Pediatric Chronic Pain: Biopsychosocial Assessment and Formulation

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Chronic pain in children is an increasingly recognized clinical problem with alarmingly high prevalence rates found in some populations. Although it is not understood why some children experience high levels of pain, the subjective experience of chronic pain (including its site, intensity, quality, unpleasantness, and associated suffering) has long been believed to result from interactions between multiple contributors, including nociceptive, affective, sociocultural, behavioral, and cognitive. Regardless of whether the antecedent of chronic pain is known or unknown, similar patterns of symptoms, behaviors, and disability are often seen. Historically, however, there has been an unhelpful tendency to dichotomize chronic pain as either physical or functional in origin. However, recent studies strongly support a biopsychosocial basis to all pain, revealing its sensory emotional nature by showing that large distributed neural networks are accessed during nociceptive processing. The development and maintenance of chronic pain involve long-term changes in multiple integrated peripheral, spinal, and brain regions interacting in a complex way to shape the individual's experience. Hence, chronic pain from any cause cannot be viewed as a purely physical or psychological phenomenon, nor should it be expected that a unimodal approach to treatment will succeed. It follows that when assessing children and young people with chronic pain, information on a wide range of developmentally relevant dimensions, conveniently classified as biological, psychological, and sociocultural, should be gathered to formulate the potential causes, contributors, and effects of pain to devise an appropriate multimodal management plan.

Chronic pain (CP) is recurrent or persistent pain that extends beyond the expected time of healing (usually ~3 months). CP in children is an increasingly recognized clinical problem with alarmingly high prevalence rates in some populations; it may be a consequence of a chronic disease process, subsequent to an injury or surgery or, often, without any specific, identifiable cause. CP is difficult to manage and is often accompanied by comorbid symptoms and behaviors that can add to overall suffering and discomfort, dramatically reduce quality of life, and even delay or prevent recovery.[1,2]

Recent studies show that regardless of whether the antecedent of CP is known or unknown, similar patterns of symptoms, behaviors, and disability are often seen.[3] In addition to the physical and emotional costs to patients and families, the estimated economic burden of pediatric CP is substantial, and investigators have thus called for better identification, diagnosis, and treatment.[4,5]

WHO GETS PEDIATRIC CHRONIC PAIN?

The prevalence of CP increases with age and more advanced pubertal development, and there is a female

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preponderance.[6,7] High rates are reported for idiopathic pain (eg, headache [23%–51%[8–10]], functional abdominal pain [1.6%–41.2%[7]], back pain [14%–24%], and musculoskeletal pain [4%–40%[11]]), with a median prevalence of 11% to 38% in community surveys.[6] Similarly high prevalence rates are reported for disease-related pain. For example, pain associated with vaso-occlusive episodes is a common consequence of sickle cell disease; episodes can begin as early as 6 months of age,[12] and they can be frequent and severe, with adolescent patients with sickle cell disease reporting greater pain intensity than postoperative pediatric patients.[13] Approximately 20% of children report persistent postoperative pain after major surgeries.[14–16] In pediatric cancer populations, the actual prevalence of chemotherapy-induced peripheral neuropathy is unknown.[17,18] However, the incidence of neurotoxicity reportedly ranges from 3% to 13% in studies of pediatric patients with cancer to ~35% in pediatric patients treated specifically for acute lymphoblastic leukemia.[19] Interestingly, there can be a striking disconnect between clinically active disease and experienced pain. In children with polyarticular arthritis, 76% reported pain on >60% of days, despite apparent successful suppression of inflammation by treatment with methotrexate, tumor necrosis factor-α inhibitors, or both.[20]

WHY A BIOPSYCHOSOCIAL APPROACH?

Although it is not understood why some children experience high levels of pain, the subjective occurrence of CP (and its associated site, intensity, quality, unpleasantness, and related suffering) has long been thought to result from interactions between multiple contributors, including neurosensory (nociceptive), affective, sociocultural, behavioral, and cognitive factors.[21,22] As a result, a biopsychosocial model of CP is widely adopted, although considerable uncertainty remains regarding the exact roles and relative contributions of different elements of the model.[22] Unfortunately, and unhelpfully, this uncertainty has often led to a tendency to dichotomize CP into mostly nociceptive or physical in origin or mostly "psychological," the latter conclusion being frequently resisted and resented by children and families.[23]

Research findings in recent years (particularly results of in vivo neuroimaging studies) have driven a radical reappraisal of the neurophysiology leading to, and maintaining, CP. Importantly, this new knowledge provides stronger theoretical support to an integrative biopsychosocial understanding, assessment, and management of CP in both adults and children. Moreover, it also allows a more convincing narrative to negate dualistic theories and justify a broader and more comprehensive approach.

Assessing morbidity from CP is not just a matter of measuring pain intensity and subsequent suffering. In 2008, the pediatric Initiative on Methods, Measurement, and Pain Assessment in Clinical Trials made recommendations concerning outcome domains (and measures) that are important to assess in children and adolescents who have acute, chronic, or recurrent pain.[24] Seven domains in addition to pain intensity were identified: physical, emotional, and role functioning; sleep; other symptoms and adverse events; economic factors; and patient's global satisfaction with treatment. These domains map to most of the domains that need to be assessed in a biopsychosocial clinical interview.

NEUROSCIENCE OF PAIN

The development and maintenance of CP have been shown to involve long-term changes in multiple, integrated peripheral, spinal, and brain neural pain networks that interact in a complex way to contribute to the individual experience of pain, with large distributed brain "networks" being accessed during nociceptive processing. Melzack[25] first described these brain networks as the pain neuromatrix, now more commonly referred to as the "pain matrix." In children, many of these peripheral, spinal, and brain networks and systems are immature at birth and undergo changes in structure and function during the process of maturation, which add further complexity to the understanding, evaluation, and treatment of pain (other researchers have discussed this topic in more detail[26–29]). A meta-analysis of human data from positron emission tomography, functional MRI, EEG, and magnetoencephalography during an acute pain experience in the adult has confirmed that far from activating a single "pain" center in the brain, pain results in widespread activation of multiple cortical and subcortical regions, including: primary and secondary somatosensory, insular, anterior cingulate, and prefrontal cortices and the thalamus.[30] Although studies investigating pharmacologic analgesia show predominant effects in these brain regions, other regions such as the basal ganglia, cerebellum, amygdala, hippocampus, and areas within the parietal and temporal cortices, have also been found to be activated depending on the precise interplay of the factors involved in shaping pain perception (eg, cognition, mood, pain condition).[31–37] Consequently, the pain matrix is not a clearly distinct entity, leading many researchers in the field to call for a move away from a rigid neuroanatomic concept toward a

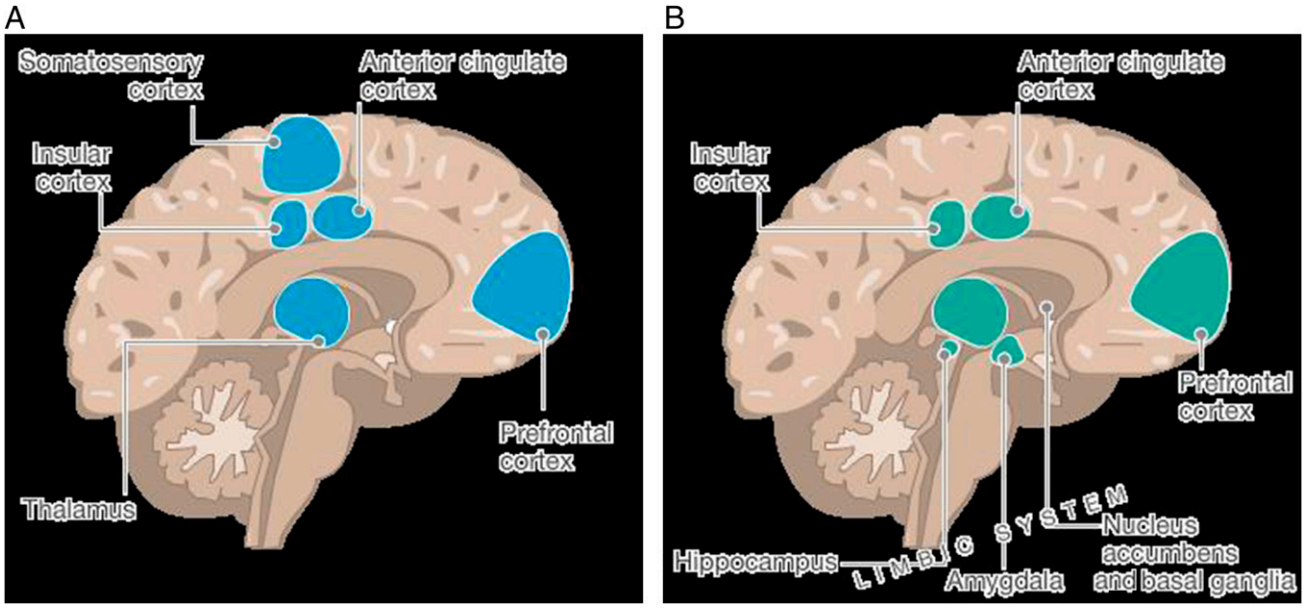

FIGURE 1
Important brain areas involved in pain perception and their respective functions (more information regarding main function is given in the Supplemental Information).

more individually unique neural "pain signature."[37–39]

For CP, the picture is even more complex. Human functional brain imaging studies indicate that CP conditions preferentially engage medial prefrontal cortical areas as well as subcortical limbic regions, especially portions of the dorsal and ventral basal ganglia, amygdala, and hippocampus.[30,37,40] Even though different types of CP, and different perceptions in individual patients, seem to engage distinct cortical and subcortical regions, overall across CP conditions there is generally a shift away from brain regions engaged in processing the sensory component of pain toward regions that encode emotional and motivational subjective states[41–43] (Fig 1). These areas are also strongly associated with functions that include learning, memory, and emotional responses and thereby are believed to relate to the cognitive and emotional problems commonly experienced by patients with CP, such as anxiety and depression,[44] impaired emotional decision-making,[45] working memory,[46] and difficulty in performing classic conditioning tasks.[47] Accumulating evidence suggests that experiences of physical and social pain (ie, social rejection, exclusion, bullying, negative social evaluation, loss of a close relationship), share neurochemical and neural substrates, an observation that provides further support for a biopsychosocial conceptualization of pain and helps explain phenomena such as improved pain control when social support is available and feelings of social isolation when in pain.[48]

Such brain imaging studies have contributed to the understanding of cerebral changes associated with CP, including structural, functional, and neurochemical alterations in patients with CP, compared with matched control subjects; there is clearly a distinct possibility that the changes observed are concurrently an effect and cause of ongoing pain.[49] It has also been noted that in most cases, a correlation exists between specific brain changes and the duration and intensity of CP (ie, the magnitude of the shift from normative data increases as the duration and intensity of pain increase).[50] This linear association suggests that long-term exposure to pain might cause central alterations such as decreased gray matter of the prefrontal cortex, and not vice versa.[51,52] Furthermore, recent studies have shown possible normalization of brain structure and function in response to effective psychological[53,54] and surgical[55,56] interventions for CP, indicating that brain plasticity can be bidirectional even in maturity. Two novel studies recorded broadly similar effects in pediatric patients after interdisciplinary rehabilitation treatment of complex regional pain syndrome (CRPS), a specific type of CP.[57,58] These findings encouraged experts to call for faster diagnosis and multidisciplinary management of CRPS in childhood.[59,60]

Peripheral nociception and the integration of nociceptive information centrally in the spinal cord and brain mature during infancy and childhood. Examples of the clinical consequences of this action in young patients include both a reduced ability to suppress incoming pain signals due to relatively slower development of

descending inhibitory systems from the brain to the spinal cord, as well as a reduced tendency to develop peripheral neuropathic pain after nerve damage.[61,62]

Although the network of brain regions that encode the cognitive, affective, and sensory aspects of adult pain have been well described,[30,37] the cortical and subcortical brain structures involved in infant and child nociceptive processing are less well known. Early indicators, however, point to a similar pattern of activation.[63] In a functional MRI study of pediatric CRPS, stimuli that evoked mechanical or cold allodynia produced patterns of central nervous system activation similar to those reported in adult CRPS.[64]

Brain structural and functional development also underlies the maturation of increasingly sophisticated cognitive abilities, and a similar shift with age from the recruitment of "bottom-up" brain processing regions toward "top-down" (ie, frontal-cortical and frontal-subcortical connections), suggesting progressive functional integration and segregation with age leading to a more mature, and controlled, cognition.[65] At a macroscopic level, brain development typically proceeds first in sensorimotor areas, spreading subsequently and progressively into the dorsal and parietal, superior temporal, and dorso-lateral prefrontal cortices throughout later childhood and adolescence.

Between childhood and adulthood, concurrent with cognitive maturation, there is progressively increased functional activation in task-relevant (lateral and medial frontal, striatal, and parieto-temporal) brain regions that mediate typically later-developing, higher level control functions such as cognitive and motivation control, timing, and attention.[65]

Overall, in brain development, there is a general pattern of functional and structural increase in connectivity and integrative processing, and a changing balance between limbic/subcortical and frontal lobe functions that extend well into young adulthood. This developmental maturation trajectory may explain the development of increasing pain-coping abilities in adolescents versus in children. A mature prefrontal cortex is necessary for good judgment, controlling impulses, solving problems, setting goals, and organizing and planning. The ability of children to use some of these complex coping strategies, such as cognitive reappraisal and acceptance, are correlated with better executive functioning (eg, working memory, cognitive flexibility, self-monitoring),[66] whereas disengagement coping is correlated with poorer executive functioning.

THE ASSESSMENT OF CP

It thus follows that when assessing children and young people with CP, information should be gathered on a wide range of relevant dimensions within a developmental context, to understand the causes, contributors, and effects of pain. Using a biopsychosocial framework, the dimensions are conveniently grouped into biological, psychological, and sociocultural factors.

Clinical history may therefore be compiled from multiple sources, including the young person themselves, parents or caretakers, teachers, and other professionals. In addition to direct questioning, where indicated and during detailed assessment, self-report and other-report scales and questionnaires are a useful adjunct, providing normative information that allows for a comparison versus the young person's peer group and an objective means for monitoring progress (Table 1).

The most commonly reported comorbid symptoms include diminished physical functioning, sleep disturbance, fatigue, and cognitive problems such as difficulties with concentration.[92] Furthermore, CP in children is associated with psychiatric comorbidity, particularly anxiety and mood disorders. Clinically elevated levels of anxiety vary widely across pain conditions, with high rates found among children with noncardiac chest pain (56%–81%),[93,94] abdominal pain (45%),[95] and fibromyalgia (58%),[96] whereas more moderate to low rates have been found among children with CRPS (20%),[97] unexplained pain (18%),[98] and headache (6%).[99] Similarly, elevated depressive symptoms are common in young people with CP[100,101] and have been associated with functional impairment[102] and problems with school functioning.[103] Risk for depression increases with pain frequency,[104] and, conversely, depressive symptoms have been identified as a risk factor for pain frequency, pain persistence, and the development of new pain problems over time.[105–107] Young people with comorbid depression and CP are at an increased risk of thinking about and attempting suicide.[108] Data from the National Longitudinal Study of Adolescent Health, a study of a nationally representative sample of 9970 adolescents in the United States, found that CP was related to suicide ideation/attempt both in the last year (odds ratio: 1.3–2.1) and during the subsequent year (odds ratio: 1.2–1.8).[109]

BIOLOGICAL DOMAIN

The initial evaluation comprises a complete medical and pain history (Table 2). This evaluation includes responses to current and previous medication complemented by physical and neurologic examination that involves observation of the

TABLE 1 Examples of Published Instruments for Assessing Young People Grouped Within the Biological Psychological and Social Domains of Relevance to CP

Domain		Construct	Example Instrument (Age Range, y)
Biological	Pain symptom	Pain intensity	VAS (10), NRS, VRS (>7)
			FPSS-R[67] (4–7), Electronic Pain Diaries[68]
		Pain characteristics	LANSS[69] (not currently standardized for children)
		Pain distribution	Body maps[70]
		Combination: intensity/distribution/quality	Varni/Thompson Pediatric Pain Questionnaire[71] (5–18)
	Comorbid symptoms	Fatigue	PedsQL (MFS) (0–18) (parent and child report)
		Functional status	FDI[72]
		Quality of life	PedsQL[73] (0–18)
		Sleep disturbance	CSHQ[74] (4–10)
Psychological	Emotional functioning	Depression	CDI[75] (7-17)
			RCADS[76] (6-18)
		Anxiety	STAIC[77] (8–12)
			STAI[78] (≥16)
			RCADS[79] (6–18)
		Anger	STAXI–2 C/A[80] (9–18)
			STAXI-2[79] (≥16)
		Combination: anxiety/depression	PI-ED[81] (8–18)
		Anxiety disorders (separation anxiety, generalized anxiety, panic, social phobia, obsessions-compulsions) and depression	RCADS[76] (6–18)
	Cognitions	Coping strategies	PCQ[82] (8–17)
		Catastrophizing	PCS-C[83] (8–17)
		Self-efficacy to manage pain	PSEQ[84] (not currently standardized for children)
Social	Environmental/Social	Family functioning	PSI-SF[85]
		Parental catastrophizing	PCS-P[86]
		Parental anxiety	BAI[78]
			STAI[87]
			HADS[88]
		Parental depression	BDI II[89]
			HADS[88]

Extensive reviews of available assessment tools for children with chronic pain have been published elsewhere.[90,91] BAI, Backache Index; BDI II, Beck Depression Inventory–II; CDI, Children's Depression Inventory; CSHQ, Children's Sleep Habits Questionnaire; FDI, Functional Disability Inventory; FPSS-R, Faces Pain Scale–Revised; HADS, Hospital Anxiety and Depression Scale; LANSS, Leeds Assessment of Neuropathic Symptoms and Signs; MFS, Multidimensional Fatigue Scale; NRS, Numeric Rating Scale; PCQ, Pain Coping Questionnaire; PCS-C, Pain Catastrophizing Scale–Child; PCS-P, Pain Catastrophizing Scale–Parent; PedsQL, Pediatric Quality of Life; PI-ED, Paediatric Index of Emotional Distress; RCADS, Revised Child Anxiety and Depression Scale; STAIC, State-Trait Anxiety Inventory for Children; PSI-SF, Parenting Stress Index/Short Form; STAI, State-Trait Anxiety Inventory; STAXI, State-Trait Anger Expression Inventory; STAXI-2, State-Trait Anger Expression Inventory–2; STAXI–2 C/A, State-Trait Anger Expression Inventory–2 Child and Adolescent; VAS, visual analog scale; VRS, Verbal Rating Scale.

child's general appearance, posture, and gait focusing on, but not limited to, an affected area. Identification of neuropathic pain is important because specific treatments may be indicated (Table 3).[62] Basic vital signs and growth parameters should be obtained during at least the first evaluation. Red flags (Table 4) are key clinical indicators of important pathologic findings that should be investigated and managed if present. Judicious laboratory and radiologic studies are useful if a specific yet undiagnosed disease is suspected.

Physical functioning, fatigue, and sleep disturbance should be evaluated and quantified where possible. Identification of sleep problems in children is particularly important because a growing body of evidence suggests a link between sleep disorders and physical, cognitive, emotional and social development,[111] depression, and physical disability in children with CP.[112,113] Improvement in sleeping habits has been proposed as one of the mechanisms of efficacy in interdisciplinary pain management programs.[114]

PSYCHOLOGICAL DOMAIN

Emotional Functioning

The high rate of comorbidity of mental health conditions along with CP requires assessment for the presence of anxiety and mood disorders. It must be emphasized that high comorbidity does not mean that one causes the other. Psychological distress is both a potential contributing factor and a potential outcome of living with CP. Each condition often involves separate interventions, which require referral to appropriate mental health services.

Pediatricians should be particularly alert to suicide ideation/attempts and comorbid depression in this at-risk population, and they should ascertain the suicidality of depressed adolescents (ie, whether and how often these adolescents think about suicide and whether they have ever attempted suicide). If suicidal ideation or recent suicidal behavior is present in a depressed teenager,

TABLE 2 SOCRATES Mnemonic: A Frequently Recommended Resource to Guide Initial Questioning in CP Assessment

Site	Where is the pain?
Onset	When did the pain start, and how did it first appear?
	Did the pain appear suddenly or developed over time?
	What were you doing when it started?
Character	What words would you use to describe the pain?
	Is it stabbing, sharp, aching, burning, shooting, hot, cold?
Radiation	Does the pain move elsewhere in the body?
Associations	Are there any symptoms, signs, or activities associated with the pain?
	Is it associated with bruising, swelling, nausea, or high temperatures? Does it always come on at certain times; for example, at meal times or when you are doing a particular activity?
Timing	Is the pain spontaneous or evoked?
	Constant, intermittent, or both?
	Background pain?
	Acute exacerbations?
	For how long have you had the pain and has it changed over time?
Exacerbating or relieving factors	What makes the pain better or worse?
	Response to previous treatments?
Severity	How intense is the pain?
	Does it stop you doing any of the things you like or need to do?
	What do you do when you have pain?
	Is there anything you avoid doing as a result of the pain?
	In what ways has your life changed since you developed pain?

TABLE 3 Differences Between Neuropathic and Nociceptive Pain[62,110]

Clinical Characteristic	Neuropathic Pain	Nociceptive Pain
Cause	Lesion or dysfunction of the nervous system	Damage or potential damage to tissues
Descriptors	Sharp, lancinating, shooting, electric-like, stabbing pain	Throbbing, aching, pressure-like pain
Sensory deficits	Common: numbness, tingling, pricking	Uncommon; if present, they have a nondermatomal or nonnerve distribution
Vasomotor signs	Temperature and color changes	Uncommon
Motor deficits	Neurologic weakness may be present if a motor nerve is affected; dystonia or spasticity may be associated with central nervous system lesions and sometimes peripheral lesions (eg, CRPS)	May have pain-induced muscle weakness
Hypersensitivity	Allodynia (ie, pain often evoked by nonpainful stimuli) Hyperalgesia (ie, exaggerated response to painful stimuli)	Uncommon
Character	Distal radiation common	Proximal radiation common
Paroxysms	Exacerbations common and unpredictable	Exacerbations less common and often associated with activity

he or she should be immediately referred to the appropriate mental health services.

Cognitions

Young people and their families hold beliefs about the pain that they experience such as what causes it, how long it will last, whether it is curable, what effects it will have in their lives, what treatments might be relevant, and whether it is understood and believed as "real" by clinicians. Beliefs are powerful and can influence not only pain perception but also treatment adherence and treatment response, and pediatricians may have to actively challenge erroneous beliefs and provide accurate pain education.[115]

Specific cognitions that need to be assessed include pain catastrophizing and coping. Pain catastrophizing, currently conceptualized as both related to a personality-based, dispositional construct and a response that varies in different situations,[116] is characterized by a negative mind-set, magnification, and rumination about pain.[117] Catastrophizing in children is distinct to anxiety and has been identified as a significant predictor of pain, functional disability, and health-related quality of life in children and adolescents with CP[118] and persistent pain and central sensitization into young adulthood.[119]

CP presents a range of stressors and challenges for young people and their families; pediatricians therefore need to assess how the young person copes with each of these factors and not just pain. Coping can be viewed as a collection of purposeful, volitional efforts that are mobilized under stress and directed at the regulation of aspects of the self (ie, emotion, cognition, behavior, physiology), known as secondary control, and interactions with others and the environment, known as primary control .[120,121] Relinquished

TABLE 4 Red Flags in Pediatric CP

Young age at presentation
Systemic upset
Fever
Malaise
Weight loss
Rashes
Lymphadenopathy
Hepatosplenomegaly
Pain that wakes at night
Bone pain
Joint swelling
Impaired growth and development
Neurologic signs
Depression, evidence of suicidal ideation or major psychiatric disorder
Suspicion of child abuse (eg, incongruence between history and presentation or pattern of physical findings)

control refers to the absence of any coping attempt.[122] In general, the degree to which a coping strategy leads to better or worse emotional and behavioral adjustment depends in part on the match between the demands of the stressor and the goals and nature of the coping response.[123] However, overall, studies have shown that secondary control coping (eg, acceptance, cognitive reappraisal, distraction) is associated with lower levels of somatic complaints and symptoms of anxiety and depression,[66,124–126] whereas passive coping (eg, behavioral disengagement, self-isolation, catastrophizing) is related to poorer adjustment.[119,127,128]

SOCIAL DOMAIN

Complex transactional processes and individual factors mediate children's and parents' emotional, cognitive, and behavioral responses to pain, ultimately influencing the child's overall functioning.[129] The 5 characteristics of family functioning commonly assessed in family systems theories[130] relevant in the assessment of the family of a child with CP are organization, cohesion, communication, affective environment, and problem solving.[131] Poorly functioning families can be those that are highly disorganized, with unclear communication and high expressions of conflict or negative affect that only become more disrupted when faced with a stressor. Poorly functioning families can also be characterized by being overly restrictive and ordered, limiting the adaptability of the system to deal with stressors, and limiting individual members' ability to express their emotions or modify maladaptive roles.

In terms of individual factors, parental cognitive responses to pain, such as parental pain catastrophizing or exaggerated negative pain appraisals, have been found to influence both parents' emotional reactions to pain and child functional disability. In addition, higher levels of parents' catastrophic thinking regarding their children's CP are associated with a greater tendency to restrict their children's pain-inducing activities and a greater tendency to prioritize attempts to control their children's pain.[132]

Behaviorally, parental protective responses to children's pain behavior (eg, increasing attention to pain symptoms, excusing the child from responsibilities) have been linked to poorer functional outcomes, serving as the proximal link between parents' internal reactions (eg, cognitions, emotional distress) and child outcomes.[133–135] Conversely, young people of parents with greater levels of psychological flexibility tend to report less physical disability, fewer depressive symptoms, and greater levels of acceptance of their own pain.[136]

Anxiety and mood disorders are prevalent among mothers of children with CP conditions,[137,138] but similar data about fathers are currently lacking. Mothers of children with functional abdominal pain are 4.9 times more likely to have a lifetime history of depressive disorders and 4.8 times more likely to have a lifetime history of anxiety disorders compared with mothers of healthy children.[137] Maternal depression is a risk factor for the socioemotional and cognitive development of children. Depressed mothers generally exhibit less attentiveness and responsiveness to their children's needs, and they are also poor models for negative mood regulation and problem solving. The pediatricians' role in maternal depression is one of screening, followed by guidance for additional evaluation and treatment.

School functioning is often negatively affected in young people with CP,[139] particularly when there is comorbidity with depressive symptoms.[103] Assessment of school functioning should include a number of dimensions,[140,141] namely: (1) school attendance, clarifying if the absence is due to pain or some other reason; (2) cognitive[142] and emotional[143] engagement (eg, self-regulation, studying habits and enjoyment, belonging, and attitudes toward every aspect of school); (3) academic performance (eg, grades across subjects, national standardized test scores, classroom participation); (4) self and teacher perceptions of academic competence[139]; (5) participation in school activities (eg, clubs, school trips); and (6) social functioning in the school setting (eg, social activities, interaction with peers). The limited number of existing studies suggests that young people with CP may have fewer friends, are more isolated, and may be subjected to increased rates of victimization by

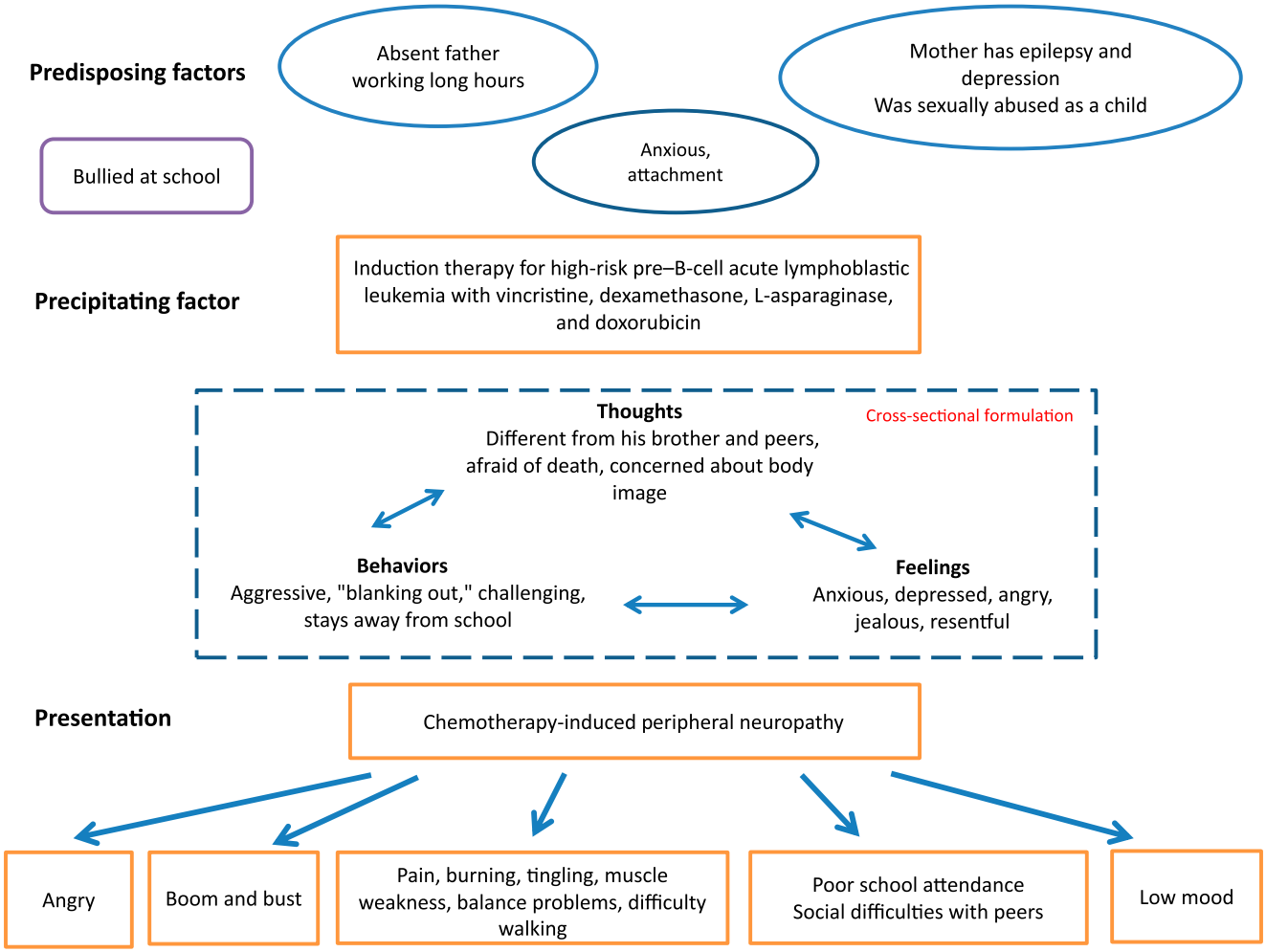

FIGURE 2

Longitudinal formulation for John's chemotherapy-induced peripheral neuropathy (the Case Study in the Supplemental Information discusses this topic in more detail).

peers compared with children and adolescents without pain.[144]

CASE FORMULATION

Case formulation provides the essential link between assessment and management planning. Assessment, formulation, and treatment are in fact dynamically linked with each other, each informing the other over time. Although case formulation has been described in adult CP,[145] limited literature exists in pediatric pain.[115,146]

Case formulation or conceptualization (these 2 terms are used interchangeably) is where theory, research, and young peoples' unique presentations come together. It is an individualized model that attempts to systematically draw together the precipitating, predisposing, perpetuating, and protective biological, psychological, and social factors believed to be idiosyncratically related to a particular young person's clinical presentation.[147,148] An effective case conceptualization should essentially answer the young person's question "why me, and why now?"

At its simplest level (ie, cross-sectional), the case conceptualization might focus on "negative automatic thoughts," which are locked into vicious cycles with dysfunctional emotions, behaviors, and somatic symptoms (Fig 2). A more comprehensive, longitudinal case conceptualization involves 3 steps. First, the clinician learns about the young person's pain problem and behavioral, emotional, and cognitive responses by observing, assessing, and measuring; second, the clinician then moves on to meaningfully organizing this information into patterns and themes; and third, he or she finishes by explaining the patterns and themes by using theory and research. When the case conceptualization is completed, the clinician should have a picture of what he or she believes has led to the young person's pain problem (etiology) and what features are maintaining or perpetuating the

problem (sustaining factors; Fig 2, see Supplemental Information). Understanding the etiology and sustaining factors will then lead to treatment planning, which uses the case conceptualization to decide how to best address, reduce, manage, or resolve the young person's pain and associated disability.

Crucially, the case conceptualization is collaboratively co-constructed with the young patients and their family and not simply presented to them. This approach allows for problems to be normalized and contextualized, and it facilitates empathy. The clinician and family work collaboratively to first describe and then explain the pain and associated issues a young person presents with.

CONCLUSIONS

Pediatric CP can be a difficult problem to conceptualize and treat, but evidence is rapidly accumulating that supports an integrative biopsychosocial approach to assessment, formulation, and management. Interdisciplinary outpatient and intensive inpatient treatment have been shown to improve pain intensity and disability in children with CP,[149,150] and the effects are maintained at the 1-year follow-up.[151] Future research could beneficially explore the association between brain reorganization seen on imaging and the chronicity of pain, with emphasis on how changes on imaging relate to pain behaviors and response to treatment longitudinally.

ABBREVIATIONS

CP: chronic pain
CRPS: chronic regional pain syndrome

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