"Pseudodementia" needs a third look. Always a "soft" diagnosis, it has never had objective, explicit diagnostic criteria or a spot in an official nomenclature.

Although the prevalence of pseudodementia is not as high as early studies suggested, it is far from trivial. Early studies suggested that up to 15% of patients with dementia had one of the reversible types and that depression accounted for about half the reversible dementias. Although depression is still considered among the most common causes of reversible dementia, recent studies, in which diagnostic criteria for dementia are applied and patients closely followed, suggest that the prevalence of truly reversible dementia is far less than thought and that only 0.5% to 1% of patients with dementia have a fully reversible pseudodementia. Given the fact that even a cursory mental status examination usually allows a clinician to distinguish pseudodementia from dementia (Table [not available online]), we would expect to find few pseudodementia patients among those who have actually been given a diagnosis of dementia. On the other hand, among patients who simply seek evaluation for cognitive symptoms, about 10% may well have pseudodementia. Current textbooks still include pseudodementia in the differential diagnosis of dementia, but experts on all sides have been calling for its demise. Before examining the reasons—and there are some good ones—behind the disenchantment, here I provide a quick look at what the concept has meant up until now.

A question of definition
The term "pseudodementia" literally means false or pretended mental disorder and, in fact, that term has sometimes been applied to any factitious mental illness. But starting in the 1960s, the term came to be applied more specifically to the situation in which a "functional" psychiatric illness mimics dementia. Early on, depression was recognized as one of the functional psychiatric illnesses most likely to present with dementia features.

As the link between depression and cognitive impairment became increasingly apparent, the concept of pseudodementia was further narrowed to its current meaning: cognitive impairment caused by depression, usually in the elderly, that to some extent mimics other forms of dementia and may be reversible with treatment. Among the defining features of pseudodementia are the discrepancy between the patient's intense distress over cognitive impairment and the minimal impairment found on objective testing, the presence of depression, and the improvement in cognition as the depression lifts.

Psychiatrists, neurologists, geriatricians, and other primary care physicians who evaluate elderly patients not infrequently come across some who complain of failing memory; are difficult to engage in formal mental status testing, claiming that they cannot answer the questions; suffer the vegetative and psychological symptoms of depression; and seem less cognitively impaired when their depression improves. That is, some patients meet all the usual criteria for pseudodementia. As the clinicians and investigators who first recognized and wrote about pseudodementia pointed out, recognition of this syndrome can, in some instances, prevent a costly and uncomfortable diagnostic workup for dementia, protect the patient and caregiver from the distress associated with a premature label of dementia, and prompt the clinician to treat the patient's depression. All good stuff. So why is there a consensus among most experts to scrap this diagnosis? "Organic" or "Functional": Evolving View of Depression
The concept of pseudodementia arose when psychiatric illnesses were still divided into the "organic,"
such as dementia, and the "functional," such as schizophrenia and depression. The "organic" conditions were understood to be associated with brain pathology; the "functional" conditions were not. From today's perspective, enlightened by brain imaging that has revealed both physiologic and structural brain changes in all the major psychiatric illnesses, the division of psychiatric illnesses into those that are organic and those that are not sounds like something from the 19th century or earlier. In fact, the organic/functional dichotomy remained in the official psychiatric nomenclature until 1994 when, with the advent of the current psychiatric Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, this distinction was dropped.

The functional/organic divide was very much in play when the concept of pseudodementia took form, however. Central to the concept was the notion that the dementia of pseudodementia is functional—not associated with any brain pathology—and not real dementia, which comes with plaques, tangles, and infarcts. Pseudodementia was not thought to be "organic" because it was caused by depression, which was not thought to be organic. This piece of the concept no longer holds. Depression comes with all sorts of biologic changes, from pituitary-adrenal overactivity, to decreased serotonin receptor activity, to shifts in hippocampal size and prefrontal cortex activity. So pseudodementia, even when it is fully accounted for by depression and reverses when depression lifts, probably involves some brain pathology.

The fact that psychiatry has rightly dropped the organic/functional dichotomy is just one of the reasons behind the position that pseudodementia has had its day. More important, we know far more now about the relationship between depression and dementia than we did 40 years ago when the concept of pseudodementia was evolving. Although far from complete, this new information suggests, among other things, that there may be nothing "pseudo" about the dementia associated with depression.

Cognitive impairment is now recognized as an integral component of the depressive syndrome. Depressed patients typically report poor concentration, difficulty in making decisions, and muddled thinking. Among elderly persons with depression, about half show significant impairment on formal tests of cognition, particularly in the areas of attention, psychomotor speed, and other executive functions. The cognitive impairments of the depressed elderly are not as severe as those seen in early Alzheimer disease (AD), and they involve fewer areas of cognition. Unlike patients with AD, for example, the depressed elderly usually do not show disturbances in language or in cued recall. However, the cognitive abilities of patients with major depression are clearly not up to those of their nondepressed counterparts, and to some extent they improve as the depression lifts.

Adding to the complexity of the depression/dementia relationship is the high prevalence of depression in patients with dementia, particularly in those with relatively early dementia and dementia of moderate severity. Estimates of depression prevalence vary widely and depend on the method of detection, but most studies report that between 20% and 40% of patients with dementia also suffer major depression and that up to 70% have some depressive symptoms. This depression warrants and responds to treatment.

Depression as a Risk Factor for Dementia

The high prevalence of depression in dementia means that the presence of depressive symptoms in a patient with cognitive impairment does not in itself suggest that the cognitive impairment is secondary to depression or is likely to reverse as the depression improves. The web gets thicker when we consider new information about depression as a prodromal feature or risk factor for dementia. Longitudinal studies indicate that dementia is more likely to develop in elderly persons with depression than in their nondepressed counterparts. So depression may place people at increased risk for dementia or may be an early manifestation of dementia. Furthermore, elderly depressed patients with cognitive impairment are more likely to develop dementia than are the elderly depressed without cognitive impairment. Finally, although the cognitive impairment associated with depression in the elderly often improves somewhat as the depression lifts, recent studies indicate that some degree of cognitive impairment usually remains. These observations in the aggregate suggest that depression in the elderly may uncover or allow the expression of early-stage dementia.

Should Pseudodementia Be Renamed?

If the recently uncovered complexities of the depression-dementia relationship were not enough to bring on the demise of pseudodementia, we have the inaccuracy of the term itself. Pseudodementia does not actually mimic true dementia. In course of illness, complaints, and cognitive impairment, the 2 conditions are quite different (Table [not available online]). Further, the term "pseudodementia" oozes political incorrectness. It implies that the dementia is not real or, even more offensively incorrect, that it is faked. The not-real business is debatable, but it is
quite clear that patients with pseudodementia are not deliberately faking cognitive impairment. Unlike the malingering patient, they do not complain of memory difficulties for any obvious secondary gain, and unlike patients with factitious cognitive impairment or the rare Ganser syndrome, they tend to give "I don't know" answers on tests of mental function rather than bizarre or approximate answers. Additionally, patients with pseudodementia often suffer the characteristic features of depression of vegetativeness and self-neglect. Thus, patients with this syndrome can be distinguished from those with Ganser syndrome and other factitious mental illnesses. The usual suggestion from the "dump-pseudodementia" camp is that what has been called pseudodementia should be renamed something such as the "dementia syndrome of depression." That certainly would get rid of connotations suggesting unreality and fakery about the illness, but it is not a happy term.

Given all the good reasons for disposing of the term "pseudodementia," it is noteworthy that it has been retained in most textbooks that grapple with the differential diagnosis of dementia. Clinicians use it and seem to feel that it means something. It is also noteworthy that at least some textbooks display an exquisite ambivalence about the term. In a widely read and respected psychiatric textbook, for example, pseudodementia crops up in several chapters. In one chapter, it is derided as an inaccurate and anachronistic diagnosis; in another, it is carefully defined and included in the differential diagnosis of dementia. So why is pseudodementia hanging in there? Is it time to drop the whole concept? I think not. The fact that it is on the list of differential diagnoses keeps alive in the clinician's consciousness the possibility that a patient complaining of memory loss does not necessarily have AD or vascular dementia. It reminds clinicians to look for depression in such patients and to treat it when they find it.

In fact, patients-usually but not always elderly-do turn up complaining of terrible memory loss. They give a lot of "I don't know" and "I can't do it" answers to formal mental status questions (in contrast to patients with AD and other dementing disorders, who try hard to come up with approximate answers), but when engaged in formal cognitive tasks, they show minimal impairment. (See Table for distinction between dementia and pseudodementia.) Such patients have the vegetative and psychological symptoms of depression. With successful treatment of depression, their complaints of memory loss diminish and their scores on formal mental status tests improve.

**Conclusion**

What we have learned recently is that the long-term prognosis for this condition is not as benign as we originally thought or as the term suggests. Elderly patients with depression and cognitive impairment, even when the impairment improves somewhat as the depression lifts, are at a substantially greater risk for dementia than their nondepressed counterparts. Pseudodementia may be an early sign of "true" dementia.

Most important, the concept of pseudodementia is useful in guiding the approach to the patient. Largely because no curative treatments are yet available for the common dementias-AD and vascular-there is no rush to detect one of these diagnoses. If the history and physical examination do not suggest the presence of normal-pressure hydrocephalus or another of the rare treatable dementias, and if there is even a small possibility that a patient's cognitive impairment is caused by depression, the best course may be to delay an extensive dementia workup and treat the depression. Tell patients and their family members that depression sometimes causes thinking problems and that the first step is to treat the depression.

**Disclosures:**

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