back of thyroid hormones on pituitary TSH secretion. Thus basal ultrasensitive TSH level has become an important tool to detect hyperthyroidism, replacing the TRH-stimulation test.

The aim of our study was to determine whether chronic treatment with levodopa was able to lower the basal TSH levels, measured by an ultrasensitive assay, in Parkinsonian patients and thus be responsible for a false diagnosis of hyperthyroidism in such patients.

Thirty two Parkinsonian patients, mean (SD) age 73-12 (9-32) years; range: 50-88 treated with levodopa (187.5 mg/day to 700 mg/day) were compared to 108 normal control subjects. These patients were clinically euthyroid, had a normal thyroid clinical examination and normal levels of free T3. Blood samples were drawn at 8:00 am in fasting patients, from 8 to 10 am after the last levodopa dose. The determination of free T3, free T4 and ultrasonic TSH were performed. These patients were not receiving any other drugs known to modify thyroid function. Thirty age matched controls and 108 normal controls were studied.

In our study, basal TSH levels were measured by an ultrasensitive TSH assay, which uses a monoclonal antibody specific to TSH (RIA Amersham). Ultrasonic TSH was measured by radioimmunoassay, using a monoclonal antibody (RIA Behring). The sensitivity of this TSH assay is 0.02 mU/L, the intra-assay and inter-assay coefficient of variations are 3.5% and 5% respectively. Comparisons of means were made with Student's t-test. The mean (SD) age of patients 76-05 (1-02) mU/L; range: 0-51-4 from controls: mean (SD) 1-45 (1-02) mU/L; range: 0-44-3-87 (table).

In conclusion, the antiparkinsonian therapy with levodopa does not modify basal TSH levels measured by an ultrasonic assay. Thus ultrasonic TSH evaluation is as efficient a method to detect hyperthyroidism in patients treated with levodopa as in the general population.

Clinical diagnosis of hyperthyroidism is often difficult in patients with Parkinson's disease because symptoms such as tremor, weight loss and sweating are common to both diseases, because thyrotropin only produces few symptoms in elderly patients, and because Parkinson's syndrome in elderly patients. Therefore, a thorough clinical examination and blood tests are necessary before treatment with levodopa.

### Table

<table>
<thead>
<tr>
<th>LevoT3 treated patients</th>
<th>Controls</th>
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<tbody>
<tr>
<td>Age (years)</td>
<td></td>
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<tr>
<td>73-1 (9)</td>
<td>76-0 (9)</td>
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<tr>
<td>Free T3 (pMol/l)</td>
<td>3-38-3</td>
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<tr>
<td>8-26</td>
<td>13-40-3 (3-24)</td>
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<tr>
<td>1-07-3-5</td>
<td>1-71 (1-05)</td>
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<td>p</td>
<td>1-05-1-7</td>
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Free T3, free T4 and ultrasonic TSH levels in levodopa treated Parkinsonian patients compared with age-matched controls, mean (SD). NS: not significant by Student's t-test.

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Vestibular and ventilatory dysfunction in sensory and autonomic neuropathy associated with primary Sjögren's syndrome

Subacute sensory neuropathy was described by Denny-Brown in association with carcinoma and has recently been described in other primary Sjögren's syndrome (SS) patients. The primary pathology is in the dorsal root ganglia and consists of lymphocytic infiltration and neuronal cell destruction. We describe a patient with cancer and sensory and autonomic neuropathy who had severe impairment of proprioception up to and including the knees. Romberg's sign was positive and the gait was severely ataxic. There was bilateral punctate keratitis in the interpalpebral distribution and the Schirmer's test was 14 mm on the right and 4 mm on the left (normal > 5). On further examination, the Schirmer's test revealed no lacrimation from either eye.

Significant results of investigations were that the RA latex test was negative, antithyroidoprotein antibody level was 8 (normal < 20) and smooth muscle antibody titre was 1:40. Screening for other causes of neuropathy, including extractable nuclear antigens, was negative. CSF protein was initially 660 mg/L (normal < 400), CSF IgG was 55 mg/L (normal 10–60) and CSF IgA/albumin was 0-14 (normal < 0-11). On a later occasion the CSF protein level was 1,100 mg/L. CSF electrophoresis did not reveal oligoclonal bands. Motor nerve conduction velocity was normal but sensory action potentials were absent in the right median, ulnar and sural.
nerves. A sural nerve biopsy showed axonal degeneration, with loss of large diameter fibres, and a sparse perivascular lymphoid infiltrate around occasional perineurial vessels. Biopsies of the lip and conjunctiva were normal. No evidence of malignancy was found.

When the limbs and trunk were reduced to the primary SS causing sensory and autonomic neuropathy was made. He was treated with intravenous methylprednisolone (500 mg per day for 5 days) and then given oral prednisone (100 mg/day). His gait improved and there was some improvement in joint position sense, although the numbness of the limbs and painful dysesthesiae persisted. After one month the steroid dose was tapered. When the prednisone dose was reduced to 80 mg/day he became worse and developed tight band-like sensations about the trunk. He developed dyspnoea and tachypnoea with resting respiratory rates up to 30 per minute. At this time he showed no signs of anxiety or emotional distress. Spirometry was normal and a chest radiograph was normal. Blood gas analysis showed a compensated respiratory alkalosis. Ten weeks after the first course, he was given a further course of 5 days of 500 mg/day of intravenous methylprednisolone and then continued on reducing doses of oral prednisone and he was treated with azathioprine (125 mg/day). Despite this, he then complained of blurred vision and intermittent oscillopsia. On examination, horizontal nystagmus was present for several weeks. Electromyography showed absent responses to caloric stimulation on two occasions. CT and MRI scans of the brain were normal. He was treated with both plasma exchange and intravenous gamma globulin and continued on prednisone and azathioprine for 8 months. At review 18 months after his first presentation, he continued to have severe ataxia of gait and widespread sensory loss with no remaining normal pain sensibility. His pattern of breathing had changed so that he no longer had tachypnoea, but had episodes when he noticed that he had ceased breathing. His wife noticed periods of apnoea lasting several minutes during sleep. A recording of respiration during sleep was performed on one occasion but apnoeic episodes were not observed.

Our patient had primary Sjögren’s syndrome and has clinical and electrophysiological evidence of a subacute sensory neuropathy which has been well described in primary SS. 

There was no evidence of CNS disease, which is described in SS.

The new features in this patient are the vestibular and ventilatory abnormalities. The ventilatory problems consisted of dyspnoea and tachypnoea but later changed to intermitent apnoea. We propose that this may have been due to involvement of primary sensory neurons of pulmonary stretch receptors in the nodose ganglia by the neuropathic process. The pulmonary stretch receptors mediate the Hering-Breuer reflex which results in the termination of inspiration and the initiation of expiration. The initial dyspnoea and tachypnoea may have occurred as an irritant phenomenon while the later apnoea may have been due to neuronal loss. Similar involvement, in the nodose ganglion, of the primary sensory neurons of the carotid and aortic body chemoreceptors may also have contributed to the ventilatory disturbance. Slowing of respiration has been observed in rabbits with experimental allergic encephalomyelitis, where there is major pathology in the dorsal root ganglia, and has been attributed possibly to lesions in the nodose ganglia interrupting the Hering-Breuer reflex.

Our patient’s oscillopsia may be due to a peripheral vestibular lesion as electrophysiology showed absent caloric responses and there was no evidence of a central lesion on CT or MRI. We suggest that his vestibular dysfunction represents an extension of the pathological process to include the primary sensory neurons of the vestibular (Scarpa’s) ganglia. Vestibular neuropathy has not previously been reported in subacute sensory neuropathy.

In our patient, high dose corticosteroid treatment was beneficial, but the patient relapsed when the dose was reduced. Treatment with azathioprine, and intravenous gamma globulin were of no apparent benefit. After the end of treatment he remained stable. The clinical response of this patient suggests that benefit can be obtained by immunosuppressive treatment during the initial phase of inflammation.