had a voracious appetite and indiscriminate eating habits which included paper towels, plants, styrofoam cups, and even faeces. At one point, he drank urine from a catheter bag. The patient was no longer his usual assertive self and had become quite docile. He tended to wander about the ward touching objects or people and made inappropriate comments of a sexual nature. On examination, his speech was dysarthric but fluent, and he had difficulty with the comprehension of multiple step commands. He had an amnesia, poor spatial orientation, and difficulty with constructions. The patient could not recognise colours, shapes, or objects by visual presentation; however, he could match the objects visually and name them by touch. He had normal visual acuity and visual fields, mild right hemiparesis, and right sided hyperreflexia. Brain CT showed a single hypodensity in the left temporoparietal area.

The source of his behaviour change was considered to be a combination of post-anoxic and epileptic injury involving both anterior temporal lobes. On the day of his death, the patient had a respiratory arrest after stuffing his mouth with surgical gauze. He had wandered about the ward picking up whatever he could find and putting it into his mouth. Neuropathological examination disclosed an old infarction in the left posterior cerebral artery territory, virtual absence of the left anterior temporal lobe, and atrophy of the right parahippocampal gyrus, hippocampus, and amygdala.

Patient No 2 was a 54 year old man with dementia who developed aggressive food seeking behaviour. He would go from room to room, take food from others' trays, and rapidly eat it. His first symptom of dementia was at the age of 50 when he displayed uncharacteristically poor judgment by exchanging a brand new car for an old one. Over the next four years, he had a progressive deterioration of judgment and memory. In addition, the patient became placid, began manually exploring his surroundings and grabbing at objects, and occasionally exposed himself to others. On examination, he was passive and mute, except for short phrase responses and echolalia. His memory was impaired, but calculation and construction abilities were preserved. He could visually recognise and name colours and objects. He had normal visual acuity, visual fields, motor testing, and reflexes.

The patient's CT showed disproportionate frontal lobe atrophy, and the patient was diagnosed with frontotemporal dementia complicated by the Klüver-Bucy syndrome. On the day of his death, he was seen to develop a breathing difficulty after a large meal. A Heimlich manoeuvre was performed, and he vomited a large amount of undigested food. There was massive aspiration of poorly masticated food in the pharynx, and the patient did not respond to resuscitation efforts. Neuropathological examination showed pronounced frontal lobe atrophy with Pick cells but no Pick bodies, and milder anterior temporal atrophy.

These two patients illustrate that hyperoral behaviour can be lethal. Both patients had the Klüver-Bucy syndrome with a tendency to engage in oral exploration of objects and hyperbulimia, or an excessive, insatiable appetite. In this syndrome, the hyperoral behaviour probably results from damage to the amygdalae in the anterior temporal lobes.² Patients with frontotempo-

ral dementia, as in patient No 2, are prone to this syndrome,¹ but the Klüver-Bucy syndrome has many aetiologies including trauma, strokes, ischaemia, and epilepsy, as in patient No 1.

Other neurological causes of hyperoral behaviour may pose a danger to patients. Lesions of the ventromedial hypothalamic "satiety centre," such as hamartomas and germinomas, can lead to hyperphagia.^{3,4} The Kleine-Levin syndrome presents with periodic hyperphagia and hypersomnolence, and there is one report of a patient with this syndrome who asphyxiated on a sausage.⁵ Additional causes of hyperphagia include bilateral thalamic infarcts and congenital disorders such as the Prader-Willi syndrome and the Laurence-Moon-Biedl syndrome.

Neurologists, psychiatrists, and others who manage these patients need to be aware of the danger of death from asphyxiation or aspiration. Close supervision and other preventive measures are indicated to avoid this complication in patients with the Klüver-Bucy syndrome and related neurological disorders.

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Anxiety disorders in non-demented and demented elderly patients: prevalence and correlates

Anxiety disorders account for only a fraction of admissions to psychiatric hospital of patients over the age of 65 and a decline in anxiety disorders has been reported in outpatients.¹ Studies on prevalence rates that have used community based samples have reported mixed results, although a vast majority found a decrease in prevalence in the elderly population.²⁻⁴ This presents an interesting problem, as factors reported to be highly associated with anxiety disorders such as decreased physical health, bereavement, isolation, and decreased autonomy are more likely to increase with age.^{5,6} A possible explanation for this anomaly might be that age related biological factors reduce the sensitivity of older people to these risk factors.

Although dementia is a common disorder in elderly people which is often accompanied by psychiatric symptoms there are few studies examining the association between dementia and anxiety. To our knowledge the only published study has reported an increased rate of anxious mood in demented persons.⁷ However, this study made no diagnoses of anxiety disorders and the patients were all at an early stage of dementia.

The aim of the present study was to estimate the prevalence of anxiety disorders in non-demented and demented subjects and to identify some of the variables associated with this in an elderly population. The participants came from a population based study in Stockholm, Sweden.

We used data from the follow up phase of a longitudinal investigation of adults aged 78 years and over residing in the Kungsholmen parish of Stockholm, Sweden. A total of 1101 persons comprised the study population. A more detailed description of the population and the methods used has been reported elsewhere.⁸

Information regarding psychiatric symptoms was derived from psychiatric examinations conducted by physicians using the comprehensive psychopathological rating scale (CPRS).⁹ The physicians were trained and attended regular meetings to ensure that the CPRS was administered accurately and consistently. Information on psychiatric history and physical health was obtained by direct examination of the participants, interviews with informants, and previous medical records. Disabilities in daily living were assessed using the Katz index, which is a hierarchical scale (0-6) measuring independence in six activities of daily living.¹⁰ Impairment in activities of daily living (ADL) was determined as decreased if the person had difficulties in two or more activities. Visual and hearing problems were graded by the physicians and only those having a practical problem were taken into account. The Swedish version of the mini mental state examination (MMSE), a global measure of cognitive functioning, was also administered to participants, with a maximum score of 30.¹¹ Dementia diagnosis was made using the DSM-III-R¹² criteria to maintain accuracy with previous phases of the study. The severity of dementia was classified according to the Washington clinical dementia rating scale (CDR).¹³ The psychiatric diagnoses were combined in three groups: depressive, psychotic, and anxiety disorders. DSM-IV criteria¹⁴ were used with the modification that even if an organic state was present—for example, dementia—the diagnosis were made. In addition to dementia only one axis I diagnosis was made. If the person had more than one diagnosis on axis I only the most clinically significant was registered.

To analyse differences in the prevalence of depressive, psychotic, and anxiety disorders, a one way analysis of variance was performed with severity of dementia as the between subjects factor. Odds ratios (ORs) and 95% confidence intervals (95% CIs) were computed to analyse differences between subjects with and without anxiety disorder.

Psychiatric information was available for 966 of the 1101 persons. Missing data were mostly due to severe cognitive impairment. Of the 966 participants 740 were women and 226 were men. The mean age of the sample was 84.2 (SD 4.3) years and the mean MMSE score was 25.5 (SD 4.5). There were 786 non-demented participants, and 180 were diagnosed with dementia. Of the 180 demented persons, 58 were diagnosed as questionable, 84 as mild, 31 as moderate, and seven as severely demented.

The table shows the prevalence of anxiety, psychotic, and depressive disorders with the population divided according to severity of dementia. Due to small numbers moderate (n = 31) and severe (n = 7) dementia

Prevalence (%) of anxiety, depressive, and psychotic disorders by dementia severity

Disorders	Demented			
	Non-demented (n = 786)	Questionable (n = 58)	Mild (n = 84)	Moderate/severe (n = 38)
Anxiety	3.2 (25)	5.2 (3)	3.6 (3)	0 (0)
Depressive	5.5 (43)	12.1 (7)	20.2 (17)	21.1 (8)
Psychotic	0.3 (2)	3.4 (2)	10.7 (9)	5.3 (2)

Actual numbers of patients are given in parentheses.

were combined. The prevalence of anxiety disorders was 3.2% in the non-demented and 3.3% in the combined demented group. Psychotic ($F = 21.1$, df 1,962, $P < 0.001$) and depressive ($F = 11.3$, df 3,962, $P < 0.001$) disorders were more common in demented than in non-demented persons, whereas anxiety disorders showed no difference. No persons with moderate or severe dementia were diagnosed as having an anxiety disorder. Mean MMSE score in those with a diagnosed anxiety disorder was 25.2 (SD 3.1), with a range from 19 to 30. In those without a diagnosed anxiety disorder the mean MMSE score was 25.5 (SD 4.5), with a range from 0 to 30. Notable was that of the 71 persons with an MMSE score ≤ 18 , none had an anxiety disorder.

Having a history of psychiatric disorders (OR = 9.8, 95% CI 9.0–10.6) and impaired activities of daily living (OR = 3.0, 95% CI 2.0–4.1) were found to correlate with having an anxiety disorder. The other potentially associated variables including marital status, sex, institutionalisation, educational level, somatic disorders (cardiac, cardiovascular, musculoskeletal, or malignant), visual and hearing problems, or dementia were not found to correlate. No substantial differences were found if demented patients were excluded from the analysis.

The results indicate that anxiety disorders were diagnosed in 3.2% of this population of very elderly adults, and the prevalence was equally distributed in non-demented and demented groups. This finding is in agreement with the prevalence rate reported in the ECA, Edmonton studies, and the Guy's age concern survey.^{2,3,15} Moreover, the present study found that in those persons who were moderate or severely demented, anxiety disorders were not present. In fact a MMSE score of at least 18 seemed to be critical for the presence of an anxiety disorder. This might have been a reflection of the fact that those participants with more severe cognitive dysfunction lacked the appropriate insight or were unable to accurately respond to questions from the CPRS (masking hypothesis), despite the exclusion of those who were unable to provide answers in data analysis. Another possible explanation for this finding may be that demented persons at that level of severity are unable to experience complex integrated emotions due to advanced brain disease (the extinction hypothesis). By contrast, the instrument in use seemed to be able to diagnose depressive and psychotic disorders, even if the person had reached a more severe level of dementia. The hierarchy of the diagnostic system also might have influenced the fact that the prevalence of depression exceeded the prevalence of anxiety disorders.

Anxiety disorders were found to correlate with having a history of psychiatric disorders and impaired activities of daily living. This supports the statement by Lindsay¹⁶ that restriction of activity and imposed independence may cause anxiety.

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Ataxic type of Creutzfeldt-Jakob disease with disproportionate enlargement of the fourth ventricle: a serial CT study

There are few reported neuroimaging studies on the ataxic type of Creutzfeldt-Jakob disease. In our patient MRI and serial CT disclosed a progressive enlargement of the fourth ventricles as a major finding.

A 64 year old woman developed progressive gait unsteadiness in June 1994; she was then evaluated in another hospital where neurological examination and CT (the scan was unavailable to us) were reported as normal. She had no history of neurosurgery or ocular surgery or family history of neurological disease. Examination in September 1994 showed gait ataxia and gaze nystagmus without dysarthria. Routine laboratory investigations and tumour markers were normal or negative. Analysis of CSF including IgG content was normal; oligoclonal bands were not detected. Brain CT (figure, A, B) was normal for the patient's age. An EEG showed slowing of background activity. As there was no evidence of systemic cancer or other known aetiologies of cerebellar degeneration, a diagnosis of idiopathic late onset cerebellar ataxia was made. Two months later there was rapid intellectual decline with complex hallucinations, emotional changes, disorientation, and confusion leading to a vegetative state within a few weeks. Multifocal axial and appendicular myoclonus was now present. Serial waking-sleep EEG showed progressive slowing of background activity with occasional periodic complexes. Serial CT (figure, C-F) showed progressive cerebellar and brain atrophy with pronounced enlargement of the fourth ventricle. Transverse diameters of the fourth ventricle at first, second, and third examination were 1.3, 1.9, and 2.5 cm (normal value 1.44 (SD 0.22)).¹ The brainstem ratio was normal in all three CT studies (for the last 0.19 (control value 0.22 (0.04)).¹ An MRI study in November 1994 corroborated the CT findings and neither brainstem atrophy nor abnormal signal of white matter or basal ganglia were noted. Two specific proteins p131 and p130 were identified by gel electrophoresis of CSF and CSF neuron-specific enolase was significantly increased at 240 ng/ml (upper limit of normal 50 ng/l; T Weber, University of Göttingen, Germany, personal communication). The patient died in February 1995.

Necropsy, restricted to the nervous system, was performed six hours after death. The brain (fixed weight 1150 g) was of normal external appearance. On section the ventricular system, especially the fourth ventricle, was seen to be enlarged and both the cerebral cortex and the cerebellar cortex were thin. Microscopically there was widespread spongy degeneration throughout the grey matter and basal ganglia, most pronounced over the frontal and occipital cortex, thalamus, and putamen. In the cerebellum there was considerable loss of granule cells with relative preservation of Purkinje cells that often exhibited axonal 'torpedoes'. The white matter was gliotic to some degree but myelin stains demonstrated no evidence of demyelination. Kurulike plaques were not found. Spongy degeneration was also seen in the molecular layer and brainstem, but cerebellar peduncles were not demyelinated and there was no cell loss in the pons and inferior olivary nuclei. Prion protein gene analysis was not performed.