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## **The Brady/Schneider Proposal: A Misdirected Reclassification of Fibromyalgia**

By John Lowe, MA, DC

In October 2001, the *Journal of Manipulative and Physiological Therapeutics* published a proposal for a new way of classifying fibromyalgia syndrome (FMS).<sup>1</sup> The authors of the proposal, David Brady,DC, and Michael Schneider,DC, wrote that the original concept of FMS as "one grand, all-encompassing clinical syndrome" was flawed. They proposed to rectify the putative flaw by henceforth dividing FMS into two different forms: **classic** and **pseudo**.

The most distinguishing symptoms of FMS are chronic widespread pain and abnormal tenderness of predetermined anatomical sites. Schneider and Brady wrote that in classic FMS, these symptoms result from abnormal sensory processing in the central nervous system (CNS). They wrote that in pseudo FMS, the symptoms are features of a variety of undiagnosed disorders other than classic FMS.

On the surface, the Brady/Schneider proposal may seem a practical revision of clinical practice - a revision that might enable clinicians to distinguish FMS from other disorders with similar symptoms and provide, or direct patients to treatments appropriate to the diagnoses. I believe, however, that crucial errors in the proposal render it of no value to clinicians. Below, I consider several errors, in descending order of importance.

### **Not a Novel Classification Scheme**

Schneider and Brady wrote, "We present a **novel** (emphasis mine) classification scheme that differentiates 'classic FMS' from various subsets of 'pseudo FMS.'" The authors' scheme is not novel. Yunus preceded Brady and Schneider by 23 years in urging clinicians to distinguish FMS (then called "fibrositis") from other disorders that produce similar symptoms and signs.<sup>5</sup> Since then, scores of other researchers, including Travell and Simons<sup>6</sup> and myself,<sup>2</sup> have echoed Yunus' entreaty. How this widely publicized advice - which

the Brady/Schneider proposal reiterates - could have escaped the authors' attention perplexes me.

### **Unnecessary Use of Synonyms**

What is novel about the Brady/Schneider proposal are the terms the authors propose: "classic" and "pseudo" FMS. These terms, however, are only synonyms for other terms FMS researchers have traditionally used. Brady and Schneider's proposed term "classic FMS" is synonymous with the long-used "primary FMS." Researchers began using this modifier some 35 years ago to refer to FMS symptoms that could not be attributed to another disorder. The authors' proposed term "pseudo FMS" is synonymous with "secondary FMS." FMS researchers began using this term at least 18 years ago to refer to FMS symptoms caused by other disorders.

As an FMS researcher, I see no advantage in replacing the traditional terms "primary" and "secondary" with "classic" and "pseudo." Nonetheless, in deference to Brady and Schneider, I will use their synonyms in this critique.

### **Error Regarding the Treatment of Classic FMS**

Brady and Schneider suggest an advantage in distinguishing patients with classic FMS is that they generally respond well to low-dose antidepressant and anxiolytic drugs, biofeedback, and psychotherapy. A positive response to these therapies, however, has not distinguished any subset of FMS patients. Contrary to the authors' statement, these treatments are **not** effective with FMS patients.<sup>2,8,9,12,13</sup> Scrutiny of published efficacy studies shows that the treatments provided only transient statistically significant improvement on a few measures of FMS status. But the improvement was not **clinically** significant - meaning that patients continued to meet the criteria for FMS and to suffer from its symptoms. The utter failure of these treatments to relieve FMS patients' suffering has strongly contributed to the recent crumbling of the rheumatology paradigm of FMS.<sup>2</sup>

### **Error in the Treatment of Pseudo FMS**

Many disorders' symptoms, especially chronic pain and fatigue, overlap those of FMS. As Brady and Schneider note, clinicians commonly fail to diagnose the other disorders. Instead, based on the overlapping symptoms, the clinicians often mistakenly give patients a diagnosis of FMS.

But some patients with other disorders, such as rheumatoid arthritis, osteoarthritis, Lyme disease, systemic lupus erythematosus (SLE), Chiari malformation, and spinal stenosis, also meet the criteria for FMS. In studies that showed the concurrence of FMS with other disorders, highly trained experts took great care to make sure patients met the criteria for both FMS and the other disorders. Among patients with Lyme disease, eight percent met the criteria for FMS,<sup>31</sup> and among SLE patients, 22 percent met the criteria.<sup>32</sup> Recently, neurosurgeons reported that many patients with brain stem or cervical cord compression due to Chiari malformation or cervical stenosis meet the criteria for FMS.<sup>10,11</sup>

When these other disorders are effectively treated, however, the patients continue to suffer from FMS. When the symptoms and signs peculiar to Lyme disease resolve with antibiotic therapy, FMS symptoms usually continue.<sup>33</sup> Similarly, when SLE symptoms and signs resolve with corticosteroid therapy, FMS symptoms usually become more clearly apparent.<sup>33</sup> And while decompression surgery relieves FMS patients' symptoms of brain stem or cord compression, the surgery does not free the patients from FMS.<sup>10,11</sup>

That patients continue to suffer from FMS following effective treatment of their other disorders compels a conclusion: Symptoms of various disorders overlap those of FMS, but FMS itself has another cause.

### **Disregard of Crucial Thyroid-Related FMS Research**

Brady and Schneider discussed hypothyroidism at length in their proposal. Unfortunately, they completely neglected crucial studies of thyroid disease and FMS, leaving them with a misguided conception of FMS.

A substantial body of credible scientific studies by several research teams indicates that nearly 90 percent of FMS patients have underlying thyroid diseases.<sup>29</sup> These include primary and central hypothyroidism and cellular resistance to thyroid hormone.<sup>7,15,16,17,18,19,20,21,22,23,24,25</sup> It is important to note that most patients' FMS symptoms caused by thyroid disease are compounded by other metabolism-impeding factors. The most common factors are poor diet, nutritional deficiencies, poor physical fitness, and metabolism-impairing drugs.<sup>2</sup>

When FMS patients receive effective treatment for hypothyroidism or thyroid hormone resistance, most are fully and lastingly freed from their FMS. Several open, but systematic trials, and several blinded studies have shown that metabolic treatment completely relieves most patients' FMS.<sup>3,7,23,24,25,26,27,28</sup> Following the treatment, patients no longer met the criteria for FMS. A one-to-five-year follow-up study showed that patients' recovery lasted long-term.<sup>26</sup>

Why Brady and Schneider ignored this body of evidence when formulating their proposal is baffling. According to their scheme they would classify patients with FMS, due to hypothyroidism or thyroid hormone resistance, as having pseudo FMS. Twice, they stated that reports of recoveries of patients with pseudo FMS are "**anecdotal** success stories of FMS remissions and cures." (Emphasis mine.) Their characterizing our published reports as anecdotal astonishes me, in view of the rigorous scientific standards to which we at the Fibromyalgia Research Foundation hold ourselves. Several of our studies in which patients fully recovered were double-blind, placebo-controlled crossover studies. I assume that the authors failed to read our published reports, despite at least one of them having them available in his library.

The importance of the authors' failing to consider our reports cannot be overemphasized. Consideration of previous research findings is crucial to a meaningful formulation of new ideas in a research field. Failing at this crucial step in scholarship is common among medical researchers.<sup>30</sup> The failure dooms many of their new ideas through misdirection. This is the plight of the ill-fated Brady/Schneider proposal. Results of the crucial thyroid studies argue against their belief that FMS is not "one grand, all-encompassing clinical syndrome." To understand why this is so, one must consider the implausible model Brady and Schneider use to explain classic FMS.

### **Probable Nonexistence of Classic FMS**

One might ask if 90 percent of FMS is underlain by thyroid disease and synergistic metabolism-impairing factors, what about the other 10 percent of FMS patients - those who do not have hypothyroidism or thyroid hormone resistance? Do these patients have the classic FMS proposed by Brady and Schneider?

The authors explain classic FMS with the "CNS hypothesis" propounded by rheumatologist I. Jon Russell. They state that some yet undiscovered abnormality of the CNS causes abnormal sensory processing within the CNS. The abnormality supposedly accounts for patients' widespread pain, abnormal tenderness, and all other features of FMS. The CNS hypothesis, however, is not scientifically credible; it is refuted by two lines of evidence.

First, a necessary corollary of the CNS hypothesis states that any peripheral features of FMS do not have peripheral causes, but are secondary to the undiscovered CNS abnormality. No proposed CNS abnormality, however, can plausibly explain the many objectively verified peripheral abnormalities in FMS patients. Among these peripheral abnormalities are an increased density of alpha2-adrenergic receptors on platelets; high serum hyaluronic acid; low serum hydroxyproline; low serum and urinary ratios of pyridinoline to

deoxy pyridinoline; low platelet serotonin; and cutaneous vasoconstriction.<sup>2</sup>

The second line of evidence is the far greater explanatory power of the thyroid-disease hypothesis of FMS. As I recently massively documented, hypothyroidism and thyroid hormone resistance credibly explain all the peripheral **and** (my emphasis) central abnormalities of most FMS patients.<sup>2</sup> By comparison, the CNS hypothesis is inadequate and incomplete. Moreover, treatments based on the CNS hypothesis have failed to free patients from FMS, while the treatment derived from the thyroid-disease hypothesis is highly effective in fully and lastingly relieving patients of FMS.

About 10 percent of FMS patients do not appear to have hypothyroidism or thyroid hormone resistance, but they nonetheless have the same symptoms and signs of hypometabolism as FMS patients with thyroid disease. The identical findings in FMS patients with and without thyroid disease suggest that in both groups the same central and peripheral metabolic pathways are impaired. In the group with thyroid disease (90 percent), the impairment results largely from understimulation with thyroid hormone; in the other group (10 percent), it results from some unknown metabolic lesion that affects both central and peripheral tissues.

If the metabolic impairment that causes FMS symptoms and signs is common to all patients who meet the criteria for FMS, the disorder - despite the Brady/Schneider proposal - constitutes "one grand, all-encompassing clinical syndrome." If this is the case, terms used to designate classes of FMS patients are superfluous.

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