had a voracious appetite and indiscriminate eating habits which included paper towels, plants, styrofoam cups, and even faeces. At one point he returned from a carpetbag.

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 sexual advances. On examination, his speech was dysarthric but fluent, and he had difficulty with the comprehension of multiple step commands. He had no control over 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 hypolucency in the left temporo-parietal 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 suffocating himself with surgical tape. He had wandered about the ward picking up whatever he could find and putting it into his mouth. Neuropsychological examination disclosed a significant aphasialike and inattentional disturbance in the left postero-temporal cerebral 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 a limited and peremptory style of response 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 disproportionality between 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. Neurological 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 who had the Klüver-Bucy syndrome with a tendency to engage in oral exploration of objects and body parts, or an oral insatiable appetite. In this syndrome, the hyperoral behaviour probably results from damage to the amygdala in the anterior temporal lobes. Patients with frontotemporal dementia, as in patient No. 2, are prone to this syndrome, 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. The Kléine-Levin syndrome presents with periodic hyperphagia and hypersomnolence, and there is one report of a patient with this syndrome who asphyxiated on a sausage. Additional causes of hyperphagia include bilateral thalamic infarcts and congenital disorders such as the Frader-Willis 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 preventative 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. Symptoms of anxiety and depression are often intermixed, and rates that have used community-based samples have reported mixed results, although a vast majority found a decrease in prevalence in the elderly population. 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 affect older than younger patients. A possible pathological basis for this anomaly might be that age-related biological factors reduce the sensitivity of older people to these risk factors.

Althought depression is more 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. 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 possible predictors of anxiety 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 and more details of the population and the methods used has been reported elsewhere. Information regarding psychiatric symptoms was derived from psychiatric examination conducted by a psychiatrist using a comprehensive psychopathological rating scale (CPRS). The physicians were trained and attended regular meetings to ensure that the CPRS was administered and scored 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 activities of daily living were assessed using the Katz index, which is a hierarchical scale (0–6) measuring independence in six activities of daily living. Impairment in activities of daily living (ADL) was determined from patients using the DSM-III-R 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). The psychiatric diagnoses were combined in three groups: depressive, psychotic, and anxiety disorders. DSM-IV criteria were used with the modification that even if a specific diagnosis 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 groups 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 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

<table>
<thead>
<tr>
<th>Disorders</th>
<th>Non-demented (n = 786)</th>
<th>Questionable (n = 38)</th>
<th>Mild (n = 84)</th>
<th>Moderate/severe (n = 38)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anxiety</td>
<td>3-2 (25)</td>
<td>5-2 (3)</td>
<td>3-6 (3)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Depressive</td>
<td>5-5 (43)</td>
<td>12-1 (7)</td>
<td>20-2 (17)</td>
<td>21-1 (8)</td>
</tr>
<tr>
<td>Psychotic</td>
<td>0-3 (2)</td>
<td>3-4 (2)</td>
<td>10-7 (9)</td>
<td>5-3 (2)</td>
</tr>
</tbody>
</table>

Actual numbers of patients are given in parentheses.

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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 ventricle 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 (MMSE 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 revealed a right 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 etiologies 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, anxiety, agitation, and confusion, and she died of a vegetative state within a few weeks. Multi-focal 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. The transverse diameter of the ventricle was 1-3, 1-9, and 2-5 cm (normal value 1-44 (SD 0.22)). The brainstem ratio was normal in all three CT studies (for the last 0-19 (control value 0-22 (0-04)). 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 p105 and p130 were found by gelelectrophoresis of CSF and CSF neuron-specific enolase was significantly increased at 240 ng/ml (upper limit of normal 50 ng/l).

Necropsy, restricted to the nervous system, was performed six hours after death. The brainweight 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 thinned. There was no spread of spongy degeneration through the grey matter and basal ganglia, most pronounced over the frontal and occipital cortex. 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 show some diagnostic plaques. There was no evidence of demyelination. Kuru-like plaques were not found. Spongy degeneration was also seen in the molecular layer and brainstem, but there were not demyleinated and there was no cell loss in the pontine and inferior olivary nuclei. Prior protein gene analysis was not performed.
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