CPP. Some patients bypass these public deficits by accessing private providers. Helping them find a pain specialist may be helpful if local providers have not been effective. Remember that recovery is possible in most cases.

Community providers

- For Otago patients, the Woman's Health Bus is a mobile health service, focused on womens health. See www.womanshealth.nz
- The Australis Specialist Pain Clinic has pain specialists who focus on chronic pelvic pain. See www.australispainclinic.co.nz/pelvic-pain-clinic

Persistent back pain

Episodes of back pain are very common and most get better by themselves. Less than 1% of all episodes of back pain are due to infection, cancer or fracture. Most of the time, back pain does not become chronic. For first episode acute back pain, the best practice now in ED and primary care is to reassure the patient that most people will fully recover, to not Xray unless there are red flags, to avoid opioids, and to encourage movement rather than rest. Some people get referred pain down their leg, which sometimes (but not always) indicates nerve entrapment. If not settling after 6 weeks, then surgery is sometimes required.

These current guidelines are intended to counteract widespread 'myths' about back pain, commonly held by patients, medical students and doctors. The myths are:

1. *"If you have a 'slipped', 'herniated' or 'ruptured' disc, you must have surgery and therapy.*
2. *Radiographs, CT and MRI scans can always identify the cause of the pain.*
3. *If your back hurts, you should take it easy until the pain goes away.*
4. *Most back pain is caused by injuries or heavy lifting.*
5. *Back pain is usually disabling.*
6. *Everyone with back pain should have a spine radiograph.*
7. *Bed rest is the mainstay of therapy."*

As outlined elsewhere in Te Kete, such myths arise on the misapprehension that all pain is mechanically straightforward and due to injury to tissues, ie is nociceptive. However, low back pain, especially when it becomes persistent, has different mechanisms to the pain from cutting your foot. To illustrate these points, there is poor correlation between pain and MRI findings (eg some people with pain have normal findings, others with abnormal findings have no pain).

It is important not to over-medicalise new patients with low back pain. This is because negative beliefs can arise from those interactions with doctors, especially if the Xrays show some sort of abnormality. The findings are then considered to be the 'cause' of the problem, a sort of self-fulfilling prophecy.[1]

With patients then, the first step is to listen to their ideas and beliefs about their ongoing problems, identifying any of the myths above as well as any 'pain catastrophising' (negative beliefs about poor outcomes, helplessness, rumination or pessimism). If someone is insistent that surgery is required, then the doctor will need to provide an alternative explanation for their pain that makes sense to the patient. Such explanations may take some time, as it can be difficult for anyone to let go of strongly held beliefs. Advanced communication skills may be required; respect for the patient and their current ideas is central to success.

Pain science education, multi-disciplinary input and graded exposure to precipitating factors contribute to best outcomes for persistent pain that is usually pain due to central sensitization, nociplastic mechanisms and concurrent muscle tension and spasm.

Here are some useful links:

- Understanding chronic back pain in less than 5 minutes and what to do about it. www.youtube.com/watch?v=C_3phB93rvI Chronic pain as the brain's perception of a stimulus.
- P4Work Neuroscience education for low back pain: www.youtube.com/watch?v=eWdm4H5yTYU
- P4Work: What everyone should know about low back pain: www.youtube.com/watch?v=9De7xeDATGU
- The Lancet series on Low Back Pain, 2018: www.thelancet.com/series/low-back-pain

1 Lin I et al. Disabling chronic low back pain as an iatrogenic disorder: a qualitative study in Aboriginal Australians. BMJ Open. 2013;3(4):e002654.

Recommended resources

Here are some useful resources if you are interested in exploring these clinical issues further. Specific resources for some of the syndromes in the Appendix are listed at the end of those sections.

Available for all OMS students

- *Pain Library Guide*. This is a recent set of resources developed by the Pain@Otago research team: see www.otago.ac.nz/pain

Somatisation: Useful podcast

- Dr Brett Mann: *Somatisation in a 15-minute consultation*. Dr Mann is the leading GP and CME educator in NZ on identification and management of somatisation: www.goodfellowunit.org/events/somatisation-15-minute-consultation

General articles and books

- Meador C. *Symptoms of Unknown Origin: A Medical Odyssey*. Vanderbilt Univ. Press, Nashville, 2005. Fascinating clinical stories from an endocrinologist who found that all his tests didn't help him make a diagnosis, so had to listen to the patient's story.
- Broom B. *Somatic illness and the patient's other story: A practical integrative mind/body approach to disease for doctors and psychotherapists:* Free Assn Books; 1997.
- Wilson H, Cunningham W. Illness without disease (Ch 7). In: Wilson H, Cunningham W. *Being a Doctor: Understanding Medical Practice*. University of Otago Press; Dunedin, 2013. This chapter explains how to approach the diagnostic dilemma when there is no 'obvious' diagnosis.
- Doidge N. *The Brain that Changes Itself: Stories of Personal Triumph, Frontiers of Brain Science*. London: Penguin; 2007.
- Stone, L. *Blame, shame and hopelessness: medically unexplained symptoms and the 'heartsink' experience*. Aust Fam Phys 2014;43(4):191-195. Common counter-transferences to these patients.
- Silverwood, V., C.A. Chew-Graham, I et al. *If it's a medical issue i would have covered it by now*. BMC Med Educ. 2017;17:160. Lack of training for these illnesses.
- Joyce E, Cowing J, Lazarus C, et al. *Training tomorrow's doctors to explain 'medically unexplained' physical symptoms: An examination of UK medical educators' views of barriers and solutions*. Pat Educ Couns. 2018;101(5):878-84.
- Hess SM. *It's not your heart: Group treatment for non-cardiac chest pain*. J Specialists in Group Work. 2011;36(4):296-315.
- Mann B, Wilson H. *Diagnosing somatisation in adults in the first consultation: moving beyond diagnosis by exclusion*. Brit J Gen Prac. 2013;63:607–8.

Pain Neuroscience: Nociplastic pain and central sensitization

- Butler DS, Moseley GL. *Explain Pain* 2nd Edn. Adelaide, Australia. Noigroup Publications, 2013. Brilliant book that needs to be read by all health professionals.
- Dunbar J, Wilson H. *Emerging models for successful treatment of complex regional pain syndrome in children and young adults.* J Prim Health Care. 2019;11(3):283-7.
- Fitzcharles, M.-A., S. P. Cohen, D. J. et al. *Nociplastic pain: towards an understanding of prevalent pain conditions.* The Lancet 2021;397(10289):2098-2110.
- Kosek E, Clauw D, Nijs J et al. *Chronic nociplastic pain affecting the musculoskeletal system: Clinical criteria and grading system. Pain.* 2021;162(11):2629-34.
- See resources listed at the start of the Pain Neuroscience section. Also:
- Dr Lorimer Moseley: Why things hurt. www.youtube.com/watch?v=gwd-wLdlHjs Educational and witty.

Re SNS, anxiety and bodily symptoms

- Porges SW. *The polyvagal theory: Neurophysiological foundations of emotions, attachment, communication, and self-regulation.* WW Norton & Company; 2011.
- Patient information sheets from The Centre for Clinical Interventions, Australia; This centre supplies many resources for clinicians to provide to patients. cci.health.wa.gov.au/Resources/For-Clinicians For example;
    - What is Anxiety?
    - The Vicious Cycle of Anxiety and Bodily Symptoms
    - Coping with Stress.

Bayesian predictive coding theory

- Van den Bergh O, Witthöft M, Petersen S, Brown RJ. *Symptoms and the body: taking the inferential leap.* Neuroscience & Biobehavioral Reviews. 2017;74:185-203.
- Edwards MJ, Adams RA, Brown H, Parees I, Friston KJ. *A Bayesian account of 'hysteria'.* Brain. 2012;135(11):3495-512.
- Fiorio M, Braga M, et al. *Functional neurological disorder and placebo and nocebo effects: shared mechanisms.* Nature Reviews Neurology. 2022;18(10):624-35.
- Fobian AD, Elliott L. *A review of functional neurological symptom disorder etiology and the integrated etiological summary model.* J Psychiatry Neuroscience. 2019;44(1):8-18.

Lifestyle and whole person approaches

- Hayes C, Naylor R, Egger G. *Understanding chronic pain in a lifestyle context: The emergence of a whole-person approach.* American Journal of Lifestyle Medicine. 2012;6(5):421-8.
- Christ A, Latz E. *The Western lifestyle has lasting effects on metaflammation.* Nature Reviews Immunology. 2019;19(5):267-8.
- Egger G. *In Search of a Germ Theory Equivalent for Chronic Disease.* Prev Chronic Dis. 2012;9:110301.

Community-based providers and on-line resources to enhance self-management

- **Curable.** Web-based app and resources. Useful site providing access to modern effective techniques for chronic pain and other issues. www.curablehealth.com Very popular and effective tools available.
- **Whole Person Healthcare:** wholeperson.healthcare Resources for both clinicians and patients, based on the patient's personal story. Led by Professor Brian Broom, this site hosts **The Illness Explorer:** wholeperson.healthcare/illness-explorer Hands-on learning tool and

self-help for patients. Regular newsletters for health professionals (admin@wholeperson.healthcare)

- **Empower Therapies.** Mel Abbott is based in Auckland and runs 4-day group workshops based on education about SNS and CBT. empowertherapies.co.nz
- **The Community Rehabilitation Centre,** Wakari Hospital Dunedin. Dr Matt Richardson is a Clinical Psychologist with an interest in FND.
- **Australis Specialist Pain Clinic** is a community-based Specialist pain management clinic for Canterbury and the South Island. www.australispainclinic.co.nz
- **A PNE-based pelvic pain app** is currently being designed in New Zealand. See www.ellahealth.co

Confidential notes re patients with chronic pain or persistent somatic symptoms

It is useful to keep learning about these illnesses by adding notes under the following headings, then seeing if there are overall patterns. Please ensure confidentiality of patient material.

Date: **Age, sex:** **Clinical setting:**

Presenting complaint:

List observable clues[1] to atypical illness:

Management offered/Clinical course and outcome:

Your observations and perspectives:

Date: **Age, sex:** **Clinical setting:**

Presenting complaint:

List observable clues to atypical illness:

Management offered/Clinical course and outcome:

Your observations and perspectives:

Date: **Age, sex:** **Clinical setting:**

Presenting complaint:

List observable clues to atypical illness:

Management offered/Clinical course and outcome:

Your observations and perspectives:

Overall impressions, including what you would like to learn:

---

1 Atypical history and signs include: Unusual symptoms for that organ system, non-anatomical distribution, hyperalgesia and/or allodynia, extended duration, normal findings on exam, normal Xrays and tests, nil 'red-flags', positive confirmatory signs, psychosocial triggers, symptoms match criteria for chronic pain or common PSS syndromes, etc.

Confidential notes re patients with chronic pain or persistent somatic symptoms

It is useful to keep learning about these illnesses by adding notes under the following headings, then seeing if there are overall patterns. Please ensure confidentiality of patient material.

Date: **Age, sex:** **Clinical setting:**

Presenting complaint:

List observable clues[1] to atypical illness:

Management offered/Clinical course and outcome:

Your observations and perspectives:

Date: **Age, sex:** **Clinical setting:**

Presenting complaint:

List observable clues to atypical illness:

Management offered/Clinical course and outcome:

Your observations and perspectives:

Date: **Age, sex:** **Clinical setting:**

Presenting complaint:

List observable clues to atypical illness:

Management offered/Clinical course and outcome:

Your observations and perspectives:

Overall impressions, including what you would like to learn:

---

1 Atypical history and signs include: Unusual symptoms for that organ system, non-anatomical distribution, hyperalgesia and/or allodynia, extended duration, normal findings on exam, normal Xrays and tests, nil 'red-flags', positive confirmatory signs, psychosocial triggers, symptoms match criteria for chronic pain or common PSS syndromes, etc.

Summary

Te Kete is an introduction to various explanatory models for patients with persistent somatic symptoms. In order to become proficient, we recommend sharing notes and ideas with your colleagues, in your peer groups and in supervision.

Further iterations of this Kete will be forthcoming, so feedback and comments are appreciated.

A/P Hamish Wilson, Dunedin: hamish.wilson@otago.ac.nz
Dr Martyn Williamson, Dunedin: martyn.williamson@otago.ac.nz
Mr John Dunbar, Te Whatu Ora, Southern: John.Dunbar@southerndhb.govt.nz
Professor Tony Dowell, Wellington: tony.dowell@otago.ac.nz
Dr Brett Mann, Christchurch: bmann@netaccess.co.nz
Clinical Professor Maria Kleinstaeuber, University of Texas: maria.kleinstaeuber@usu.edu
Dr Jim Ross, Dunedin: Jim.ross@otago.ac.nz

Design and typesetting by Flavia Rose Wilson.