Letters to the Editor

Systematic treatment of chronic pain with antidepressants

To the Editor:

I have read with interest the recent article on the above subject by Dr. D. Blumer and his associates (1). Because of the nature of their article, a few aspects of chronic pain were not alluded to that are of interest to internists and practising physicians. I would therefore like to add some comments on the subject of fibrositis, fibromyositis, and "muscular rheumatism."

The fibrositis syndrome is a common, chronic pain problem. It may be primary or secondary and is commonly associated with disabling pain. Many believe that the "fibrositis" syndrome is a disorder of pain modulation. Patients who meet pre-defined criteria complain of sleep disturbance and intensification of pain, stiffness, and fatigue upon awakening. Often, a major change in sleep habits may be identified if the syndrome followed upon a single dramatic event. It has been suggested that the pain or an underlying state of tension causes the sleep disturbance (2).

The studies of Moldofsky, et al (3) and Moldofsky and Scarisbrick (4) have provided evidence for a specific disturbance in sleep physiology, operating as a factor in the pathogenesis of pain in this syndrome. Their subjects showed an overnight increase in measures of muscle tenderness and also a coincidental disturbance in non-rapid eye movement (non-REM) sleep. They also inferred from their studies that the spontaneous occurrence of alpha intrusion into the slow non-REM rhythm of the "fibrositis" subjects was due to an endogenous arousal response. The disturbed sleep, pain, fatigue, and emotional distress may then become locked into a self-perpetuating cycle.

The disturbed sleep physiology seems to account for the perceived relationship between depression and anxiety in this syndrome and thereby lends itself to logical therapy. The sleep disturbance is little helped by barbiturates or benzodiazepines because of their failure to abolish alpha intrusion and thereby restore sleep. Tricyclic antidepressants such as amitriptyline or imipramine can reduce alpha intrusion with effects better tolerated over prolonged periods. While it is tempting to speculate that a fundamental abnormality of absolute or relative deficiency of endorphin could account for the pain, direct experimental support for its central role has not yet been forthcoming (2).

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References

Reply to Dr. Venkatasubramaniam's Letter

Fibrositis is viewed by many rheumatologists as a somatic disorder, a form of nonarticular rheumatism, even though it is merely characterized by soft tissue pain with some trigger points and by definition lacks any of the signs of a rheumatic disorder. Many rheumatologists consider patients with fibrositis as depressed. The studies of Moldofsky and his group quoted by Dr. Venkatasubramaniam, providing evidence for a specific disturbance in sleep physiology associated with fibrositis, are of considerable interest. The alpha-delta sleep observed in fibrositis patients, however, is also characteristic of individuals with depressive disorders. It was first described as a finding in the sleep of depressed patients by Hauri, et al (1). Fibrositis may be viewed as a form of the "pain-prone disorder," which we consider a
variant of depressive disease. The response of patients with fibrositis to antidepressants would not be surprising. It would be of interest to us to evaluate patients diagnosed as suffering from “fibrositis” in order to establish if they indeed present with the entire range of signs and symptoms characteristic of the pain-prone disorder.

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Active Tuberculosis First Diagnosed at Autopsy
To the Editor:
Active tuberculosis that is not recognized clinically represents an obvious public health threat, especially to those in close contact with the undiagnosed case, but also to hospital personnel and to pathologists who are called upon to perform postmortem examinations.

In both 1960 and 1977, the U.S. Center for Disease Control, Tuberculosis Control Division, reported that 4.8% of all cases of active tuberculosis were first registered at death (1,2). This statistic is ambiguous, as it represents a mixture of cases clinically known and reported only at death and cases unknown until autopsy.

For economically developed Western nations other than the U.S., the frequency of clinically undiagnosed active tuberculosis discovered at autopsy varies from 0.1% to 0.85% of all autopsies (3-10). The recent medical literature does not contain a comparable study from within the U.S.

We wish to report that at Henry Ford Hospital, Detroit, Michigan, the autopsy incidence of clinically undiagnosed active tuberculosis is 0.27% for the ten years from 1970 to 1979 (13 cases among 4,797 autopsies). Morphologically, there were two cases of localized fibrocaseous pulmonary tuberculosis, four cases of fibrocaseous pulmonary tuberculosis with dissemination, one case of caseous pneumonia, five cases of miliary tuberculosis, and one case of isolated one-organ extrapulmonary tuberculosis. The involved patients averaged 64.5 years of age and were frequently afflicted with other debilitating diseases, four with disseminated neoplasm, two with chronic renal failure, two with cardiovascular problems, and three with alcoholism. The average length of hospitalization before death was 15 days.

The significance of this letter is to serve as a reminder that while the number of new cases of tuberculosis has declined in the U.S. (37,137 new cases in 1970 and 27,669 new cases in 1979), a small percentage of cases still remains undiagnosed until autopsy. Tuberculosis should always be considered in diagnostic problems in the elderly, debilitated, and in persons with neoplasm, alcoholism, and chronic renal failure.

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References