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 fulminant illness.

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 lymphoma, 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. These signs may be the initial manifestation of 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. 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-relapsing neuropathy due to non-cutaneous T cell lymphoma infiltrating peripheral nerves, to vasculitis or, more likely, to the neurotoxic effects of cytotoxins. Cytotoxins, especially TNF, are secreted in large amounts in macrophage activation syndrome, and TNF can induce general side effects and cerebral damage.4

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Paraneoplastic opsonolus associated with cancer of the gall bladder

Opsonolus is an ocular dyskinesia consisting in ample, conjugated, arrhythmic, multifocal ocular movements which persist even with the eyes closed.1 This syndrome has been described during the course of different cancers.2 In infancy, neuroblastoma is the cancer most often associated with opsonolus (in 2% to 7% of the cases).3 In adults, opsonolus is less common. Nevertheless, it is associated with a tumour in 20% of cases.4

Here, we report a case of opsonolus associated with a cancer of the gall bladder. A 72 year old, treated hypertensive woman, experienced the sudden onset of vertigo followed by impaired consciousness. Her first examination at our hospital showed she had acute hypertensive encephalopathy. Her Glasgow score was 13. She showed opsonolus associated with a bilateral kinetic cerebellar syndrome. The cranial nerves were intact and there was no sensory or motor deficit. Complete physical examination only showed conjunctival icterus.

Brain MRI showed a left frontal angiomata measuring 7 mm in diameter without any impingement on 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), γ-glutamyl transferase (100 IU), CA 19-9 (1347 U/ml) and CA 125 (111 kU/l). Abdominal ultrasound showed a heterogenous, polypoid tumorous structure in the gall bladder associated with hypochogenic 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 choleretic, microscopic picture consistent with adenocarcinoma most suggestive of a pancreatico-biliary origin.

Tests for anti-Hu, anti-Ri, and anti-Yo antibodies were negative. Immunoglobulin IV (0-4 g/kg/day) and corticosteroids (Solumedrol, 0-5 g/day for five days) was ineffective. The patient died five weeks later. No necropsy was performed.

The diagnosis of opsonolus remains clinical. It usually has an abrupt onset.1 It is probably the result of a diecephalic or mesencephalic lesion with production of the abnormal movement by removal of normal saccadic generator inhibition.2 Dysfunction of the pause neurons, which play a part in inhibiting the phasic neurons responsible for the appearance of jerks, is likely. The first eye angiomata does not explain the opsonolus. The opsonolus was considered paraneoplastic because it was not associated with an infectious or tumoral cerebral lesion. Opsonolus possible causes (toxic, metabolic, degenerative, and vascular) were excluded. The normal MRI, lumbar punctures, and the absence of anti-Ri antibodies, which have been associated with paraneoplastic opsonolus occurring with carcinomas of the breast, did not cast doubt on the diagnosis. Breast cancer and small cell cancer of the lung represent 70% of reported cases associated with opsonolus in adults1 and are sometimes discovered during necropsy. 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 opsonolus.

Lethal hyperoronal behaviour from the Klüver-Bucy syndrome

Clinicians have not sufficiently appreciated the danger of hyperoronal behaviour in neurologic disorders. A particular case of this behaviour is the Klüver-Bucy syndrome.1 Originally described in monkeys after anterior bitemporal lobectomies, this syndrome includes indiscriminate dietary behaviour and a tendency to examine objects by mouth.2 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 hyperoronal behaviour.

Patient No 1 was a 40 year old man with epilepsy, who developed persistent hyperoronal 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 faeces. At one point he came from a carries 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 to sexual nature during an examination, his speech was dysarthric but fluent, and he had difficulty with the comprehension of multiple step commands. He had trouble with spatial orientation, 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 hypoplasia 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 snuffing out with surgical forceps. He had wandered about the ward picking up whatever he could find and putting it into his mouth. Neuropathological examination disclosed bilateral temporal lobe infarcts and congestion 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 age 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 occasional 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 disproportion- ate frontal lobe atrophy, and the patient was diagnosed with frontal-temporal 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 undigest food. There was massive aspiration of poorly masticated food in the pharynx, and the patient did not respond to resuscitation efforts. Non-lesional demyelination examination suggested pronounced frontal lobe atrophy with Pick cells but no Pick bodies, and milder anterior temporal atrophy.

These two patients illustrate that hyper- oral behaviour can be lethal. Both patients had the Klüver-Bucy syndrome with a tendency to engage in oral exploration of objects and hypervoraciously undigestible inedible objects. In this syndrome, the hyperoral behaviour probably results from damage to the amygdala in the anterior temporal lobes. Patients with frontotempo-

Anxiety disorders

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 out-patients. Several recent studies that have used community based samples have reported mixed results, although a vast majority found a decrease in prevalence in the elderly population. 

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 out-patients. Several recent studies that have used community based samples have reported mixed results, although a vast majority found a decrease in prevalence in the elderly population. While there have been various attempts to identify factors that contribute to the development of anxiety disorders in the elderly, such as depression, these have not been successful in explaining the anomaly. A possible explanation for this anomaly might be that age related biological factors reduce the sensitivity of older people to these risk factors.

Alcohol dependence is a significant 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. This study made no diagnoses of anxiety disorders and the patients were all at an early stage of dementia.

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 out-patients. Several recent studies that have used community based samples have reported mixed results, although a vast majority found a decrease in prevalence in the elderly population. These results are of interest, as factors reported to be highly associated with anxiety disorders such as decreased physical health, bereavement, isolation, and decreased autonomy are more likely to be found in the elderly population. A possible explanation for this anomaly might be that age related biological factors reduce the sensitivity of older people to these risk factors.

Anxiety disorders are common in elderly persons with dementia and are 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. 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 esti- mate the prevalence of anxiety disorders in non-demented and demented subjects and to identify some demographic factors that are 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 Kungsholmens parish of Stockholm, Sweden. A total of 1101 persons comprised the study popula- tion; more details about the investigation and the methods used has been reported elsewhere.

Information regarding psychiatric symp- toms was derived from psychiatric examina- tions conducted by the physicians using a comprehensive psychopathological rating scale (CPRS). The physicians were trained and attended regular meetings to ensure that the CPRS was administered methodically and consistently. Information on psychiatric his- tory 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. Impairment in activities of daily living (ADL) was determined from the scores made using the DSM-III-R criteria to maintain accuracy with previous phases of the study. The severity of dementia was clas- sified according to the Washington clinical dementia rating scale (CDR). The psychi- atric diagnoses were combined in three groups: depressive, psychotic, and anxiety disorders. DSM-IV criteria were used with the modification that ever present—for example, dementia—the diagnosis was 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 reg- istered.

To analyse differences in the prevalence of depressive, psychotic, and anxiety disor- ders, a one way analysis of variance was perform- ed with severity of dementia as the between subjects factor. Odds ratios (ORs) and 95% confidence intervals (95% CI) 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 diag- nosed 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 disorders were combined. The prevalence of anxiety disorders was highest among the non-demented group and decreased with increasing severity of dementia.
Lethal hyperoral behaviour from the Klüver-Bucy syndrome.

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