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Fibromyalgia: revisiting the literature

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Fibromyalgia has a distinct clinical presentation. With no distinct characteristic beyond the presence of 11 or more tender points and chronic pain in all four quadrants of the body, it represents one extreme of a normal distribution of pain states. Research exploration utilizing the ACR criteria has not found solid empirical evidence to link the finding of multiple tender points to a specific pathological process. These points may be present as a concomitant finding with psychological disease states but they have not lead to further etiological understanding. Measurement of outcomes is difficult and the prognosis for patients in the speciality care setting is poor, however in the general population the prognosis is variable, and includes improvement without treatment. The success of treatments has been limited mainly to helping patients improve their ability to cope with, but not to eliminate the tender points.
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La fibromyalgie se présente différemment sur le plan clinique. Ne comportant aucune caractéristique distincte hormis la présence d'au moins 11 points douloureux et de douleur chronique dans les quatre quadrants du corps, elle représente un extrême de la distribution normale de la douleur. Une exploration de recherche utilisant le critère RAC n'a pas trouvé d'évidence empirique permettant de lier la découverte de points douloureux multiples à un processus pathologique. La présence de ces points peut coïncider avec la découverte d'un trouble psychologique, mais ne permet pas d'en arriver à une compréhension étiologique plus poussée. Les résultats sont difficiles à mesurer et le pronostic pour les patients devant recevoir des soins spécialisés est sombre. Cependant, dans la population générale, le pronostic est variable et des améliorations peuvent être apportées sans traitement. Le succès des traitements se limite principalement à aider les patients à améliorer leur capacité de composer avec les points douloureux, mais sans les éliminer.
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MOTS CLÉS : fibromyalgie, épidémiologie, traitement.

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Fibromyalgia has been recognized for nearly a century as "chronic muscle pain" of unknown origin, unremitting over time, and notoriously hard to treat.[1] Not an epidemiologically meaningful disease entity based on criteria outlined by Makela, it is important that fibromyalgia be presented as a distinct concept.[2] In this paper the authors have searched the Medline and Ovid databases in order to retrieve and review the pertinent literature relating to Fibromyalgia and chronic pain syndromes to create a synthesized view of this condition. The literature spans primary research, opinion and editorial papers by distinguished authors and meta-analysis of primary research.

Epidemiology

The now familiar American College of Rheumatology 1990 Criteria for the Classification of Fibromyalgia[3] is able to distinguish chronic widespread pain meeting the ACR criteria from that which does not. Although not definitively able to identify sufferers from controls, researchers find that the clinical/demographic variables of pain severity, weakness, 24 or more hours of severe fatigue after activity and self reported glandular neck swelling may identify those patients likely to meet the 11 of 18 tender point threshold.[4] Clinical criteria based on tenderness, pain and symptoms tend to capture the same group of patients and identification of only 40% of the "painful" tender points may be sufficient.[5] The weight of concomitant complaints such as sleep disturbance, stiffness or fatigue may lead to diagnosis when present with chronic wide spread pain. Patients may also have symptoms related to other physiological systems and there are many similarities between all of the unexplained syndromes (e.g. Restless Leg and Irritable Bowel Syndrome).[1,6]

Population studies estimate the prevalence to be 2% (women 3.4%, men 0.5%).[7,8] However estimates are sensitive to even minor modifications in definition.[9–11] Women are more likely than men to have 11 tender points and subjective symptoms including "pain all over", sleep disturbance, and fatigue. Prevalence peaks in the senior years (60–79) reaching greater than 7% for women over 70 years of age.[8,9] Associated demographic features include lower education, lower household income, divorce, and disability.[4] Reports vary between ethnic groups, and it is unclear whether differences in pain perception, socio-cultural factors, methodological approach, or genetics are at the root of this variation, but education level is likely the important factor.[12–14]

Less than 3% of chronic pain following trauma is diagnosed as fibromyalgia and although trauma increases the prevalence, most injuries do not lead to symptoms.[15,16] Patient's medicolegal status does not differentiate outcomes and there is no significant difference between the demographic data, nature of accident, symptoms and disability between patients with resolved and unresolved claims.[17]

Consensus

In 1992 the Copenhagen Declaration (CD) consensus group helped to define the diagnosis of Fibromyalgia under the World Health Organization's International Classification of Disease (ICD M79.0, 10th revision).[16] The Vancouver Fibromyalgia Consensus Group (VFCG) convened in 1994 to address research issues.[18] Whether viewed as part of a wider syndrome (CD) or seen to be concurrent with, and not mutually exclusive from other diagnoses (VFCG), the syndrome of fibromyalgia presents several clear issues:

1 Fibromyalgia is not an epidemiologically distinct disease. Causality and relevant risk factors remain unclear and there is still a need to understand its longitudinal course. Laboratory, muscle, radiographic, neuroendocrine, and immunologic testing are of no value in diagnosis and therefore a waste of valuable resources. There is a recognized sleep anomaly, but "soft" findings are adequate for clinical diagnosis.

2 A clinically significant decrease in self-report of function is not considered to cause a large loss in the ability to maintain gainful employment, but may make competitive employment difficult. Patients generally have a perception of decreased support and/or understanding from family and friends, and a lowered quality of life. Costs associated with clinical care are comparable to those for the osteoarthritis patient.[7] In 1996 the average cost per patient in one multicenter study was $2274 (USD), and in London, Ontario, Canada the direct cost for health services used by fibromyalgia patients is $493 (Cdn) above matched controls.[19,20]

3 Primary treatment recommendations are for multidis-

ciplinary/combined care focused on physical and psychological interventions. The use of antidepressants (amitriptyline and cyclobenzaprine) is recommended and narcotics are to be avoided. Generally pharmacological treatments are associated with significant improvements in physical status and self-report of symptoms, but over the short term only.[21]

4 The ACR criteria are the required standard for clinical research use but standardized outcome measures need to be determined. Methodological differences between the most stringent studies make interpretation of findings difficult, causing meta-analysis of outcomes studies to be limited in power. The ACR criteria should not be used for diagnostic and medicolegal purposes.

Thousands of papers have been published examining fibromyalgia, yet its etiology is only vaguely understood. Different health specializations create clinical designs that match the practices common to their own discipline (Chiropractic included) causing significant variation in study methodologies.[22] Never the less it is possible to distil the research initiatives of the past 15 years into one of three categories:

1) Altered nociception
2) Psychological distress/disease
3) Physiological disease of a tissue/neurohormonal system.

Research Initiatives

1) Altered nociception

Central sensitization

Fibromyalgia is diagnosed on the identification of allodynia and hyperalgesia. Both are states of central sensitization in the dorsal horn where spinothalamic tract neurons become sensitized following activity of nociceptive primary afferents (i.e. thinly myelinated Aδ and unmyelinated C fibres). A reduction in the firing threshold of second order neurons results in an increased responsiveness, an increase in receptive field size and a new responsiveness to non-nociceptive fibres.[23]

The N-methyl-D-aspartate (NMDA) receptor, one of the primary types of excitatory synapses found in the central nervous system (CNS) is a potential route to central sensitization. For discussion of the mechanism of action for the NMDA receptor see A.H Chapman, in General Pharmacology.[24] This receptor becomes activated with sustained nociceptive inputs and its properties partially contribute to the phenomenon of "wind up", an increased potentiation of second order neurons wherein additional stimuli cause a sustained increase in responsiveness of the postsynaptic neuron resulting in 60 minutes of increased response from 3 minutes of initiating stimulation.[25] In wind up the dorsal horn neuron becomes responsive to non-noxious stimulation from Aß fibres and other wide dynamic range interneurons, in addition to noxious Aδ and C fibre volley. However it is likely that peptide receptors and excitatory amino acid receptors are both activated to initiate wind up.[23,26]

Evidence demonstrates fibromyalgia patients are experiencing wind up; higher reports of pain than normal controls to brief but repeated thermode contacts, and the size of the response by the fibromyalgia subjects would normally require the presence of wind up and central sensitization.[27]

Following wind up, a rapid change in gene expression within the dorsal horn is significant to the development of chronic pain. Depolarization of the postsynaptic neuron results in the activation of c-Fos, an "immediate early gene" that codes for the protein Fos. Expression of c-Fos may be a marker for a cascade of events that results in adaptive responses by the spinal cord to repetitive or continuous noxious stimulation.[28]

Long-term changes

In chronic nerve injury, Fos expression is seen primarily in the laminae 3 and 4 of the dorsal horn, which are the terminus of the large diameter non-nociceptive myelinated Aß fibres, as well as in other nociceptive laminae (1,2). Enhanced Fos expression may be maintained by low intensity noxious stimulation as well as by stimulation of Aß fibres. Peripheral nerve injury causes sprouting of nerve fibres between the lamina, and within the dorsal root ganglion. The later offers an explanation for the increased sympathetic activity reported in fibromyalgia (i.e. Raynaud's phenomenon).[16] Aß fibres are known to sprout into laminae 1 and 2, but there may be sprouting between other layers as well, possibly as an adaptive response to injury aimed at reducing the effects of such.[24]

Therapies designed to induce descending inhibitory

pathways through low level stimulation of the A fibres (e.g. transcutaneous nerve stimulation) are of limited effect. The increased Fos expression which results from long standing noxious stimuli, may make it intolerable for the fibromyalgia patient to receive tonic stimulation of the Aß fibres at a level sufficiently intense to induce the endogenous anti nociceptive (opioid) system.[15]

Normally, dorsal root neurons are under tonic inhibitory control of descending GABAminergic neurones and blockade by glycine or γ-aminobutyric acid (GABA) receptor antagonists also results in sensitization similar to that which is C fibre induced, however it is still unclear whether disinhibition occurs during windup.[29]

Following peripheral nerve stimulation a number of neuropeptides are released in the dorsal horn that have either excitatory or inhibitory actions.[30] In fibromyalgia substance P is confirmed to be increased two to three fold in the cerebrospinal fluid (CSF) compared to normal, and present in a large proportion of cases (> 85%). There is a relationship between pain and tenderness ratings and progressive increases in substance P levels in the CSF. Oddly in fibromyalgia, this correlates with decreased blood flows found at the caudate nucleus and thalamus even though substance P is a vasodilator.[31] Substance P is modulated by serotonin, which has regulatory functions affecting deep sleep, mood, pain perception by the thalamus, and stress response. Serotonin cannot cross the blood brain barrier, so care should be taken not to consider findings of lowered serotonin in the sera to reflect levels of serotonin in the CSF, which has yet to be measured.[32–34] Neuropeptides also affect the endogenous opioid system, and patients are found to have an increase expression of delta and kappa opioid receptors in the skin, likely due to persistent and severe pain, but without analgesic effects from these receptors.[35]

The mechanisms of central sensitization are not static, nor globally expressed in the central nervous system. Dorsal root changes from continued nociceptive input may result in changes in the thalamic nuclei, causing changes in the cerebral cortex that eventually become independent of the initiating predecessors.[23] One confirmed change is decreased cerebral blood flows in the thalamus and caudate nucleus, which may indicate impaired central inhibition of pain.[32]

2) Psychological distress/disease

One basis for diagnosis is the patient's response to a pressure stimulus. The measurement of which is predominantly by self-reports of pain existence, area, and magnitude. However the patient's experience of pain is more than a report regarding noxious stimulation. The pathways through which a stimulus becomes a pain response (vocalization, avoidant behaviour, rubbing) are not limited to those transmitting a noxious event. A number of psychological processes; comparisons to prior painful events, ranking of the magnitude of the pain, and the patient's social context, affect perception and expression of nociception.[36]

The psychological study of chronic pain is concerned with acquisition, and how mental status or disease plays a role in maintenance of the pain state. Nociceptive alterations may change the reactivity in the higher centers of the nervous system. For example changes have been noted in the cognitive abilities of patients with fibromyalgia. Another likely change is alteration in the reactivity of the neuroendocrine system.[37]

Patients demonstrate an increased sensitivity to stressful influences and act in a manner similar to "sickness behaviour", typified by development of anxiety, depressive tendencies, avoidance of movement, withdrawal, possibly with changes in eating habits and loss of interest in the surroundings. The "stress hyperactivity pain model" supposes that the somatic consequence of stress are displayed in the muscles as bracing and holding behaviours and that pain is a result of peripheral nociception of these events when they occur at a relatively high frequency.[38]

Muscle tension increases can be classically conditioned in patients by the formation of memories for pain. Responses are not general in nature but rather are conditioned to pain relevant stimuli. The formation of these memories has been shown to lead to an increased physiological response when a matching and therefore memory-activating stimulus is presented even at times long distant from initial conditioning.[39]

Personality/Mood Disorders

Other investigations seek relationships involving personality disorders, or mental illness. The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) requires aspects of an individual's personality

traits to be inflexible, pervasive and significantly different from those normally displayed within one's society and culture. In clinical cohorts, rates as high as 50% have been reported for diagnosable personality disorders; considerably higher than in the general population. The Minnesota Multiphasic Personality Inventory (MMPI) can identified homogeneous and replicable subgroups of patients, that demonstrate excessive somatic concern and depression, who are also likely to exhibit pain related behaviours. But note, it is unclear that behaviour interpreted as personality disorder is actually a disorder according to the DSM. These may represent traits that only appear to be consistent with diagnosis of personality disorder when an unfamiliar patient presents to the clinic affected by the distress that chronic pain exerts. The social consequences of experiencing chronic pain along with cognitive and affective components may cause the premorbid character to appear to be suffering from a personality disorder. Screening instruments such as the 12-item General Health Questionnaire (GHQ-12) show many patients are distressed.[40]

In the general population persons seeking care for chronic widespread pain experience more distress, have more mental disorders than persons who do not seek care, and have 300% increase odds of diagnosis for mood (depression and dysthymia) and/or anxiety disorders over persons without pain.[41] But only mood disorders are statistically associated with chronic pain, and studies show conflicting variability for age, gender, and duration of pain as associated parameters. Patients with mood disorders tend toward avoiding problem focused behaviour and social support while engaging in self-blame, but it should be remembered that many patients do not have a psychiatric diagnosis.[42]

Somatization disorder, although rare in the general population, has been reported to be prominent within chronic pain patients seen in clinics.[41] The definition most applicable in fibromyalgia states that "Somatization [is] a ubiquitous and diverse process in medicine, linking the physiology of distress and the psychology of symptom perception."[43] People are likely to express distress in somatic terms rather than in psychological terms as this vocabulary is more readily accessible. But the notion that somatization provides the patient with a socially acceptable way to communicate their psychological distress seems simplified. It removes the notion that central processing of peripheral signals is continuous and the perception of symptoms by the patient is in fact "real".

Cognition

Cognitive strategies commonly employed for coping with chronic pain are diverting attention, praying, hoping, catastrophizing, ignoring, and positive/negative self-statements. The variance in psychological distress for chronic pain patients can be accounted for in 50% of cases by helplessness alone, suggesting that it is more important to reduce negative self-statements and catastrophizing than to increase positive self-statements in order to enhance adaptive coping. [23] Cognitive factors are important in the perception and subsequent reaction to pain. Patients who poorly understand the source of their pain or who find it mysterious use maladaptive over adaptive coping strategies compared to patients who believe that their pain will be short lived and who understand the cause of their pain. Perceived self-efficacy is an effective coping strategy providing there exists a belief in having the requisite skills to apply. But self-efficacy is a changeable state dependent on emotional and psychological arousal, performance, vicarious experience and verbal or social persuasion. In fact all coping strategies are changeable. The relationship between sleep, pain and attention to somatic symptoms has been established and shows a reciprocal interaction between nightly sleep quality and daily attention to pain that is independent of the pain's intensity, suggesting that poor sleep undermines efforts to use cognitive coping strategies.[44]

3) Physiological disease of a tissue/neurohormonal system

Sleep

Identification of a measurable abnormality, episodic arousal (7.5–11 Hz wave pattern on electroencephalography) during stage 4 (non-REM) sleep, gave hope that a more widespread pathological process could be found.[45] Although transient fibromyalgia-like symptoms can be induced in health volunteers by recreation in the laboratory setting, it is not clear that the sleep anomaly is a primary etiological factor. Cyclobenzaprine, a commonly employed tricyclic antidepressant therapy has little effect on the sleep anomaly, suggesting that it is a secondary finding.[46] Base line ratings of alpha-delta sleep have not been

correlated with symptom severity and are not predictive of clinical response to treatment with amitriptyline.[47]

Investigations into other sleep disorders, including sleep apnea syndrome, have found no significantly associated pathologies.[48,49] The significance of lessened overnight hemoblogin oxygen saturation (SaO_2) is yet to be determined.[49] Sleep's role in the symptomatology of fibromyalgia is still a research focus because of the diagnostic role of fatigue and non-restorative sleep. Increasing attention is being paid to the diurnal patterns of neuroendocrine function and the affect that the alpha non-REM sleep anomaly may play here.[50]

Hypothalamic-Pituitary-Adrenal Axis

Growth hormone (GH) therapy has demonstrated significant improvement in symptoms, although without complete remission. Approximately 80% of the daily secretion of GH is released during stage 4 sleep; the other major stimulus for secretion being exercise.[45] GH stimulates production of, and works in conjunction with, somatomedin-C (insulin related growth factor-1) to control muscle homeostasis (mass and muscle function). Sleep disruptions are related to decreased secretion of somatomedin-C and fibromyalgia patients are found to have a significantly lower level of somatomedin-C as compared to controls.[48,51] GH abnormalities are likely to be a secondary phenomenon, for two main reasons. Firstly not all patients display deficiency and secondly normal somatomedin-C levels tend to fall over time when patients are followed with repeated measures of this hormone.

Evaluation has also focused on the hypothalamic-pituitary-adrenal (HPA) axis and the role that it plays in regulation of growth hormone.[52] It is known that an intact HPA axis is required to induce an analgesic response to stress in pain experiments on rats.[53] Fibromyalgia patients display a paradoxic pattern of HPA axis disruption. They exhibit an attenuated adrenocorticotropic hormone response to corticotrophin releasing hormone while at the same time exhibiting a blunted cortisol response to adrenocorticotropic hormone release.[15,32]

All of the questions regarding the association between the neuroendocrine axis, GH, stress and the sleep anomaly have not yet been answered. But it cannot be overlooked that GH is associated with a wide array of fibromyalgia's symptoms, including low energy, poor general health, decreased lean body mass, muscle weakness, reduced exercise capacity, impaired cognition and dysthymia.

Muscle

Muscle was the primary focus of early investigation. Patients occasionally display a slight tremor, with limited and slow movements (bradykinesia) along with the pain of muscle work. This suggests some similarities to akinetic movement disorders such as Parkinson's disease, and could be a result of change in the complex projections of the basal ganglia.[54]

Functionally patients have been estimated to be approximately as aerobically efficient as normal sedentary controls matched for age, although 80% are below the average level of aerobic fitness defined by the American Heart Association.[55]

They also exhibit a significantly lower maximal effort of knee and elbow angular velocity testing for muscle strength. Lower central activation of muscle is not entirely attributable to psychopathology and exercise induced pain could be partly explained by alteration in the neuroendocrine reactivity during exercise.[56] Muscle biopsy showing microtrauma within the muscles of patients is no different from biopsy of normal controls.[57] It is suggested that mild microscopic abnormalities, including "moth-eaten" type I fibres, "ragged-red" fibres, myofibrillar lysis, papillary projections, subsarcolemmal glycogen and interfibrillar lipid, are frequent phenomena that result from various daily mechanical actions, psychologic stresses, ischemia or subclinical injury of muscle spasm.[58]

Fibromyalgia patients are unlikely to be suffering from defects in mitochondrial oxydative enzymes, but they do show abnormal glycolysis, which in part can account for subsarcolemmal glycogen, muscle weakness, reduced ATP and hypoxia.[59] No excess production of serotonin or other neuropeptides has been found in patient's muscle tissue that would cause the induction of peripheral pain.[60,61]

A synthestized view of fibromyalgia

The action of wind up and the resulting sensitization of the dorsal horn is very effective in preventing subsequent damage to an injury during early healing phases and should not be considered a pathological process. Widening of the receptive fields protects from further injury by heightening the response to touch in anatomically close

but non-injured areas, allowing the quickest recovery. Granted, when no physical trauma is identified the activated mechanism of nociception would appear to be a disease process, but can there be adequate maintenance of wind up without down regulation of inhibitory mechanisms from the higher centers, where factors other than those involved in nociception come into play?

Emotion, learning and memory are complicated yet plastic processes involving diverse areas of the encephalon, and affecting a myriad of other brain functions. Learned behaviours with regards to pain may be new or cover a lifetime of experiences. The beliefs and coping skills of individuals will also differ between persons for a given situation and individually may differ over a variety of stressful stimuli. In this complex role the higher brain systems and functions exert a dual affect. One affect would be to the dorsal horn via inhibitory functions that could explain the initiation of nociceptive cascade in the absence of injury. A second would affect the neuroendocrine system and would have implications in the periphery.

The HPA axis plays an important part in the body's response to infection, injury, sleep deprivation, emotional stimulation, and in metabolism and water balance. Changes in neuroendocrine regulation caused by conditioned/learned behaviour could have whole body consequences. Reports of fibromyalgia being initiated by stressful events (post traumatic stress disorder) or following a period of poor health (Epstein-Barr virus infection) can be explained by initiation at the neurohumoral axis. Changes in muscle homeostasis involving decreased connective tissue and muscle mass are one possible expression of HPA axis alteration. Other expressions could account for many of the other unexplained syndromes including chronic fatigue, restless legs, and irritable bowel. Of course general health and genetic factors also have a role to play in the expression of pain.

The soft tissues are still the likely primary source of stimuli to the sensitized dorsal horn, causing activation of nociceptive processes and central responses to those stimuli. The locomotor tissues are a rich source of sensory fibres and when responding at a normal depolarization threshold, in the sensitized individual, would be experienced as noxious. In injury the C fibre volley of muscle tissue can evoke a strong central hyperexcitability, and one that lasts longer than those produced by cutaneous C fibres.[23] It may be that muscle tissue sends a volume of information to the CNS which overrides information coming from other tissues and therefore is registered by the patient more readily. Conversely patients may have more cognitive constructs with which to vocalize for muscle pain than for any other system. Their deconditioned status may be a result of limited muscle use resulting from pain or they may have an inability to reciprocally affect the neuroendocrine system, not having adequate ability to stimulate growth hormone production via exercise. Growth hormone production is normally decreased in advancing years, and the lowered levels of somatomedin-C in patients have been suggested to equal an age related changes of 33 years.[52] Recalling that the prevalence of fibromyalgia also increases with advancing age it has to be wondered if its symptomatology is being created by normal processes which when initiated in combination results in the experience of chronic pain.

In this modification of the "biopsychosocial model", fibromyalgia is presented as a cyclic condition, with many channels serving as entry points for influential factors to initiate chronic pain. No one component is able in isolation to cause the complex presentation of clinical symptomatology, however once initiated the components acting in concert present as a disease state. Each component of this closed cycle could contribute symptoms that make up the clinical presentation of fibromyalgia. The entire cycle is represented in Figure 1.

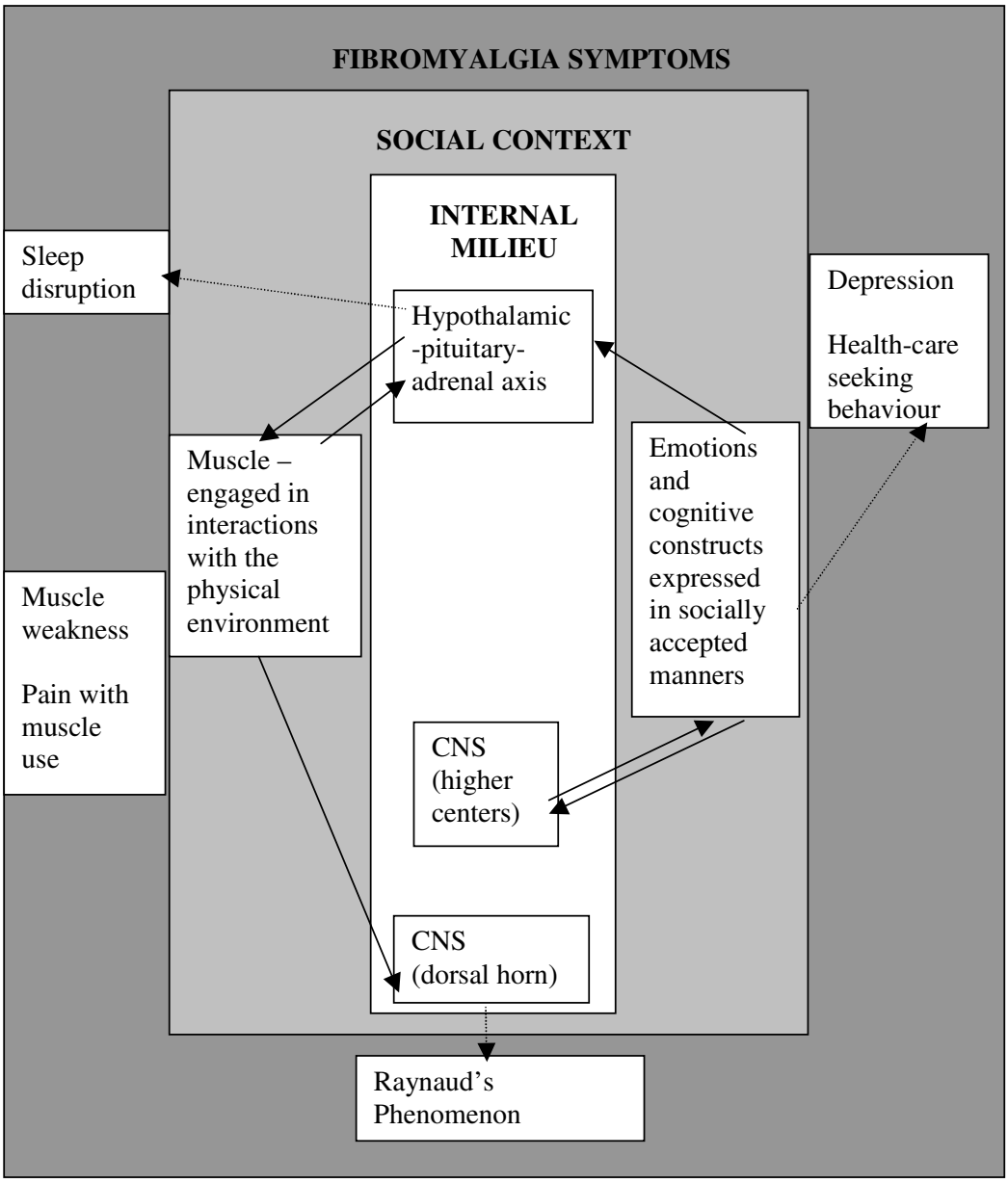
Treatment

Treatments encompass a wide variety of pharmacological and non-pharmacological intervention and patients often utilize one or more of these therapies at any time. Pharmacological therapies include antidepressants, analgesics, and miscellaneous chemical agents that facilitate, inhibit or replace naturally occurring products. Non-pharmacological therapies include biofeedback, exercise, hypnotherapy, cognitive behaviour therapy, and "alternative medicine/complimentary therapies".[62,63]

Pharmacological interventions

The vast majority of fibromyalgia patients utilize at least one drug at any given time (90% +) and try a much larger array of drugs over a multi year period. About one third of patients using antidepressants will achieve improvements in the short term that wane over time. The same

FIGURE 1



A SYNTHESTIZED VIEW OF FIBROMYALGIA: Fibromyalgia is presented here as a cyclic condition, with many channels (solid arrows), each which can serve as an entry point for influential factors that may initiate the cycle in motion. Once initiated the components acting in concert are presented as chronic pain. Each component of this closed cycle could result in symptoms, that together sum to the clinical presentation of fibromyalgia syndrome.

percentage is likely to have used non steroidal anti inflammatory drugs (NSAIDs) even though both naproxen and ibuprofen have been found to be no more effective than placebo when administered alone.[64] Other chemical compounds (Ketamine, S-adenosylmethionine, lignocaine, human growth hormone, interpheron-alpha and gama-hydroxybutyrate) are being investigated, but their long term efficacy and practicality for administration remains in question.[65,66,67,68,51,69,70]

Non-pharmacological interventions

Non-pharmacological treatments are associated with improvements in physical status, self-report of symptoms, psychological status and daily functioning. These treatments include the physically based (e.g. aerobic exercise, muscle strengthening, manipulation and stretching), the psychologically based (biofeedback, relaxation, and cognitive-behavioural), and combination therapies. Non-pharmacological treatments have been shown to produce significant effects when used in conjunction with pharmacological treatments (i.e. multidisciplinary/combined therapy), and interestingly when utilized alone they produce a greater relative effect size.[21]

Cognitive behavioural training has been shown to be effective.[64] Here patients focus on the role of stress in the maintenance of the emotional and physical symptoms, and practice strategies for improving self-management of pain and distress, which include relaxation, cognitive restructuring and learning problem solving skills. These practices help to foster self-efficacy through information, practice and mastery, in order to supplant feelings of passiveness, helplessness, and incompetence.[71] They are best proven to predict improvement in pain, depression and self reported pain behaviours.

Exercise therapies have a decrease in adherence once supervised programs end, and therefore treatment strategies need to promote ongoing activity. Large drop out rates for exercise therapies are reported due to patients experiencing increased pain. Programs should try to emulate the most successful trials to date, which often employ moderate training levels (60%–70% VO2 max), progressively increasing work loads, manageable exercise times and a reasonable number of exercise periods per week.[72]

Physically based treatments are not shown to produce significant improvements in daily function if they are administered alone, and therefore should be incorporated into a combined protocol in order to increase effectiveness.[21] Combination therapies could include manipulation, relaxation training, exercise interventions, and patient education of self-management.[73] Patients who receive combination therapies are more likely at long-term follow up to have maintained improvements in self-efficacy for function.[74] Individual differences and preferences of patients may play a large role in the efficacy of treatment outcomes as well. Self-efficacy measures may really represent measures of expectancies of outcomes. This may result in patients who have low beliefs of self-efficacy discontinuing their therapy early because failure is anticipated.[74] The expectation of effectiveness and a treatment's actual effectiveness may differ, resulting in patients and practitioners acting on beliefs rather than actuality.

Patients show a strong preference for many alternative non-allopathic therapies.[75,76] It would seem that the reverse should be true, avoiding manipulative techniques where treatment would be expected to stimulate muscle and joint nerve endings. But more than 90% of fibromyalgia patients seek alternative therapies (i.e. acupuncture, chiropractic, homeopathy, physiotherapy, psychotherapy). Although efficacy of these therapies is not confirmed, patients express satisfaction with them and often seek treatment of their own accord.[62] No high quality studies have been conducted to address whether patients who seek alternatives are able to limit escalation in their symptoms severity, or are able to prevent the development of disability in comparison to patients who do not seek non-physician therapies. Thus it is possible that worsened outcomes, and more physician interventions are avoided by the use of these therapies.[76] Chiropractors are in a very good position to provide "alternate" care plans, but these therapeutic interventions should be of a combination format.

The development of multidisciplinary treatment protocols has arisen in order to capture the most effective of the therapies and to provide them in one environment, by a wide range of specialized practitioners. Again treatment aimed at the multiplicity of symptoms of fibromyalgia suggests a multimodal program, but these may be expensive to provide and it is hard to assess their effectiveness due to a lack of standardized measures.[73]

Outcomes

No valid instruments are available to evaluate disability from fibromyalgia in the work place, or litigation setting.[7] Pain and fatigue are difficult to measure objectively and the correlation of pain and disability is only fair. Many commonly employed measures do not have application in determining disability. Neither the The McGill Pain Questionnaire, nor The Health Assessment Questionnaire is appropriate for this task.[77] The Fibromyalgia Impact Questionnaire (FIQ) likely underestimates functional impairment.[78] More recently developed, the Fibromyalgia Health Assessment Questionnaire (FHAQ) requires testing for its sensitivity to change in order to prove its validity.[79]

Measures need to cover a diverse spectrum of types, other than ones based upon self-report.[80] Tender point counts based on the ACR criteria would continue to provide high specificity and sensitivity, while meeting the requirement of objectivity if dolorimetry is employed.[81,15] Patients have been found to be resistive to progressive tenderness with repeat examination using dolorimetry, and mechanical dolorimeters have been found to be more reliable in detection of tenderness at fibromyalgia tender points than electronic models.[82,83] The Multidimensional Pain Inventory (MPI) has been updated, improving the classification of chronic pain patients into subgroups based on psychosocial adaptation.[84] Adaptation profiles are relatively independent of physical findings and classify persons into coping groups, some of whom may not require all of the components of a multidisciplinary treatment program.[85]

Measurement of severity of pain by self-report may be predictive of prognostic outcomes, however it is an insensitive measure for treatment efficacy.[73] If central sensitization results in long standing CNS changes that become independent of any initiating states and these changes finally result in new memory and learned behaviours, then self-reports of pain reduction may be attenuated due to the inherent plasticity of the central nervous system. It may be that in order to effect deconditioning, and/or to create new learning and memory for previously painful stimulation, very long periods will need to be employed in order to record a change. "Wind down" may be very slow, as pain-inhibiting processes slowly build, eventually equal and then surpass pain-initiating processes.

Conclusion

Patients arrive at the clinician's door for a variety of reasons, many which may have little to do with the number of tender points, or the level of fatigue. Often presenting as their coping mechanisms begin to fail, or when distress reaches a level at which the patient is no longer able to cope with their symptoms.

Fibromyalgia continues to be resistive to treatment in any clinical setting. Patient responses to treatment will differ, as will their outcomes. In the compensation and speciality care setting outcomes are poor, leaning towards unremitting. However in the wider community the prognosis is variable with occasional remission of symptoms even without treatment.[7] Successful treatment has been limited to helping patients improve their ability to cope with, but not to eliminate the tender points.

Currently care should continue to be patient driven focusing on pain relief, limitation of chronicity, management of maladaptive coping behaviour, and rehabilitation of normal physical functioning. With improved identification of patient subtypes, therapies should become more successful in responding to each patient's special needs. Improved recognition of the patient's expectations and beliefs will help to identify patients who are ready to undertake a significantly increased responsibility for their care. Some patients can receive effective therapy from one practitioner through a comprehensive care program. This is the type of program that is easily implemented within a solo chiropractic practice. Less responsive patients may continue to require a multiplicity of practitioners provided in a multidisciplinary care setting.

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The 4th Chiropractic Research Symposium will be held September 17–19, 2004 in Montreal. The call for abstracts will be issued in June by Dr. Jean Boucher PhD. Young investigators are eligible for an award sponsored by the Canadian Chiropractic Association.

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