1. What do you understand by the term pseudodementia?

Resembles an organic dementia (18), potentially reversible with/without treatment (6), functional (9), depressive (9), found usually in elderly (5), memory impairment an important component (12), and "hysterical", including Ganser's syndrome (7).

2. What do you consider to be its cause(s)?

Depression, especially in the elderly (21), anxiety/stress/loss (8), "hysteria"—dissociative (7), "Ganser's syndrome" (3), schizophrenia/schizophreniform (5), malingering (2). Parkinsonism, hypovitaminoses, hypothyroidism and other metabolic causes, and "age-related brain changes" were mentioned by individual respondents.

3. Please comment on the current use and validity of the term.

A reminder to the physician to search further (6), a retrospective diagnosis—with or without the use of treatment (4), a useful term if criteria are stated (4), should be confined to elderly depressives with a dementia-like syndrome (3), a useful term (3), a useless/confusing term (3), a term of rare applicability (3), and, lest we forget, depression and dementia may co-exist (2).

Our general impression was that pseudodementia, like "pseudodepression" (Feinberg & Goodman, 1984), is a loosely held concept amongst psychiatrists. We would suggest that it be replaced by a clearer statement of the findings. As an illustration of how this might be done we would refer to Feinberg & Goodman's (1984) "four 'Ideal Types' of depression plus dementia syndromes": depression presenting as dementia ("depressive pseudodementia"), dementia presenting as depression ("pseudodepression"), depression with secondary dementia ("dementia syndrome of depression"), and dementia with secondary depression ("depressive syndrome of dementia").

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Huntington's Chorea Without Dementia

SIR: We would like to comment on the case report of Turner (*Journal*, May 1985, **146**, 548–550) concerning a patient who developed chorea in middle age, had a positive family history of Huntington's chorea, but who showed no evidence of dementia on formal psychological testing. The author questions the value of genetic counselling in this case and in particular whether it was justified, given that there were doubts about the diagnosis. We agree that the likely diagnosis for this patient is Huntington's chorea and would not be surprised by the lack of dementia, particularly with a relatively late age at onset. Indeed, lack of mental deterioration in some cases was noted by Davenport & Muncie (1916). Caution is required if there is a family history or non-progressive choreiform movements from childhood without dementia, since a likely diagnosis is benign hereditary chorea (Harper, 1978).

Genetic counselling should be part of the management of any family in which a relative is diagnosed to have Huntington's chorea, and the consequences of its deficiency are well documented by Martindale & Yale (1983). The form of the counselling is important and a general family meeting is inadequate; risks for an individual need to be considered separately and worries and fears dealt with on an individual basis. We question the statement that the seven living siblings had outlived their major risk period, since 10% of Huntington's chorea patients have an age of onset after 60 years (the age of the eldest sibling). The risk to the youngest sibling aged 42 years is considerably higher—approximately 38%, based on the life table method of Newcombe (1981).

Concern was expressed about the daughter of the index case, who may have broken off her engagement for a number of reasons. A full discussion of the genetic aspects might have eased her problem; in particular, information about the recent developments in DNA technology which may allow her to have children at low risk for developing Huntington's chorea, without changing the risks to herself (Harper & Sarfarzi, 1985).

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Treatment of Mania with the Cholinomimetic Agent RS 86

SIR: According to observations that cholinomimetic agents such as physostigmine may counteract mania and can cause depression, Janowsky (1972) formulated the cholinergic-adrenergic imbalance hypothesis of affective disorders. It postulates that depression is due to a central nervous hyperactivity of the cholinergic system in relation to the adrenergic system and that the opposite is the case for mania. In contrast to physostigmine, which has a half-life of only 10–20 minutes and the injection of which is frequently accompanied by unpleasant vegetative side-effects requiring the application of the peripherally acting antidote methscopolamin, the spiropiperidyl derivative RS 86 is a more suitable drug for studying the question of whether cholinomimetic agents possess an anti-manic effect. RS 86 is a direct muscarinic agonist, passes the blood brain barrier, has a half-life of six to eight hours and because of the drug's minor peripheral side-effects its combination with an antidote is not necessary (Spiegel, 1984).

In a double-blind study, using a placebo-drug-placebo-drug design, RS 86 was given to six female and four male patients aged between 19 and 52 years (mean = 35.6, SD = 10.6); nine patients fulfilled the RDC for mania, one for hypomania. The length of the placebo phases varied from two to seven days; the drug phases lasted six days with a successive increase of the doses generally up to 4 mg RS 86 per day. If clinically necessary, chloral hydrate, paraldehyde or levopromazine (maximally 400 mg per day) were administered. The degree of mania was assessed daily by two independent raters using the Inpatient Multidimensional Psychiatric Scale (IMPS) (Lorr, 1974).

Three patients did not show any improvement in their manic syndrome after the intake of RS 86. In one of them, even the increase of the dosage up to 6 mg had no effect on the psychopathology but caused nausea. Two patients displayed a marked improvement of the manic disorder during the drug phase, a relapse during the following placebo phase, and once again an improvement in the second drug phase. Five patients experienced a continuous improvement in their manic symptomatology which also lasted throughout the following placebo phase. As indicated by the relevant IMPS items, the improvement of the manic symptoms, which was observed two to four days after RS 86 intake, included not only psychomotor disturbances but also euphoria, grandiosity and superiority. Except for the nausea reported by the one patient who did not even respond to the 6 mg RS 86 dose, only minor side-effects such as increased salivation or sweating were reported.

Our study confirms former findings that cholinomimetic agents possess antimanic properties. The lack of effectiveness of RS 86 in three of the ten patients cannot be explained by the fact that the non-responders suffered from a more severe manic psychopathology than the responders, as this was not the case. A different pathogenetic mechanism, not influenced by the muscarinic agonist, or individual differences in the bioavailability of RS 86 might be responsible for the varied clinical responses. Surprisingly, five of the seven RS 86 responders did not show a relapse during the second placebo phase. As a spontaneous remission occurring in each of these patients during the first days of RS 86 medication seems to be rather unlikely, a RS 86 induced "switch process" terminating the manic episode has to be considered.

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Is Mania Really Incompatible with Down's Syndrome?

SIR: As we were already surveying the Down's syndrome population of our hospital, for psychiatric illness, we were very interested in the observation of Sovner *et al* (*Journal,* March 1985, **146,** 319–320). Their hypothesis that Down's Syndrome precludes the development of mania enhances our understanding of the aetiology of psychosis.

We identified 60 cases of Down's syndrome from among a hospital population of 1014. Apart from the Standard Psychiatric Interview we used Feigner's criteria and ward staffs' observations in