How painful is juvenile chronic arthritis?

W Kuis, C J Heijnen, J A Hogeweg, G Sinnema, P J M Helders

Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage and it is one of the major symptoms in arthritis. Pain is also the sensory modality that alerts patients so that it leads to protective responses. Pain also informs the patient that there is a potential noxious threat, which brings him or her to the doctor for diagnosis and treatment of his illness (or ailment). Unfortunately, a warning signal like acute pain can turn into chronic pain and in this stage pain itself can be the most dominant symptom and threat for the quality of life of the patient.

In the pathogenesis of inflammatory pain, receptors sensitive for noxious stimuli generated as a consequence of inflammation, have a role. These receptors, called nociceptors, activate predominantly unmyelinated, small diameter sensory nerves, mediating nociceptive information to higher order neurones in the dorsal horn of the spinal medulla. On spinal as well as supraspinal level, the nociceptive signal is modulated by regulating pathways (fig 1).

Besides these regulating pathways there are many substances that are able to stimulate directly and/or sensitize primary afferent nociceptive fibres (table 1).

Children express pain in a different way than adults do. This is often interpreted as if children feel less or even no pain as in the case of neonates. The idea that newborns and children differ fundamentally from adults in the perception of pain, has led to approaches that are not humane. In a survey of British paediatric anaesthetists in 1985 it appeared that 48% of the respondents never provided postoperative analgesia of any kind to neonates during or after major surgery.

Differences in pain perception explain why there are so many discrepancies in the literature with respect to pain in children with juvenile chronic arthritis. Some investigations report that children with juvenile chronic arthritis experience substantially less pain or even no pain in comparison to adults with rheumatoid arthritis. However, we and others have found that children with juvenile chronic arthritis do express pain. By using algometry, we found an enhanced sensitivity in children with juvenile chronic arthritis to noxious stimulation in comparison to healthy age related controls.

When juvenile chronic arthritis patients had actively inflamed joints, the pain threshold was reduced by 31%. There was a significant inverse correlation between indices of disease activity and the pain threshold. We studied the relationship between pain threshold and pain expression as measured with visual analogue scales in a group of 57 children with juvenile chronic arthritis. By correlating three articular disease activity indices (active joint count, swollen joint count, and Ritchie index), visual analogue scales, and the functional child health assessment questionnaire with the mean pain threshold, we found that all the above mentioned items correlated negatively with the pain threshold. The latter may be interpreted that the pain threshold is related to disease activity and pain experience.

No correlation was found between the mean pain threshold and the C reactive protein.

Huskisson and Hart found a similar correlation between the Ritchie index and pain threshold in adults with rheumatoid arthritis. In contrast, Walco et al found no correlation between the pain threshold and the visual analogue scales in children with juvenile chronic arthritis. This difference can be partly explained by the choice of pressure sites and/or by patient selection. We found a significant inverse correlation between the pain threshold and pain experience measured with visual analogue scales, which could explain 25% of the variance. However, the pain threshold and the visual analogue scales measure different aspects of pain. The pain threshold as measured by the algometer might be easier to express and may be closely connected to the discharge of nociceptive afferents. In juvenile chronic arthritis the pain threshold is probably decreased as a result of the existing tissue damage in the inflamed joints. In comparison, the pain score measured with visual analogue scales is more influenced by the emotional experience associated with the disease.

An interesting phenomenon is that in juvenile chronic arthritis patients we measured a lower pain threshold of the involved joint as well as of the non-inflamed joints. In children with juvenile chronic arthritis in remission, we also found a lower pain threshold on all sites of measurement, although the effect was less pronounced than in children with active disease. In other words, the change in the pain threshold persists even after the input of the injured site has subsided. This shows that sensory induced changes and the impact they have on the central memory can be long lasting. The neurones on which these sensory induced changes can have such a longlasting impact are dorsal.
Table 1 Stimulating and sensitising substances for primary afferent nociceptive fibres

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<th>Sensitising substances</th>
<th>Stimulating substances</th>
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<tr>
<td>Substance P</td>
<td>5-hydroxytryptamine, serotonin, noradrenalin, leucotrienes, tachykinins, and many inflammatory mediators (interleukin-1, interleukin-6, tumour necrosis factor-α)</td>
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Figure 1 Neuronal events when inflammation in a joint is present. TST = tractus spinthalamicus; TSR = tractus spinoreticularis; NS = non-nociceptive nerves; WDR = wide dynamic range nerves; II, IIIa = myelinated afferents type II and IIIa, IIIb, IV = thinly or unmyelinated nociceptive afferents type IIIb and IV.

Chronic arthritis is accompanied by synovial effusion and tissue oedema. Together with the increased blood flow this may lead to an increase of the intra-articular pressure. The increased intra-articular pressure triggers the receptive endings of nociceptive fibres. The increased intra-articular temperature may increase neural velocity. An example of non-nociceptive mechanical stimuli applied to sites in the same segment as the inflamed joint inhibit presynaptic primary nociceptive afferents as well as postsynaptic activation of secondary neurons in ascending tracts, involved in nociceptive processing (fig 2). There are indications that the effects of afferent stimulation in patients with chronic pain last much longer than in healthy controls. An example of a non-nociceptive mechanical stimulus is transection of peripheral nerve fibres.
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Although its place in the treatment of chronic arthritis may contribute to the effectiveness of both types of afferent mediated inhibition has still to be evaluated. In juvenile chronic arthritis, not only serves to diminish pain, but may become increasingly important to modulate the degree of inflammation.

Figure 2 Non-nociceptive stimuli applied to sites on the same segment as the inflamed joint stimulate somatosensory afferent nerves. Somatosensory nerves activate inhibitory local interneurons in the substantia gelatinosa (lamina II), causing presynaptic inhibition of the primary afferent nociceptive nerves.


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