Pseudodementia is a term that has been widely adopted in clinical practice, yet vastly misunderstood. As its name suggests, pseudodementia infers a “functional artifact” rather than a “true dementia” that is based on neuropathological disease. Wernicke first coined the term in the 1880s to describe “chronic hysterical states mimicking mental weakness.” It was not widely used until it was reintroduced by Madden in 1952 to describe patients with signs and symptoms of dementia that disappeared with successful treatment of an underlying psychotic illness. In 1961 Kiloh described 10 cases that mimicked irreversible dementia which resolved with treatment and time. Kiloh asserted that endogenous depression was the most frequent cause of pseudodementia and cautioned against misdiagnosing it as irreversible dementia. He argued that pseudodementia should be used as a descriptive term and not a diagnosis. In 1981, Caine proposed diagnostic criteria for pseudodementia (Table 1).

With an increasing awareness of cognitive changes in late-life depression, the concept of pseudodementia was further narrowed to describe cognitive impairment caused by depression, commonly in the elderly, that mimics dementia and resolves when the depression is successfully treated. Estimates on the prevalence of pseudodementia as it relates to depression were reported to be between 10% and 20%. This underscored the risk of misdiagnosing dementia in individuals with a potentially reversible disorder. However, over time, it became apparent that all the so-called reversible dementias, especially the “pseudodementia of depression,” were much less prevalent than previously thought. A meta-analysis of 39 studies from 1987 to 2002 identified potentially reversible dementias in 9% of patients but only 0.6% actually reversed and only 0.31%...
Loss of interest/apathy, loss of confidence, difficulties in making decisions, agitation, irritability and changes in sleep and appetite are common in patients with dementia, independent of whether or not they are depressed.
tributes pathophysiologically to the development of AD, whether it is a prodrome of AD, or both. Prospective follow-up of elderly patients presenting with cognitive impairment to a memory disorders clinic showed that many of these patients had symptoms of depression. When followed over time, most of them developed AD despite successful treatment of their depression and initial improvements in cognition. Also, depression, similar to other medical conditions, such as renal or cardiac disease, may bring forward the expression of dementia in patients with AD pathology.

The more distant the depressive episode is from the onset of dementia, the more likely that it is a contributor to the cause of the dementia as opposed to being a prodrome or part of the dementia syndrome. Taking this into consideration, a systematic evidence review concluded that a history of depression is likely an independent risk factor for dementia in general, and for AD specifically. This concept is supported by the glucocorticoid cascade hypothesis which postulates that prolonged adrenal glucocorticoid secretion in patients with depression has toxic effects on the hippocampus, leading to hippocampal atrophy, which is also an early hallmark of AD.

The debate on depression as an independent risk factor is unresolved, however, given the complexities in the relationships between depression and dementia and the misperceptions regarding the potential reversibility of dementia in elderly patients with depression, it is increasingly apparent that the concept of depressive pseudodementia, as a common cause of reversible dementia, should be abandoned.

A Return to Wernicke and the Concept of Conversion Pseudodementia

In examining the evolution of pseudodementia, one has to wonder whether Wernicke was correct in conceptualizing this condition in terms of a conversion disorder. Traditionally, conversion disorders have been used to describe the unconscious conversion of anxiety into physical symptoms. Little has been written about the potential of converting anxiety into cognitive symptoms. In a case series, Hepple described 10 older individuals with “conversion pseudodementia.” He reported that the core features of this disorder are: apparent cognitive impairment, regression and increasing physical dependency beginning in late-middle or early-old age, without evidence for an organic dementia from investigations or from taking into account the course of the illness. More recently, Delis and Wetter proposed diagnostic criteria for “Cogniform Disorder” and “Cogniform Condition.” They claim that excessive symptomatology involving cognitive complaints is a pervasive problem that has been extensively documented in peer-reviewed neuropsychological journals. Cognitive symptoms can be produced or exaggerated in an intentional, voluntary manner for an external incentive as in malingering, to adopt a “sick role” as in factitious disorder, or unintentionally, as in conversion disorder. As opposed to conversion disorder and malingering, where the feigned or exaggerated symptoms are either completely unconscious (conversion disorder) or completely conscious (malingering), the authors argue against the dichotomous approach of the DSM-IV for a continuum, from completely unconscious at one end of the spectrum, to partial and then full consciousness at the other end, whether or not in the presence of an external incentive (e.g., litigation, disability) or an adoption of the “sick role” (factitious disorder). The terms disorder and condition differentiate the degree to which the individual exhibits cognitive dysfunction in widespread areas of everyday life.

It is likely that cases of exaggerated cognitive symptoms are under-
reported as it is challenging for physicians to make judgments about their patients as possibly exaggerating their symptoms, whether intentionally or unintentionally. They are unlikely to know whether an external incentive is present or whether the patient has adopted a “sick role.” Furthermore, few patients assessed for cognitive impairment undergo rigorous neuropsychological evaluations with validity testing, and even if they do, today’s patients have a wealth of information at their disposal from the internet, media, or coaching from experienced litigation lawyers which may fool even the experienced examiner.

**Conclusion**

In summary, depressive pseudodementia evolved from a concern about the improper labeling of elderly patients with depression as having irreversible dementia. Recent data on the prevalence of reversible dementia has shown that this condition is extremely rare. Subsequently, depression is less often an imitator of dementia than a predictor of dementia or a symptom of dementia. Treating depression remains important. While it may not cure the cognitive disorder or reverse the dementia, it will likely improve the patient’s quality of life.

In today’s era of disease-modifying clinical drug trials for AD, mild depressive symptoms in patients with dementia should not delay their diagnosis nor should it exclude them from participating in AD research.

Lastly, cases of feigned or exaggerated cognitive complaints or symptoms are likely underappreciated. Clinicians should be urged to consider this as a possibility when assessing patients whose cognitive complaints or symptoms are not substantiated by the clinical interview or cognitive assessment, especially when reinforced by an external incentive or the adoption of a “sick role.”

**References:**