tion syndrome has a poor prognosis, with a 50% mortality rate. The nervous system is seldom involved in the syndrome. If such involvement appears, it usually does so towards the end of the course of the disease. A patient with sensorimotor neuropathy related to axonopathy and occasional demyelination has been recently reported, but in the context of a fulminating illness.1

The distinctive feature of our finding is the occurrence of transient cranial nerve involvement as the probable first sign of macrophage activation syndrome. It could be claimed that the symptomatology is related to the lymphoma. However, very little is known about neurological complications in T cell lymphomas, and their occurrence is probably rare.2 Kaufman et al3 have reported an involvement of the nervous system in 14 patients out of 104 cases, eight being related to direct complications. In only one patient, palsy of the sixth cranial nerve was the first sign. Neurological signs occurred between 10 and 102 weeks after diagnosis of lymphoma. If polyneuropathy occurs in T cell lymphoma it is due to infiltration and the clinical evolution is usually stereotyped with slowly evolving sensorimotor signs.4 Because there is no histological examination in our case, infiltration of peripheral nerves cannot be eliminated; but it is unlikely, considering the improvement in neurological signs. Meningoradiculitis could be evoked, but if that were so, there would have been a worsening of the initial signs.4 Moreover, CSF examination and cerebral MRI were normal. All these indications lead us to suggest that the neurological signs in our patient could be related to a remitting/relapping neuropathy due to non-cutaneous T cell lymphoma infiltrating peripheral nerves, to vasculitis or, more likely, to the neurotoxic effects of cytokines. Cytokines, especially TNF, are secreted in large amounts in macrophage activation syndrome, and TNF can induce general side effects and cerebral damage.5

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Figure 2. Lymphoid cells with azurophil granules. Myelogram (originaly × 100).

Paraneoplastic opsonus associated with cancer of the gall bladder

Opsonus is an ocular dyskinnesia consisting in ample, conjugated, arrhythmic, multifocal ocular movements which persist even with the eyes closed.6 This syndrome has been described during the course of different cancers.6 In infancy, neuroblastoma is the cancer most often associated with opsonus (in 2% to 7% of the cases).7 In adults, opsonus is less common. Nevertheless, it is associated with a tumour in 20% of cases.7 Here, we report a case of opsonus associated with a cancer of the gall bladder.

A 72 year old, treated hypertensive man, experienced the sudden onset of vertigo followed by impaired consciousness. At initial examination, her Glasgow score was 13. She showed opsonus associated with a bilateral kinetic cerebellar syndrome. The cranial nerves were intact and there was no sensory or motor deficit. Complete physiological examination only showed conjunctival icterus.

Brain MRI showed a left frontal angiomata measuring 7 mm in diameter without any impingement on the cerebral parenchyma; the brain stem was normal. Two spinal taps were normal. A chest radiograph was normal. Laboratory studies showed an increase in alanine aminotransferase (43 IU/L) (111 kU/L). Abdominal ultrasound showed a heterogenous, polyploid tumorous structure in the gall bladder associated with hyperchogetic lesions in the liver and a thrombosis of the portal vein. Abdominal CT disclosed thickening of the left lateral wall of the gall bladder, liver metastases, and hilar adenopathy. Liver biopsy showed a sclerosing, endothelial derived angio- carcinoma most suggestive of a pancreatico-biliary origin.

Tests for anti-Hu, anti-Ri, and anti-Yo antibodies were negative. Immunoglobulin in the serum was 5.0 (4.6 g/day) and cortisone (Solumedrol, 0.5 g/day for five days) was ineffective. The patient died five weeks later. No necropsy was performed.

The diagnosis of opsonus remains clinical. It usually has an abrupt onset.6 It is probably the result of a diencephalic or mesencephalic lesion with production of the abnormal movement by removal of normal saccadic generator inhibition.7 Dysfunction of the pause neurons, which play a part in inhibiting the phasic neurons responsible for the appearance of jerks is likely. The fact that angio- carcinoma does not explain the opsonus. The opsonus was considered paraneoplastic because it was not associated with an infectious or tumoral cerebral angiopathy. Other possible causes (toxic, metabolic, degenerative, and vascular)7 were excluded.

The normal MRI, lumbar punctures, and the absence of anti-Ri antibodies, which have been associated with paraneoplastic opsonus occurring with carcinomas of the breast,7 did not cast doubt on the diagnosis. Breast cancer and small cell cancer of the lung represent 70% of reported cases associated with opsonus in adults7 and are sometimes discovered during necropsy.8 In the present case, the histological differentiation seen during liver biopsy was strongly suggestive of a primary lesion in the gall bladder. Therefore, it is not likely that the lesions discovered were metastases from one of the above cited cancer localisations.

The possibility of this association means that the gall bladder should be included in the investigation of a paraneoplastic opsonus.

Lethal hyperoral behaviour from the Klüver-Bucy syndrome

Clinicians have not sufficiently appreciated the danger of hyperoral behaviour in neurologic disorders. A manifestation of this behaviour is the Klüver-Bucy syndrome.9 Originally described in monkeys after anterior bitemporal lobectomies, this syndrome includes indiscriminate dietary behaviour and a tendency to examine objects by mouth.3 The complete syndrome also results in placidity, hypersexuality, hypermetamorphosis or a tendency to attend to any visual stimulus, and visual agnosia. We report two patients with the Klüver-Bucy syndrome who died as a consequence of their hyperoral behaviour.

Patient No 1 was a 40 year old man with epilepsy who developed persistent hyperoral behaviour after prolonged status epilepticus lasting several hours. On resolution of the seizures and recovery of consciousness, he
had a voracious appetite and indiscriminate eating habits which included paper towels, plants, styrofoam cups, and even faces. At one point he came from a catch-all 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 and sexual gestures, often striking with surgical instruments. He had wandered about the ward picking up whatever he could find and putting it into his mouth. Neuropathological examination disclosed an infarction in the left posteromedial cerebral cortex, 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, eat food from other's 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 judgement and memory. In addition, the patient became placid, became manually exploring his surroundings and grabbing at objects, and occasionally exposed himself to others. On examination, he was passive and mute, except for a 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 showed normal visual acuity, visual fields, motor testing, and reflexes. The patient's CT showed disproportionate atrophy of the temporal lobe, with or without 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 prominent frontal lobe atrophy with Pick cells but no Pick bodies, and milder anterior temporal atrophy.

Two of these patients illustrated that hyperoral behaviour can be lethal. Both patients had the Klüver-Bucy syndrome with a tendency to engage in oral exploration of objects and hypsarrhythmia, or an excessive 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 etiologies 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 'emotion' centres, such as hamartomas and germinomas, can lead to hyperphagia. The Kleine-Levin syndrome presents with periodic hyperphagia and hypersomnolence, and there is often a temporal atrophy with this syndrome who asphyxiated on a sausage. Additional causes of hyperphagia include bilateral thalamic infarcts and congenital disorders such as the Frader-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.

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

Anxiety disorder accounts 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. 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. 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 as people age. 4 A possible explanation for this anomaly might be that age related biological factors reduce the sensitivity of older people to these risk factors.

Alcohol dependent patients also present 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 of the variables using the patient 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. More detailed information on the population and the methods used has been reported elsewhere. Information regarding psychiatric symptoms was derived from psychiatric examinations conducted by the investigators using the comprehensive psychopathological rating scale (CPRS). The physicians were trained and attended regular meetings to ensure that the CPRS was administered standard 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 information 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 for individuals living in the community according to the ADL scale. 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. Diagnosis was made 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 every patient was not 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 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