symptoms had jejunoileal considerable and appeared, affected state was of pseudobulbar sign. Her and per hour. range examination showed biopsy with stabilised months probably disappeared; the unaffected creased to 1.96 of disorder drome, 47 supranuclear Progressive Richardson Olszewski weight gain did not increase to 1990. An of paresis about 188, she kept distal oculomotor palsy. Whipple's disease from an anterior portion cerebral arteries. This necessitates "tumoral" decision may explain why antibodies with good BBB diffusion are not effective in this CNS disease.

**Table Binswanger's clinical and neuropathological criteria for the diagnosis of "Binswanger's disease"**

<table>
<thead>
<tr>
<th>Clinical criteria: (verbaism from reference 2, page 1184)</th>
</tr>
</thead>
<tbody>
<tr>
<td>the disease begins at the onset of senility (early in the fifties) or in advanced old age (early in the sixties); slow impairment of intellectual capabilities manifesting primarily by the progressive impairment and ultimate disorientation. In the vast majority of cases the onset of dementia is slow and insidious; most frequently observed are apasic disturbances (as in the present case), hemianopia or hemiparesis with loss of the sense of position, or touch; these circumscribed deficits are of a stable character during the fully developed disease and they are combined with the slow and relentless deterioration of intellectual performance; (..) the patients resemble decerebrate laboratory animals.</td>
</tr>
</tbody>
</table>

**Neuropathological criteria: (from reference 2, page 137)**

- We found a pronounced atrophy of the hemispheric white matter, either restricted to one or more gyri in one brain area or of several hemispheric regions affected with variable severity; these changes are most clearly found in the area of the occipital and temporal lobes, so that the temporal and occipital horn are widened into bag-like cavities, while the anterior portion of the lateral ventricle shows relatively little enlargement and the frontal white matter is spared.
- The cortex does not show any remarkable macroscopic change apart from a slight narrowing. Invariably, these cases show severe atrophy of the cerebral arteries.

**Binswanger's clinical and neuropathological criteria for "Binswanger's disease"**

Bennett et al have made a timely and critical attempt to standardise the diagnostic criteria for "Binswanger's disease". They have based their suggestions on current English and French literature. It might be of interest to compare these modern criteria to Binswanger's original description, which had only been available in English in a grossly truncated form.

The table shows the clinical hallmarks
and the neuropathological findings which Binswanger considered essential to a diagnosis of ‘general degeneration of the nervous system’ (the so-called ‘chronica progressiva’). These ‘reliable criteria’ were introduced to differentiate ‘encephalitis subcorticalis’ from ‘arteriosclerotic brain degeneration’ (which also affects the cortex) that may be a ‘general paralysis of the insane’ and from senile dementia, which he knew also could be associated with white matter changes.1

The similarities between Bennett’s1 and Binswanger’s criteria are obvious. Nevertheless, several striking discrepancies appear noteworthy. The white matter atrophy in Binswanger’s patients was most pronounced in the occipital and temporal areas, whereas radiological changes are most commonly found in the frontal lobes. According to Binswanger, ‘encephalitis subcorticalis’ slowly and relentlessly progressed to a state of decompensation, whereas Bennett et al excluded patients with severe dementia. Binswanger assumed that arteriosclerosis was the cause of disease and mentioned the invariable presence of cerebral arteriosclerosis (which he did not describe extensively). He did not describe hypertension or other evidence of systemic vascular disease.

It has already been pointed out that the relationship between Binswanger’s findings and the modern ‘Binswanger’s disease’ remains open to question. Binswanger did not present a full account of the histopathological features. This was left to Alzheimer, who first used the term ‘Binswanger’s disease’, and to Nissl. Inconsistencies in Binswanger’s original description may support the speculation that he eventually regarded the differentiation of such vascular dementias as too difficult or too unwarranted.

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Pseudotumour cerebri and chronic benzene hexachloride (lindane) exposure

Pseudotumour cerebri, the syndrome of idiopathic intracranial hypertension and papilloedema as a result of a tumour or obstructive hydrocephalus, may be associated with exposure to drugs or toxins.1,2 We report a patient, repeatedly exposed to the pesticide benzene hexachloride (lindane), who developed the syndrome of intracranial hypertension.

A 45 year old man (weighing 80 kg) who kept hounds noted fleeting episodes of blurred vision in his right eye usually related to changes in posture. The blurring became persistent after three months and then he developed fleeting black spots in his left eye. Shortly after he noticed early morning occipital headaches and tinnitus. He had used benzene hexachloride at least twice a month for about 30 years to rid his beagle hounds of fleas and ticks. He had used a 20% cream concentrate to make dip and spray applications but wore a mask and appropriate protective clothing. He was well built but not obese. His neurological examination yielded normal results, except for fundal findings: his right eye showed a very best corrected visual acuity was 6/36 OD and 6/9 OS. He had a right relative afferent pupillary defect. Ocular motility and slit lamp examinations were normal. Intraocular pressures were 14 and 23 mm Hg, respectively. Ophthalmoscopic examination showed distinct swollen optic discs with small cups, loss of the nerve fibre layer in the right eye, and a small pseudodrusen in the left eye, typical of chronic papilloedema. Goldmann perimetry showed visual field loss characteristic of chronic papilloedema.

MRI of the head was normal except for a few small areas of white matter lesions; venous sinus thrombosis was not seen. A spinal tap showed an opening pressure of 400 mm CSF with one monocyt per cu mm, protein 0.34 Gm/l, glucose 2 mmol/l, and no bacterial infection. Other laboratory values were notable only for elevated cholesterol and triglyceride concentrations and mildly abnormal results of liver function tests. Thyroid function tests were normal, but antinuclear and antineutrophil cytoplasmic antibodies were negative. Toxic screens for lead, mercury, and arsenic were negative. Management included dietary advice (weight loss), diuretics, and prednisone, but he subsequently had 23 mm Hg, respectively.

Fenetisation because of progressive visual field loss. Ten months after diagnosis his field defects were stable, but his visual acuity remained impaired.

Lindane, a gamma isomer of hexachlorocyclohexane used as a pesticide and an ectoparasicide, is metabolised by the liver and distributed and stored in depot fat and other lipohelial tissue, the latter commonly prescribed topically as a 1% solution for scabies but is available in concentrations of 0.5%-99%; our patient used a 20% veterinary concentration for his dogs. Lindane is a powerful CNS stimulant known to cause headache, nausea, vomiting, diarrhoea, convulsions, muscle spasms, respiratory failure with cyanosis, coma, and death.2,4 Optic neuritis after ‘improper use’ of lindane powder has also been reported.1 Heuser and Heuser briefly described ‘pseudotumour cerebri’ in a farmer with localised brain oedema, after ‘prolonged professional inhalation’ of lindane; but the appearance of the optic discs and elevated intracranial pressure were not reported. The mechanism of lindane toxicity is unknown, although it is highly lipid soluble and may act as a gamma-amino-butyric acid-A (GABA-A) receptor antagonist to produce convulsive effects and interference with the production and utilisation of free ammonia in the brain.3,5 Chlorocodone, a cyclodene insecticide which also induces seizures, has been implicated in causing pseudotumour cerebri by inhibition of ATPase activity, resulting in impaired resorption of cerebrospinal fluid across the arachnoidal villi.1 Lindane may have similar effects on the arachnoid villi as they are both lipid soluble, neurotoxic chlorinated hydrocarbons.

Our patient stopped using lindane when the association of pseudotumour cerebri and lindane was brought to his attention; this was coincidentally reinforced when a neighbour’s puppies convulsed and died after exposure to a 20% solution. Despite discontinuation of the pesticide the patient’s intracranial pressure remained elevated and headaches continued 11 months later when a lumbo-peritoneal shunt was inserted. Removal of the toxin should result in alleviation of increased intracranial pressure. Perhaps the patient’s liver damage was caused by previous chronic alcohol consumption or exposure to lindane is unclear. The relation with lindane exposure may not be coincidental because other pesticides have been linked to pseudotumour cerebri in the past.2 The use of lindane should be discontinued when patients have unexplained raised intracranial pressure.

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Motor neuron syndrome in the arms after radiation treatment

Radiation myelopathy is a rare but well established complication of radiotherapy leading to diagnostic difficulties with neurological complications of the primary neoplasm, like epiduralis or spinal metasta-sis. We report a rare case of radiation motor neuron syndrome that developed three years after local radiotherapy in which spinal cord magnetic resonance imaging (MRI) showed a cervical spinal cystic lesion.

A 44 year old man without relevant history presented with dyspnoea and a rapidly growing cervical anterior mass. We found a mal-
Binswanger's clinical and neuropathological criteria for "Binswanger's disease".

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