and the neuropathological findings whichBinswanger considered essential to a diagnosis of 'Binswanger disease'. These 'reliable criteria' were introduced to differentiate "encephalitis subcorticalis" from "arteriosclerotic brain degeneration" (which also affects the cortex) and the "general paralysis" of the insane and from senile dementia, which he knew could also be accompanied by white matter changes.2

The similarities between Bennett's 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 lobes, whereas radiological changes are most commonly found in the frontal lobes. According to Binswanger, "encephalitis subcorticalis" slowly and relentlessly progressed to a state of decrepitude, 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 called "angiosclerosis") in his patients.2

It has already been pointed out that the reference to Binswanger's findings and the modern "Binswanger's disease" remains open to question. Binswanger did not present a full account of the histopathological changes. This is left to Alzheimer, who first used the term "Binswanger's disease",1,4 and to Nissl.1 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.2

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Pseudotumour cerebri and chronic benign hexachloride (lindane) exposure

Pseudotumour cerebri, the syndrome of idiopathic intracranial hypertension and papilloedema, is one of a tumour-like 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 intracranial hypertension and papilloedema. 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 intermittent blurring 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 used a low concentration 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 and findings are the 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 21 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 mild 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 white matter lesions; venous sinus thrombosis was not seen. A spinal tap showed an opening pressure of 400 mm CSF with one monocyte per cu mm, protein 0.34 Gm/l, glucose 2 mmol/l, and no neoplastic cells. Of the laboratory values were notable only for elevated cholesterol and triglyceride concentrations and mildly abnormal results of liver function tests. Thyroid function tests were normal; rheumatoid factor and antinuclear 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 mild optic nerve fiber sheath meningitis 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 ectoparasiticide, is metabolised by the liver and distributed and stored in depot fat and other lipophilic tissues.2 Heuser1 reported systemic poisoning caused by an unknown organophosphate (lindane), which was found topically as 1% solution 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.3,4 Optic neuritis after "improper use" of lindane powder has been also reported.2 Heuser and Heuser briefly described "pseudotumour cerebri" in a farmer with localised brain oedema, after "prolonged professional ingestion" of lindane; but the appearance of the optic discs has been reported to cause a pseudotumour cerebri but not in the patient under description. Heuser and Heuser reported a case of "pseudotumour cerebri" in a farmer with localised brain oedema, after "prolonged professional ingestion" of lindane; but the appearance of the optic discs has been reported to cause a pseudotumour cerebri but not in the patient under description.

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 high 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. Nevertheless, lindane may be present in fat cells for an extended period and have a long lasting effect on CSF absorption. Whether the patient's liver damage was caused by previous and 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 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 epiduritis or spinal metastasis. We report a rare case of radiation-induced syndrome that developed three years after local radiotherapy in which spinal cord magnetic resonance imaging (MRI) showed a central cervical spinal cord lesion, as well as a thoracic lesion.

A 44 year old man without relevant history presented with dysphonia and a rapidly growing cervical anterior mass. We found a mal-