Paradigms matter: why persistent pain is different and how dentists can help

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Key points

The experience of pain is influenced by both organic pathophysiology and (changes in) the pain signalling system. It is therefore important to assess for and communicate about the impact of each. As pain persists over time, typically, the role of organic pathophysiology becomes less and that of the pain signalling system more important to the ongoing experience of pain. Clinicians need to both understand the complexity of pain and to be able to communicate collaboratively about it with their patients. Differences in how pain is understood (organic pathophysiology versus changes in the signalling system) within a consultation are likely to reduce the likelihood of effective management.

Abstract

Dental professionals often expect, and are used to treating, pain that has a clear, organic and likely pathological cause. Patients visiting the dentist are also likely to share this expectation. However, in addition to potential organic contributions to the experience of pain, the nociceptive system (pain signalling system) also plays an important role. Alongside organic contributions, it is important to also consider that persistent pain is different to acute pain and requires different explanations and different management. Dental professionals need to be equipped to understand and explain persistent pain and to incorporate this understanding into their ongoing patient management so that patients can be educated in why the two are different and therefore require different approaches.

Why persistent pain is different and how dentists can help

Managing pain is part of routine dental practice. In many cases, this pain will conform to anatomically based expectations, for example, a decayed tooth causing signs and symptoms of irreversible pulpitis (an acute pain). Persistent (or chronic) orofacial pain is different. It is defined as pain that persists for more than three months,¹ yet, its differences from acute pain go far beyond this chronological distinction. Understanding such differences is key to being able to provide the right care to the right person in the right circumstances.

Taking temporomandibular disorders (TMD) as an example, jaw pain that has

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Refereed Paper. Submitted 5 September 2023 Revised 12 October 2023 Accepted 24 October 2023 https://doi.org/10.1038/s41415-024-7283-1 persisted for more than three months could be caused by organic factors, a small number of which $(\sim 1\%)^{2.3}$ represent pathophysiology that mimics TMD (see Table 1). Once such causes are ruled out, pain occurring beyond the time of normal tissue healing is likely to represent persistent pain, which is unlikely to be resolved by management that addresses only biological presumed sources of pain.

It is also important for clinicians to place the presence of organic findings into context so as not to overtreat. Again, using the example of TMD, (painless) joint noise(s) in the form of clicks or pops will be detected in up to one-third of the general population.⁴ In this situation, the noise does represent organic dysfunction (an asymptomatic disc displacement with reduction), yet treatment is not advocated beyond explanation and reassurance of the patient. Similarly, the extent of organic issues detected should also not be the determining factor in justification of invasive treatment strategies. Detection of osseous change within the temporomandibular joint complex has been shown to be comparable in symptomatic (90%) and asymptomatic (86.7%) joints. Furthermore, regardless of extent of organic changes,⁵ arthrogenous TMD are significantly more likely to remain stable or improve over time and outcome measures for conservative management compared to invasive surgical procedures (for example, arthrocentesis) are comparable in the vast majority of cases,⁶ supporting the use of reversible, symptombased management.⁷

Once a diagnosis for pain is established, it is important to understand that all types of pain have some degree of emotion associated with them which can influence management. For example, managing the acute pain of 'toothache' in an individual who is highly anxious is likely to involve a different approach to carrying out a similar procedure with somebody who is not anxious. The emotional component of pain is confirmed by the International Association for the Study of pain, who define pain as 'an unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage'.⁸

Beliefs about pain are just as important as emotions evoked by it or the thought of it. A seminal study⁹ illustrated that when participants believed they were receiving intravenous morphine but were in fact OPINION

Table 1 The origin of conditions which could mimic the presentation of TMD. Signs, symptoms and/or clinical history findings which can present in addition to pain and appropriate onward referral strategies are outlined for dental clinicians

Possible origin	History/sign/symptom(s) some or all of which may present	Onward referral	
Odontogenic conditions	Pain on biting; pain on eating/drinking; dental or periodontal disease	Dental clinician to complete comprehensive clinical ± radiological examination of all aspects of oral cavity and appropriately manage odontogenic disease	
Otological conditions	Ear pain (without temporomandibular joint pain); ear fullness; hearing change; tinnitus	Referral to GP to investigate ear symptoms or Ear, nose and throat (ENT) specialist	
Primary or secondary headache condition	Headache, not affected by jaw function. Other features may include aura, sensitivity to light and sound	Referral to GP to investigate headache symptoms	
Neoplastic conditions	Detectable swelling or mass;* history of previous malignant tumour with new onset facial pain or headache;* lymphadenopathy;* unplanned weight loss;* neurological signs/symptoms (for example, loss of smell or hearing, visual problems, neurosensory or motor change);* trismus;* voice change;* persistent mouth ulcers;* occlusal changes*	Onward referral to appropriate secondary care medical team	
Infective conditions	Malaise; fatigue; lymphadenopathy;* pyrexia (±) swelling and trismus;* neurological signs/symptoms (for example, loss of smell or hearing, visual problems, neurosensory or motor change);* trismus;* persistent mouth ulcers*	If infection odontogenic in origin, management should be completed by general dental practioner. If odontogenic infection severe or infection not dental in origin, onward referral to appropriate secondary care medical team	
Autoimmune conditions	Fatigue; lymphadenopathy;* neurological signs/symptoms (for example, loss of smell or hearing, visual problems, neurosensory or motor change);* persistent mouth ulcers*	Onward referral to GP or appropriate secondary care medical team	
Systemic conditions	Fatigue; malaise; lymphadenopathy;* unplanned weight loss;* persistent mouth ulcers*	Onward referral to GP or appropriate secondary care medical team	
Trauma	Trismus;* soft tissue trauma; occlusal changes*	Onward referral to appropriate secondary care medical team	
Key: * = Red flags which justify urgent/cancer waiting time referral to appropriate medical team			

receiving saline, pain reduced. When they were receiving morphine but believed it to be saline, pain increased. A recent study has taken this understanding further, demonstrating impressive improvements in persistent low back pain in patients receiving pain reprocessing therapy, a psychological therapy which aims to help patients to understand the role of the pain signalling system and apply techniques to change its' outputs.¹⁰ The clinical changes following this therapy were also evidenced by the changes in structural magnetic resonance imaging (MRI) scans of the brains of study participants. To make sense of these findings and many everyday examples of mismatches between amount of tissue damage and pain, we must recognise that pain is far more than a simple (direct) representation of tissue damage.

In any situation where pain is experienced, we might recognise that we are dealing with two distinct phenomena. The first is (potentially) compromise to the tissues (organic changes). Tissue damage is not necessary for the experience of pain but is frequently present. Metaphorically we can liken this to structural or physical problems in a building – a wall collapses or is compromised, a hinge becomes stuck, fire

exits become blocked with clutter. There are visible issues that require physical solutions. These issues, of course, need to be identified and addressed. The second phenomenon is the pain signalling system. We know that the experience of pain is influenced by factors beyond tissue damage. We can think of the pain signalling system as the body's health and safety officer. It aims to keep us safe by detecting potential problems and using various means (frequently pain) to alert and motivate us to prioritise and resolve them. In managing pain, we need not only to deal with any physical issues but also to convince the health and safety officer that everything is well-managed and there is no need for (further) concern.

Discrepancies between the level of tissue damage and intensity of pain are common in all types of pain, but these differences become particularly important when pain persists beyond three months. At this stage, pain is typically defined as persistent, or chronic pain.¹¹ Psychosocial factors are more accurate than physical measures at predicting the transition to, and prognosis of, persistent orofacial pain, such as TMD.¹² This does not, however, mean these psychosocial factors 'caused' the pain. As is the case with a fire,

which can be started in the right conditions by a spark but could not be distinguished by removing the first spark, commonly, the factors that perpetuate pain are different to those that were initially triggers. In terms of the metaphor above, we can imagine that even after initial issues are resolved, we still need to convince the health and safety officer, who has now been exposed to evidence of several potential problems, that everything is now well-managed and in hand. Different methods may be needed to achieve this for different people. The important take away is that for persistent pain, remaining tissue damage becomes less important and functioning of the pain signalling system more important. Management of persistent pain involves convincing our internal health and safety officer that we are no longer at risk.

One management approach commonly used is medication. Centrally acting drugs, such as amitriptyline and duloxetine, are commonly prescribed by general medical practitioners (GPs) or specialist pain teams for persistent pain, and in some cases can serve to 'quieten' the pain signalling system. To avoid/minimise adverse effects, such medications should be started at a low dose and slowly titrated up under close monitoring by the prescribing

Table 2 Options for non-medical management of persistent orofacial pain (alongside medical management or when medical causes have been excluded)

Modality	Purpose and rationale	Outcome and timeline
Online resources to support self-management, such as www.livewellwithpain.co.uk	Assist people to develop skills to self-manage their pain by understanding pain, reducing exacerbations and prioritising valued activities	Pain may reduce. A good outcome would be the ability to "live well with pain" and continue to engage in valued activities
Talking therapies. Access by searching online for local talking therapies service which will accept self-referral from patients	Psychological therapy for mental health problems that may co-occur with pain, including anxiety and depression	Therapy may take approximately three months. A good outcome would be the resolution or significant reduction of the mental health problem. This may also have a beneficial effect on the co-morbid pain. There can be long waiting times for treatment
Specialist psychology for pain management. This will usually be accessed through your local pain clinic. You may be able to make a referral or GP referral may be required. In some areas, community provision of pain psychology may offer direct access	Psychological therapy focused on managing pain. This may include education about how pain works and an assessment of how factors, such as emotions, thoughts and the impact of previous experiences may be specifically influencing pain. Based on the individual assessment, a collaborative therapeutic plan may focus on reducing pain exacerbations or increasing the ability to live well with pain	Therapy may take approximately three months. A good outcome may be the ability to live well with pain. A degree of pain reduction may also occur but is often not the primary aim of the therapy. There can be long waiting times for treatment
Pain management programme. Access will vary depending on location	Multidisciplinary group which provides education about pain and introduces methods of self-management	Groups usually run weekly for 6–8 weeks. Ability to live well with pain and engage in valued activities would be considered a good outcome

team. Beneficial effect can take up to 6-8 weeks to develop and the balance between potential side-effects and potential pain management benefit needs close individual consideration. They are recommended by current National Institute for Health and Care Excellence guidelines,13 not as a sole line treatment, but alongside consideration of regular exercise, psychological therapy and acupuncture, all of which, in some circumstances can help to calm the nervous system. Importantly, a person-centred and collaborative approach is also recommended, including getting to know the person, their circumstances and what is important to them. Potentially all these approaches could help to soothe our inner health and safety officer, given one important condition: we (the patient, clinician and wider professional community) need to have a shared understanding of what we are trying to achieve. All the above recommendations are eminently sensible if we are trying to calm the nervous system and if doing so is seen as a valid (and universally relevant) approach to pain management. However, if one or more parties in the conversation think that the default cause of pain is tissue damage, such recommendations can seem not only pointless but also dismissive, invalidating and potentially stigmatising. Returning to what we know about the importance of beliefs in mediating pain,9 such dissonance does not bode well for successful pain management.

For managing pain, paradigms matter and a shared patient-clinician understanding of paradigms matters. We cannot successfully manage persistent orofacial pain without understanding and developing a shared understanding with our patients about the nature of persistent pain. This includes a clear awareness of the integral importance of the pain signalling system as part of, rather than an adjunct to, the complex nature of pain.

Management options where this shared understanding exists may include psychological therapies or supported selfmanagement. Some avenues available to the interested patient are illustrated in Table 2. Alternative therapies, which patients may pursue according to their own belief systems, may also be helpful in this regard.

Historically, pain that is not accompanied by visible signs has been presented as 'psychological', a term that is understood by many as 'not real' or 'all my fault'. This is an unhelpful distinction. Psychological and social factors play a part in most pain and do so as part of a complex pattern of responses. The term 'chronic primary pain'13 and recognition that this can include cases where pain experience exceeds that expected to be caused by any known disease process is both more helpful and accurate. The concept speaks to the need to pay dual attention to tissue damage and the pain signalling system and to the routine importance of both. Dental professionals can capitalise on current understanding of pain and on current guidelines by routinely referring to tissue damage and heightened pain signalling in their everyday work.

A good example where dental professionals can see these heightened pain signalling

states is allodynia – noxious response to nonnoxious stimulus – which is commonplace in dental professionals' endodontic diagnostic armamentarium in the form of mechanical testing tenderness to percussion (TTP). Teeth may be TTP due to changes in their periapical status (inflammation or infection) and remain so for some time following treatment, but equally virginal teeth adjacent may also be simultaneously TTP but perfectly healthy.¹⁴

If dental professionals can take the opportunity to even at least briefly discuss with patients in lay terminology the ease at which we can have heightened pain signalling that can persist for a while (past the point of healing), in doing so, they will communicate that both acute and persistent pain are valid forms of pain that may need to be addressed. The leap from acute to persistent pain in terms of understanding and management may then be easier to relate to and less likely to be understood as pointless or dismissive.

Ethics declaration

The authors declare no conflicts of interest.

Author contributions

Chris Penlington conceived the idea, wrote the first draft and amended and agreed changes. Justin Durham conceived the idea and amended and agreed changes. Emma Beecroft revised the manuscript and amended and agreed changes. All authors agree to the final article.

References

 Treede R-D, Rief W, Barke A et al. A classification of chronic pain for ICD-11. Pain 2015; 156: 1003–1007.

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- Yanagi Y, Asaumi J-I, Maki Y et al. Incidentally found and unexpected tumours discovered by MRI examination for temporomandibular joint arthrosis. Eur J Radiol 2003; 47: 6–9.
- Riberio R F, Tallents R H, Katzberg R W et al. The Prevalence of Disc Displacement in Symptomatic and Asymptomatic Volunteers Aged 6 to 25 Years. J Orofacial Pain 1997; 11: 37–47.
- Bullitt E, Tew J M, Boyd J. Intracranial tumours in patients with facial pain. J Neurosurg 1986; 64: 865–871.
- Shahidi S, Salehi P, Abedi P, Dehbozorgi M, Hamedani S, Berahman N. Comparison of the Bony Changes of TMJ in Patients With and Without TMD Complaints Using CBCT. J Dent (Shiraz) 2018; 19: 142–149.
- Schiffman E L, Ahmad M, Hollender L et al. Longitudinal Stability of Common TMJ Structural Disorders. J Dent Res 2017; 96: 270–276.

- Schiffman E L, Velly A M, Look J O et al. Effects of four treatment strategies for temporomandibular joint closed lock. Int J Oral Maxillofac Surg 2014; 43: 217–226.
- Raja S N, Carr D B, Cohen M *et al.* The revised International Association for the Study of Pain definition of pain: concepts, challenges, and compromises. *Pain* 2020; **161:** 1976–1982.
- Bingel U, Wanigasekera V, Wiech K et al. The effect of treatment expectation on drug efficacy: imaging the analgesic benefit of the opioid remifentanil. Sci Transl Med 2011; 3: 70.
- Ashar Y K, Gordon A, Schubiner H et al. Effect of Pain Reprocessing Therapy vs Placebo and Usual Care for Patients With Chronic Back Pain: A Randomized Clinical Trial. JAMA Psychiatry 2021; 79: 13–23.
- 11. International Association for the Study of Pain. Definitions of Chronic Pain Syndromes. Available at

https://www.iasp-pain.org/advocacy/definitions-ofchronic-pain-syndromes/ (accessed August 2023).

- Fillingim R B, Slade G D, Greenspan J D et al. Longterm changes in biopsychosocial characteristics related to temporomandibular disorder: findings from the OPPERA study. Pain 2018; 159: 2403–2413.
- National Institute for Health and Care Excellence. Chronic pain (primary and secondary) in over 16s: assessment of all chronic pain and management of chronic primary pain. 2021. Available at https:// www.nice.org.uk/guidance/ng193/chapter/ Recommendations#managing-chronic-primary-pain (accessed April 2022).
- Erdogan O, Malek M, Gibbs J L. Associations between Pain Severity, Clinical Findings, and Endodontic Disease: A Cross-Sectional Study. J Endod 2021; 47: 1376–1382.

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