Mechanisms-based clinical reasoning of pain by experienced musculoskeletal physiotherapists

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Abstract

Objective In light of recent advances in understanding of the neurophysiological basis of pain, the use of mechanisms-based clinical reasoning strategies for pain has been advocated within physiotherapy. The purpose of this qualitative study was to investigate the nature and extent of mechanisms-based clinical reasoning of pain by experienced musculoskeletal physiotherapists, in relation to three different clinical pain presentations.

Design/participants Guided by an interpretative approach, a qualitative multiple-case studies design was used. Three videotaped patient–therapist clinical interviews were produced, each describing a different pain presentation. During and after the viewing of each of the three pain presentations, an audiotaped semi-structured interview was carried out with a purposive sample of seven experienced physiotherapists. The therapists were encouraged to verbalise their thoughts on aspects of each patient’s pain presentation. All interviews were subsequently transcribed, coded and analysed.

Results Four main categories of mechanisms-based clinical reasoning were identified. These were: (1) nociceptive; (2) peripheral neurogenic; (3) central; and (4) autonomic/sympathetic. There was some evidence to suggest that reasoning within these categories variously influenced therapists’ prognostic decision making as well as the planning of physical assessments and treatment. There was minimal evidence of reasoning according to the cognitive–affective mechanisms of pain, and no evidence of reasoning associated with motor, neuroendocrine and immune mechanisms and influences on nociception.

Conclusion The mechanisms-based clinical reasoning of pain by the participants in this study appeared to reflect the integration of a limited understanding of the neurophysiological basis of pain into clinical decision making associated with patients with musculoskeletal disorders. Physiotherapists may benefit from continuing education in order to broaden and update their knowledge of applied pain neurophysiology.

Keywords: Clinical reasoning; Pain mechanisms

Introduction

Pain mechanisms

Recent advances in the pain sciences have started to elucidate the pathophysiological mechanisms and processes involved in pain transmission, modulation and perception. In response to these developments, a number of authors from the scientific, medical and physiotherapy communities have advocated a mechanisms-based approach to the clinical reasoning and/or classification of pain [1–5].

The generation, modulation and perception of pain involves simultaneous parallel processing in multiple, interrelated body systems. For the purposes of description, the underlying neurophysiological mechanisms responsible for the generation and/or maintenance of pain have been broadly categorised into: (1) peripheral mechanisms of nociception, including nociceptive pain and peripheral neurogenic pain [6–8]; and (2) central mechanisms of nociception, including ascending pain pathways, central pain and central sensitisation, descending pain control and the cognitive–affective mechanisms of pain [9–11]. In addition, attention to autonomic, motor, neuroendocrine and immune system influences on nociception has also been advocated [3].
Clinical reasoning of pain in physiotherapy

Based on a growing body of evidence within the pain sciences, a number of authors have advocated focused attention towards the clinical reasoning of pain mechanisms within physiotherapy [3,5,12,13]. In one approach, it is suggested that clinical presentations of pain may be categorised into one or more of five distinct subcategories of pain mechanisms [5,12], including: (1) peripherally evoked nociception; (2) peripherally evoked neurogenic symptoms; (3) centrally evoked nociception; (4) autonomic and motor mechanisms; and (5) affective mechanisms.

In an alternative approach, the mature organism model [13] describes a conceptual framework for the clinical reasoning of pain based on the integration of knowledge from the science of stress biology with the neurobiological mechanisms of pain related to input into (nociceptive and peripheral neurogenic), processing within (central neurogenic and cognitive–affective) and output from (autonomic and motor) the central nervous system and other homeostatic systems (neuroendocrine and immune system influences).

An integrated clinical reasoning strategy for pain based on its underlying pathobiological mechanisms and the input/processing/output approach proposed in the mature organism model [13] describes a conceptual framework for the clinical reasoning of pain based on the integration of knowledge from the science of stress biology with the neurobiological mechanisms of pain related to input into (nociceptive and peripheral neurogenic), processing within (central neurogenic and cognitive–affective) and output from (autonomic and motor) the central nervous system and other homeostatic systems (neuroendocrine and immune system influences).

In light of this new finding, the previous presentation of results that only permitted a brief overview of mechanisms-based findings [17], and a growing consensus of opinion concerning the advocacy of mechanisms-based approaches towards the interpretation and treatment of patients’ pain, the aim of this paper was to: (1) describe in greater detail the nature and extent of mechanisms-based clinical reasoning of pain by experienced musculoskeletal physiotherapists in relation to three different presentations of pain; and (2) describe the apparent clinical utility of mechanisms-based reasoning.

Methods

Study design

Guided by an interpretative approach, a qualitative multiple-case studies embedded design was used in this investigation [18]. In a case study design, previous theory may be used to select the cases to be studied and to help identify relevant variables from the case studies database. In addition, such theory may be used as a template with which to compare the empirical findings of the case studies [18]. The conceptual framework and guiding propositions for this study were current scientific knowledge and theory concerning the neurobiological mechanisms of pain, together with their application to clinical practice [1–13]. In this investigation, the cases were the seven physiotherapists and the units of analysis were the therapists’ mechanisms-based clinical reasoning processes. In an embedded multiple-case study design, attention is also given to subunits of analysis within each case [18]. In this study, the subunits embedded within each case were the therapists’ reasoning processes in relation...
to three different pain presentations. Patients with differing presentations of pain based on their temporality, geography, pathology and history of onset were chosen in order to allow study participants to reveal potentially diverse orientations of mechanisms-based reasoning.

A purposive, criterion-based sampling strategy was used in this investigation [19]. Guided by the literature, a sample size of seven experienced musculoskeletal physiotherapists took part in the study [18]. Purposive sampling in qualitative research offers a strategy that better enables the researcher to study the phenomenon (i.e. mechanisms-based clinical reasoning of pain) under investigation, in contrast with probability sampling that enables statistical inferences to be made [19]. Criterion sampling requires that all participants have potentially experienced the phenomenon being studied and are able to articulate their conscious thoughts [19]. Such a strategy may enhance confidence in analytic findings on the grounds of representativeness [20]. Inclusion criteria in this study were at least 10 years of postgraduate clinical experience in musculoskeletal physiotherapy, and engagement in formal postgraduate study. The characteristics of the study participants are displayed in Table 1. The study took place in the physiotherapy department of a university teaching hospital in Dublin. All patients and physiotherapists gave signed consent prior to their participation in the study. To enhance validity, three principles of data collection were followed, namely the use of multiple sources of evidence, construction of a case study database, and the maintenance of a chain of evidence [18].

### Table 1

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<th>Profile of study participants (n = 7)</th>
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<td>Mean years since qualification</td>
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Process

Each participant viewed three videotaped patient-therapist clinical interviews, each describing a different pain presentation. Patient presentation A described the pain report of a female patient with a 4-year history of low back and leg pain following discectomy/spinal fusion after a work accident, patient presentation B contained the pain report of a female patient with complex regional pain syndrome (CRPS type I) 3 months after distal radius fracture, and patient presentation C depicted the pain report associated with a male patient 10 days after an ankle sprain from a sports injury.

Before viewing the three prerecorded patient-therapist clinical interviews, each study participant was given similar instructions based on those used in a previous qualitative study [21]. As each participant viewed each videotaped clinical interview, an audiotaped, semi-structured interview was carried out with the primary researcher (K.S.), a senior musculoskeletal physiotherapist working in a Dublin city university teaching hospital. At predetermined intervals, to facilitate comparison, the videotapes were paused and participants were encouraged to verbalise their thoughts regarding aspects of each patient’s pain presentation with the use of open-ended questions [22]. In addition, participants were free to pause the videotapes at any time in order to express any thoughts they might be having. None of the participants were prompted to express mechanisms-based reasoning, and the researchers had no preconceived ideas or expectations regarding the nature of the mechanisms-based clinical reasoning that participants may or may not have expressed. In response to answers from participants, follow-up questions were often asked by the researcher to encourage participants to further explain, clarify or elaborate upon aspects of their mechanisms-based reasoning. Examples of the types of open-ended and follow-up questioning tactics employed were:

‘Just after hearing the brief dialogue there, are you having any immediate thoughts or impressions regarding this patient’s pain presentation or potential pain presentation?’

and:

‘Can I ask you what you mean by the term “nociceptive pain mechanism”?’

All interviews were subsequently transcribed and coded. The final case study database comprised 21 transcripts.

Data analysis

Data analysis was guided by defined principles and tactics appropriate to qualitative methodologies [20]. An initial coding scheme was generated from the interview data in order to identify thematic categories of mechanisms-based clinical reasoning. The coding scheme was revised to reflect the multiple perspectives of study participants. Details of the final coding scheme are given in Table 2. All case study reports were coded using this final coding scheme, and intra- and intercoder reliability were checked. Kappa coefficients were calculated for overall agreement and showed intra- and intercoder agreement of 0.83 and 0.76, suggesting excellent and good agreement, respectively [23].

In order to further enhance validity, six verification procedures were incorporated into the relevant stages of this study, namely prolonged engagement and persistent observation, peer review or debriefing as an external check of the research process, triangulation of data through use of multiple data sources, negative case analysis (to purposely seek any inconsistent or disconfirming evidence for conclusions), rich thick description, and clarifying researcher bias, such that the researcher’s background, position and motivation were known along with how these may have impacted upon the
enquiry [19]. A within-case and cross-case analysis was constructed from all case study reports [18].

Results

Data are presented, in the form of verbatim quotes from the case study database, according to the five subdivisions of the defined code for mechanisms-based reasoning (see Table 2). All seven study participants demonstrated clinical reasoning of pain related to concepts associated with the neurophysiological mechanisms of pain. All citations are followed by a letter, corresponding to the pain presentation, and a number from 1 to 7, corresponding to each participating physiotherapist, in order to give a sense of the spread of the data. Four main categories of mechanisms-based reasoning were identified in this investigation. These were: (1) nociceptive; (2) peripheral neurogenic; (3) central; and (4) autonomic/sympathetic. There was minimal reasoning associated with a fifth ‘cognitive–affective’ category of pain mechanisms.

Nociceptive

Five out of seven study participants demonstrated clinical reasoning related to nociceptive pain mechanisms. Such reasoning was most commonly expressed in relation to patient presentation C (ankle sprain). For example, one participant stated:

‘So I think looking at the possible categories under which pain can be, I think that his is, in as much as it can be because pain is never that simple, but it’s almost 100% peripheral nociceptive in origin.’ (C3)

When asked to explain what was meant by ‘nociceptive pain’, only one participant responded with a neurophysiological-based explanation:

‘Pain from A delta and C fibres, local fibres within tissues where pain fibres are involved with tissues.’ (C3)

Reasoning in relation to nociceptive mechanisms of pain was more commonly explained in relation to the incidence or aetiology of a traumatic episode and the presence of pathological processes:

‘There’s local tissue damage and from the mechanism of injury I would expect there to be local tissue damage, and so he’s very much in the healing phase of that local tissue damage, so peripheral nociception, it’s acute, it’s recent, and he’s actively healing at the moment.’ (C4)

One other therapist made inferences about nociceptive pain mechanisms from the identification of a characteristic relationship between stimulus and response, for example:

‘Predictable stimulus response, so his pain is appropriate to the damage, he’s had an injury, his injury’s recent, he’s having pain that you’d expect to have with that sort of injury at this early stage.’ (C5)

Peripheral neurogenic

Five out of seven study participants demonstrated clinical reasoning related to peripheral neurogenic mechanisms of pain. Such reasoning was expressed almost exclusively in relation to patient presentation A (back/leg pain). Explanations regarding the meaning of neurogenic pain were often expressed in structural or anatomical rather than neurophysiological terms, for example:

‘Well neurogenic pain, I’m really just thinking she could be getting that from nerve tissue . . . she could still be getting leg pain from neurogenic, from neural tissues.’ (A4)

When expressed, reasoning related to peripheral neurogenic pain mechanisms appeared to be based on cues related to the geographical distribution of pain and the type of medication used. For example, one therapist suggested:

‘Well she’s having neurontin, so it confirms that there’s some kind of neurogenic component to it, or neuropathic, whatever you want to call it.’ (A7)
One study participant reasoned a distinction between neurogenic and nociceptive pain based on its quality and behaviour:

‘A neurogenic-type pain has got a different quality to say nociceptive pain, a mind of its own sort of pain, sharp in quality whereas her back pain she describes as nagging, you wouldn’t get that description of nerve pain.’ (A5)

Central

All seven study participants demonstrated reasoning with regard to central mechanisms of pain. Such reasoning was most often expressed in relation to patient presentations A (back/leg pain) and B (CRPS type I after fractured wrist). However, two study participants made reference to the absence of centrally generated mechanisms of pain in relation to patient presentation C. Study participants’ understanding of central pain mechanisms was generally expressed in broad neurophysiological terms related to pain processing. For example, one therapist explained:

‘I mean that either at her spinal cord level or at the brain level, that her pain processing has become altered, at a biochemical level basically. So, in terms of her perception of pain, she is getting ... a kind of reverberation around that central nervous system circuit.’ (A4)

Reasoning concerned with central mechanisms of pain also appeared to be linked to reasoning associated with the chronic nature of patient presentation A:

‘Looking at the length of time, we’re looking at a 4-year history so again you’re looking at a chronic pain presentation with central sensitisation.’ (A2)

During interpretations of patient presentation A, three study participants also demonstrated reasoning that distinguished between different categories of pain mechanisms, as explained by one participant:

‘Well initially when she first said, “I had an accident and got a prolapsed disc”, I would have been thinking, OK it’s a nociceptive type of thing, now I would be thinking there is obviously a central component to it all.’ (A7)

One participant also showed some awareness of the links between the emotional aspects of pain and central pain mechanisms:

‘The thinking behind it is that a depressed mental being will have altered central processing.’ (B1)

Autonomic/sympathetic

Reasoning related to autonomic/sympathetic mechanisms of pain was demonstrated by all seven study participants exclusively in relation to patient presentation B, and was expressed in both general and specific terms. One therapist observed:

‘I would put her pain down as sympathetically driven.’ (B5)

More specific references to autonomic/sympathetic mechanisms of pain were often made in conjunction with the recognition of other clinical features associated with patient presentation B, as typified in the following example:

‘The mottled skin, the poor circulation, discolouration, maybe making me think autonomic.’ (B6)

Reasoning in relation to autonomic/sympathetic mechanisms of pain was also associated with more diagnostic-based reasoning and the identification of clinical syndromes:

‘Well again it’s fitting into the sympathetic element of a causalgia or an RSD (reflex sympathetic dystrophy).’ (B4)

Participants’ understanding of the meaning of autonomic/sympathetic mechanisms of pain was expressed in various and broad neurophysiological terms related to, for example, overactivity or abnormal modulation of the sympathetic nervous system (SNS) and altered pain processing. For example, one therapist stated:

‘The sympathetic system for unknown reasons can become overactive.’ (B1)

Such variable expressions appeared to indicate that, within the group of study participants, there was no one uniform or singular meaning regarding what was understood by autonomic/sympathetic mechanisms of pain.

Cognitive–affective

There was minimal evidence from three participants of reasoning associated with the cognitive–affective mechanisms of pain. Furthermore, such reasoning was expressed indirectly rather then explicitly. For example, one participant appeared to make a mechanistic link between cognitions and behaviours in relation to patient presentation A:

‘If she perceives further pain as if, that’s another disc gone, or am I loosening the bony fusion, like that’s a real perpetuating mechanism for fear avoidance.’ (A1)

One other study participant made a limited reference to affective mechanisms, but only in what appeared to be its broadest sense, for example:

‘I would be definitely thinking that there is a central component but there is also an affective component to it all as well, so I don’t know if you call it a psychological or emotional or whatever but that affective component of pain.’ (A7)

The clinical utility of mechanisms-based reasoning

In addition to providing neurophysiological explanations for patients’ pain, there was also some evidence that mechanisms-based reasoning contributed to and influenced subsequent decision making associated with the planning of
Three study participants described how the presence of neurogenic mechanisms might invite a more cautious physical examination. For example, one participant stated:

‘Given that there’s a very strong neurogenic, and I suppose you would put RSD into the neuropathic component, I really don’t want to aggravate her too much in her examination. So although she’s 3 months down the road, I’m going to be careful in her assessment.’ (B4)

Mechanisms-based reasoning of pain also influenced four participants’ prognostic reasoning, where reasoning associated with nociceptive mechanisms tended to suggest a more favourable prognosis, and reasoning associated with neuropathic/central mechanisms of pain tended to suggest a less favourable prognosis:

‘I think it’s peripheral nociceptive and I think you’d want to do something really terrible to him not to make him better ... you can’t lose, this is my favourite kind of patient.’ (C3)

‘Her’s [pain] may be irreversible because of dorsal horn changes. And for that reason it might be unrealistic to expect that the pain can be cleared completely long-term you know so there may be always an element of pain there that we can’t actually influence.’ (A5)

There was also some evidence from four participants that mechanisms-based reasoning influenced subsequent thinking concerning approaches to treatment, particularly in regard to patient presentations A and B where the presence of neuropathic and/or central mechanisms led some participants to question the use of manual therapy, as the following comments illustrate:

‘It’s going to influence my treatment because the mechanically generated pain I might be able to have ... some more direct influence over by the techniques I might choose. Whereas the centrally generated pain I will perhaps be using a totally different strategy.’ (A6)

‘The nociceptive source of pain I don’t think is a huge element any more, I think that her whole pain processing is different, so she has a central component to her pains. Doing something on that single level trying to make it flex or extend is not going to make her go back to work full time.’ (A7)

Discussion

The results of this study provide some evidence for the use, amongst a sample of experienced physiotherapists, of one of the more contemporary approaches towards the clinical reasoning of pain described in the literature, i.e. based on consideration of the neurophysiological mechanisms of pain. Specifically, evidence was found of mechanisms-based clinical reasoning of pain in four out of the five categories of pain mechanisms described in one approach proposed in the literature [5,12]. Evidence of mechanisms-based clinical reasoning of pain has only recently been reported within physiotherapy [17], which may suggest a relatively recent trend towards the integration of basic pain neurophysiology into aspects of clinical decision making. In addition, this study also provides some evidence to support the theory that mechanisms-based reasoning may influence subsequent clinical decision making associated with the assessment, treatment and prognosis of patients presenting with musculoskeletal disorders [12]. However, the extent to which mechanisms-based reasoning of pain positively influences such decision making requires further empirical study.

Only minimal evidence was found of reasoning related to the fifth, affective, class of pain mechanisms [5,12]. Affective mechanisms of pain refer to the neurophysiological activity within, and the influence of, those central nervous system structures and pathways associated with the emotive aspects of pain. Whilst all seven participants attended to the emotional aspects of each patient’s pain as part of a broader biopsychosocial perspective [17], three participants made limited reference to affective mechanisms by name or indirect inference alone, and no participants demonstrated evidence of reasoning related to the emotive mechanisms of pain from an inherently neurophysiological perspective [24,25].

In addition, no evidence was found of reasoning related to the motor, neuroendocrine or neuroimmune mechanisms of pain, as described in the mature organism model [13] and pathobiological mechanisms-based approaches [3] associated with the clinical reasoning of pain. This may reflect a general lack of knowledge and understanding of the pathophysiological processes associated with these mechanisms, together with their clinical relevance, compared with the other four categories of mechanisms-based reasoning positively identified from this study. Alternatively, these absences may also highlight the difficulties that clinicians have in distinguishing such mechanisms from any clinical presentation of pain. A possible reason for the absence of reasoning associated specifically with motor mechanisms may be that the neurophysiological mechanisms underlying the links between the motor and nociceptive systems remain poorly understood both experimentally and clinically [26]; therefore, the absence of such reasoning from study participants may simply reflect the absence of any valid and reliable model to explain such links.

There was no direct evidence of mechanisms-based pain reasoning grounded within the input/processing/output or stress biology perspectives as conceived and conceptualised in the mature organism model and pathobiological mechanisms-based methods [3,13], suggesting that the mechanisms-based clinical reasoning of pain by the participants in this study most closely resembled the five-subcategory approach outlined previously [5,12]. This finding raises questions regarding the level of awareness and/or usefulness of reasoning associated specifically with the
input/processing/output and stress biology concepts inherent in these models amongst musculoskeletal physiotherapists.

Reasoning related to nociceptive mechanisms of pain was most apparent in relation to patient presentation C. Such reasoning was consistently based on the recognition of tissue trauma and, to a lesser degree, on the influence of inflammatory processes, and as such appeared to correlate at a general level with the neurophysiological mechanisms and features of nociceptive pain described in the literature [3,7,8].

Reasoning related to peripheral neurogenic mechanisms of pain was most evident in relation to patient presentation A. Therapists’ reasoning was predominantly based on recognition of the geographical distribution of pain, and was described in broad structural-oriented terms related to the involvement of nerve tissue. There was only minimal evidence from one participant of reasoning based on the pathophysiological processes associated with peripheral neurogenic mechanisms of pain, such as ectopic neural discharging, as described in the literature [3,6]. The results of this study therefore suggest that reasoning associated with peripheral neurogenic mechanisms of pain was grounded more in structural- than neurophysiological-oriented thinking.

Reasoning with respect to central mechanisms of pain was based on recognition of the longevity of patients’ pain and was expressed in basic neurophysiological terms associated predominantly with alterations in pain processing. This finding suggests a more general and superficial level of understanding, on the part of study participants, of the underlying pathophysiological basis of central mechanisms of pain in comparison with the literature [3,10]. No evidence was found of any broader based or deeper level of understanding of central mechanisms of pain, such as the role of the descending pain control and modulation systems, as described in the literature [11,27].

Reasoning in relation to autonomic/sympathetic mechanisms of pain was expressed exclusively and consistently in relation to patient presentation B. Such reasoning was based largely on the recognition of particular clinical signs and symptoms such as the appearance of mottled skin or skin discoloration, and was described in various and broad pathophysiological terms related to, for example, overactivity of the SNS. Such reasoning appears to be at odds with the current consensus of scientific opinion, which suggests that the mechanisms underlying SNS contributions to pain are likely to occur more as a result of neurophysiological coupling between the SNS and sensory nervous systems and abnormal sensitivity to normal sympathetic activity rather than from any abnormal functioning or overactivity of the SNS itself [28,29].

The findings from this study suggest a relatively multidimensional but basic level of clinical reasoning associated with the four categories of neurophysiological pain mechanisms that were identified positively. Whilst participants were able to broadly categorise clinical presentations of pain as nociceptive, for example, the results appear to suggest some limitations in the ability of study participants to fully describe and explain and therefore demonstrate a deeper understanding of the neurophysiological mechanisms underlying clinical presentations of pain. There also appeared to be some absences and inaccuracies in the mechanisms-based reasoning of pain by the therapists in this study compared with the literature. It has been suggested that ‘... perhaps the greatest limitation to therapists’ reasoning is the breadth, depth and accuracy of their clinically accessible knowledge’ [30]. However, it has been acknowledged that the body of pain science literature is vast and is often presented at a level of neurophysiological detail that may be difficult for clinicians to assimilate into and apply to clinical practice [30]. There is also some evidence to suggest that the basic sciences, including pathophysiology, do not readily inform and influence clinical decision making [31]. More optimistically, however, it has been shown that given suitable training, clinicians are capable of improving their knowledge and understanding of the neurophysiology of pain [32]. However, the extent to which such knowledge may benefit clinical practice and patient care is not yet known.

**Implications for physiotherapy**

The findings from this study may encourage physiotherapy clinicians and educators to reflect consciously upon the extent to which mechanisms-based reasoning of pain underlies and informs their practice and teaching. In addition, such considerations may stimulate professional debate regarding the potential and relative usefulness of mechanisms-based interpretations of patients’ pain. Scientists and clinicians alike have advocated focused attention towards the neurophysiological mechanisms of pain in the hope that greater understanding of such mechanisms may enable more effective treatment and management of clinical presentations of pain. Musculoskeletal physiotherapists may be required to engage in continuing education in order to develop the breadth and depth of their knowledge of applied pain neurobiology.

**Limitations of the study**

In view of the specificity of the group under investigation, i.e. experienced musculoskeletal physiotherapists, the results cannot be considered transferable across other clinical specialities within physiotherapy or to musculoskeletal physiotherapists without postgraduate education or with less experience. Future research might seek to compare the mechanisms-based clinical reasoning of pain between different groups of clinicians based on levels of expertise and education. In addition, a larger sample size or multiple researchers may have yielded different results. The fact that participants viewed prerecorded clinical interviews and did not conduct them firsthand may also have limited or altered the scope of their demonstrable mechanisms-based reasoning.
Conclusion

The findings from this study provide evidence of pain-oriented reasoning linked to four categories of pain mechanisms outlined in the literature [5,12], namely nociceptive, peripheral neurogenic, central and autonomic/sympathetic mechanisms of pain. Such reasoning appeared to have some influence on subsequent decision making associated with the assessment, treatment and prognosis of patients’ pain. The mechanisms-based clinical reasoning of pain by the participants in this study appeared to reflect the integration of a limited understanding of the neurophysiological mechanisms of pain into clinical decision making associated with patients with musculoskeletal disorders. The potential for physiotherapists to benefit from continuing education in order to broaden and update their knowledge of applied pain neurophysiology is highlighted.

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