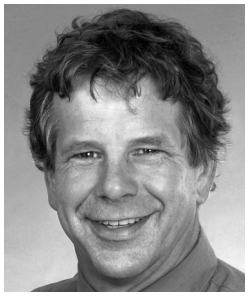
Editorial

A Pain Psychologist's View of Tenderness in Fibromyalgia



In this issue of *The Journal* Harth and Nielson provide an excellent review of the use of the tender point (TP) criteria for the diagnosis of fibromyalgia (FM)[1]. They acknowledge that the tender point criteria assess an unknown combination of tenderness and psychological distress. These authors also note that pain psychologists have developed improved methods of measuring pressure pain sensitivity, but argue for the continued use of the simple tender point criteria on the basis of simplicity, brevity, and cost. This is a reasonable conclusion with the present evidence and technology. With these issues at hand, what is the next step or steps? What if we pain psychologists develop an inexpensive, simple, and fast measure of true tenderness that could be applied in the clinic? What would it tell us?

For one thing, this measure would swell the ranks of those who suffer from chronic widespread pain (CWP). Harth and Nielson point out that without the TP criterion, 10% to 15% of the population would present with CWP. As a pain psychologist, understanding and treating this pain is the primary challenge. If we cull this group by the TP criterion, we get a subset of patients who are tender and/or distressed. Without disagreeing with the broad clinical utility of TP in tending to select those who may be in greater need of treatment, this approach leads to a clinical concern for those with CWP who do not meet the TP criteria. There is also a concern that TP are easily influenced. Recent data from our group suggest that only a small fraction, perhaps one-sixth, of a cohort of 97 patients with FM were actually noticeably tender[2]. Distress likely had some influence, but an additional hypothesis is that some patients with FM may be invested in having the diagnosis and through the Internet, written media, and word of mouth, have simply learned what to say in a TP examination. Thus there are a large number of patients in pain who fail to obtain, or perhaps shouldn't have, the diagnosis of FM. Are they any different in any meaningful way from those who are truly tender?

There are a number of possibilities that need to be considered. The first is the unified process implied by the American College of Rheumatoloty criteria, in which FM reflects a pathology that augments pain sensitivity, and this augmentation is reflected in both spontaneous CWP and pain evoked by blunt pressure. This is the "linked" scenario, in which both spontaneous and evoked pain abnormalities are symptoms of underlying pathology. However, since the accumulated data suggest that only a fraction of the CWP population is tender, other scenarios suggest that the CWP and tenderness components may be independent processes. In addition, the recent findings of tenderness in other pain disorders[4,5] suggest that tenderness may not be a unique feature of FM but rather a general feature found in a subset of patients with many types of pain disorders.

In contrast to the linked scenario, there are possibilities with varying associations between tenderness and CWP. In the "independent but relevant" scenario, tenderness is a genetically determined trait that can be present in any spontaneous pain condition or in persons who are free of spontaneous pain. When both tenderness and CWP coexist, there could be synergistic action that makes the whole worse than the sum of the parts, leading to a clinical interest in determining tenderness in CWP. In the alternative "independent and irrelevant" scenario, tenderness may have no bearing on the diagnosis or treatment of FM.

In a related "risk factor" scenario supported by a longitudinal study of orofacial pain[3], the presence of genetically determined tenderness may be a risk factor for subsequent development of CWP. In this scenario there may be some linkage between the mechanisms of tenderness and CWP, in that the presence of one predisposes the other. This factor may be relevant for diagnosis and treatment of the subsequent CWP. Alternatively, it is important to point out that the mechanisms mediating tenderness and CWP could be independent, in which the presence of tenderness would

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have no bearing on either the diagnosis or treatment of the CWP. An example of such an effect is provided by the relation of cigarette smoking and lung cancer. Smoking is clearly a risk factor for developing lung cancer. Yet it is neither completely sensitive nor specific; it is possible to develop lung cancer if you have never smoked, and possible to smoke 3 packs a day and never have lung cancer. Most importantly, once you have developed lung cancer, the treatment may be the same whether you were a smoker or not. Smoking influences the probability of acquiring cancer, but once acquired does not influence the mechanism of cancer or its treatment. In a similar vein, the presence of tenderness may increase the probability of acquiring CWP, but once acquired does not influence the treatment of CWP. In this case, it may be more accurate to downgrade the previous tenderness from risk factor to "risk marker" for CWP (W. Maixner, personal communication).

In yet another "developed tenderness" scenario, CWP could lead to tenderness in a genetically determined subset of the CWP population. This could be a feature that determines the nature of the CWP (i.e., causing FM) or it could be a general effect that is independent of the mechanism of CWP and its treatment. It may also be specific to FM or, as recent data suggest, a nonspecific effect found for a number of chronic pain syndromes. As with the risk factor scenario, the developed tenderness effect could influence the mechanism of the spontaneous pain and its treatment, or have no influence.

As a pain psychologist currently focusing on tenderness in FM, I find the independent and irrelevant scenario and the independent variants of the risk factor and developed tenderness scenarios troubling because in these situations the presence of tenderness is not important for either diagnosis or treatment. It would be ironic if my own studies of tenderness rendered these studies irrelevant to FM. Fortunately, there may always be some relation. For an example common to many potential genetic discoveries, identification of risk markers may be important for application of novel prophylactic treatments that prevent rather than manage the disease. These markers would provide a "wake-up call" similar to evaluation of cholesterol concentrations or to an attack of mild angina.

While further work may establish the relevance of tenderness, such investigations should not lose sight of the primary goal. CWP is a source of immense suffering that brings the patient to the doctor. The sensitivity to physical stimulation may be relevant. Other symptoms such as mood or cognitive function may be more closely aligned with spontaneous pain and thus more important for diagnosis and treatment. Once established, this list of moderator variables will be useful adjuncts to the primary goal of diagnosing and treating chronic widespread pain.

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