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Pseudodementia

On the face of it there is no sense in calling a disorder "pseudo"—one thing may mimic another, and presumptive diagnoses are often wrong. But despite a recent call¹ for the term to be abandoned "pseudodementia" is probably with us to stay, for the name both meets a need and draws attention to a problem in the classification of disease. Pseudodementia has no precise nosological meaning—by contrast with pseudohypoparathyroidism, which is a specific disease, albeit named

by reference to what it is not. The term is used to describe those disorders which present with the features of dementia but which on closer study or because of their subsequent course turn out to be of different origin—and in old people the underlying disorder is most often depression.

Kiloh's review in 1961 put pseudodementia on the map.² He wrote: "The picture of dementia may be very closely mimicked and (patients) may be in danger of therapeutic neglect and perhaps of unnecessary neurosurgical investigations. It is to such cases that the term pseudodementia may be applied," and it is to be distinguished from the Ganser state ("hysterical pseudodementia") of younger adults. Kiloh emphasised that old people suffering from depression are at particular risk of being labelled as demented. Wells,³ who saw pseudodementia as a "caricature" of dementia rather than mere mimicry, drew attention to the lack of disturbed behaviour in his patients. But in other respects his 10 patients, all aged under 69, were atypical, for only three of them were depressed and "chronic characterological deficits" were prominent. In a recent review Caine⁴ presented a series of patients (again only one was over 70) in whom the most common form of intellectual deficit resembled the subcortical dementias.

Several recent reports have shown that 10-20% of patients who are referred for investigation of their dementia turn out to have other disorders. Occasionally the diagnosis of dementia is confirmed after investigation, but after several years the true diagnosis becomes obvious. The study of Naguib and Levy,⁵ in which the diagnosis of senile dementia remained stable after two years in all the survivors, is an exception to this observation, but their patients were older—between 70 and 90 years old. Most of the recent series, however, have included few patients over 65 and virtually none over 80; some have confined their studies to presenile dementias—for example, Nott and Fleminger⁶ and Ron and her colleagues.⁷ When patients over 65 are included there is often no breakdown of ages within this "elderly" group⁸ or details of age are incomplete.^{9,10} Many series originate from major referral centres and represent not only selected but often particularly difficult groups of patients—one such series is from the National Hospital for Nervous Diseases, reported by Marsden and Harrison.^{10,11} This report shows that there are likely to be differences between those patients who are referred to neurologists and those referred to psychiatrists or to general physicians. A recent review has put in perspective the balance between the pursuit of potentially reversible causes and attention to established disabilities.¹²

A further problem is posed by authors who fail to record the final diagnosis and merely state that certain patients who were originally thought to be suffering from degenerative dementia either had potentially "treatable" conditions or appeared to be suffering from something else. The question may then be raised, Should the diagnosis of dementia depend on a malignant course? A useful working definition, based on that of Lishman,¹³ is that dementia is "an acquired global impairment of memory, intelligence, personality, and skills, commonly progressive, without impairment of consciousness." This relates to a clinical syndrome rather than to its aetiology or prognosis—though it recognises the progressive nature of the typical case.

Rabins¹⁴ reviewed "reversible dementia" and found that in old people this syndrome is most commonly due to depression, but he rightly emphasised that not only may depression present as dementia but dementia may also coexist with depression. Many depressed old people (especially the very old) present

in a clinical state characterised by inaccessibility, little coherent speech, rejection of food and drink, and apparent disorientation, but without overt affective features. Sometimes these are confusional states, when befuddlement is a prominent feature; such a presentation should strictly be described as confusional or delirious rather than as pseudodementia. But clouding of consciousness is rarely prominent in these cases and may therefore be difficult both to identify and to measure. The patient may then be incorrectly diagnosed as suffering from dementia. Why depression should present in this way in the aged is a question to which we do not yet have an answer, but it seems likely that study of the neurochemistry of the aging brain may provide the answer. The term pseudodementia is best confined to these dementia-like presentations of depressive illness. Diagnosis may be especially difficult in the aged when the remediable depressive component is obscured by a degree of actual dementia, but such patients may benefit from skilled care.

What guidelines exist which may enable the clinician to diagnose depression when it masquerades as dementia? Firstly, a short history should raise suspicions, and the duration of the illness and its mode of onset must be established. This will include finding out whether or not there have been "life events," most commonly losses, which might be relevant. Secondly, a family history of depressive illness must be sought (this may be suppressed by relatives).¹⁵ Thirdly, Post has suggested that patients with pseudodementia are apt to complain of their memory defects—indeed "they may almost parade them"—in contrast with the common "anosognosia"—where there is a loss of the ability to recognise one's illness—of true dementia.¹⁶ In pseudodementia patients tend to give clear accounts of their illness and their past life in response to open ended questions, and reply "I don't know" only to direct questions. They often become distressed and make little effort to perform even simple tasks, while patients with dementia do their best, and it is only when they overstretch themselves that they may exhibit a catastrophic reaction.

In his clinical guidelines, Post suggests that the term pseudodementia should be reserved for patients who are capable of cooperating with detailed cognitive assessment. But even elderly, disorganised patients whose affective state may be difficult to assess may have a depressive illness. Hence it is important to seek for evidence of depression, which may be provided by relatives or nursing staff who look after the elderly patient around the clock. They may witness a transient stage when the patient emerges from incoherence or inaccessibility to utter suspiciously depressive statements, for example, "I don't deserve this food." Sensitive observation may detect a sad facial expression or demeanour, which may be transient but points to the diagnosis.¹⁵ If such observations are linked with either a previous history or a clear family history of depression, or other corroborative evidence such as a potentially relevant "life event," a trial of antidepressive treatment may be justified. It is important to ensure that the patient's fluid and electrolyte balance is maintained, for old people who are mentally ill may be incapable of maintaining an adequate fluid intake. Such patients often need the joint care of psychiatrists and physicians.¹⁷

TOM ARIE

Professor of Health Care of the Elderly,
University of Nottingham,
Sherwood Hospital,
Nottingham NG5 1PD

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Unequal legs

A difference in the length of a child's legs is a source of considerable anxiety to parents. The first important task for the doctor is to establish whether the discrepancy is due to a true difference in the length of the limbs or is an apparent difference—or a combination of the two.¹ Apparent inequality may be due to a hip deformity such as congenital dislocation, a spinal deformity such as scoliosis with tilting of the pelvis, a fixed flexion deformity of the knee, or an equinus deformity of the ankle. In such cases treatment must be directed to the underlying abnormality.

True inequality alters with growth in several different ways. By no means all discrepancies in length increase at a constant rate with time. Various patterns have been identified: some slow down as growth proceeds, some may reach a plateau before the cessation of growth, while others may at first increase, then stabilise, and then decrease—without surgery.² This variation reflects the disparate causes. Poliomyelitis may produce considerable shortening, particularly if the paralysis affects the limb early in life. Hemiplegic cerebral palsy usually produces some shortening, but rarely more than 4-5 cm. Soft tissue abnormalities that produce hyperaemia, such as neurofibromatous and haemangiomatic malformations, may increase the length of the leg, as may infections and fractures. Underdevelopment of the femur and leg bones with or without absence of the whole or part of these bones may be due to congenital defects.

Overgrowth is usual after a fracture of the leg.³ In a series of 74 children under 13 years with fractures of the femoral shaft the average femoral overgrowth was 0.92 cm. This overgrowth occurred regardless of the age of the child at the time of the fracture, its level, or the position of the fracture at