and the neuropathological findings which Binswanger considered essential to a disease with "hyperactivity of the subcortical chronicosa." These "reliable criteria" were introduced to differentiate "encephalitis subcorticalis" from "arteriosclerotic brain degeneration" (which also affects the cortex), from "general paralysis of the insane" and from senile dementia, he knew which could also be accompanied by 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 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 de cerebration, 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 was the hallmark of Binswanger's disease,2 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 benign hexachloride (lindane) exposure

Pseudotumour cerebri, the syndrome of idiopathic intracranial hypertension and papilloedema, is a feature of a tumour, obstructive hydrocephalus, may be associated with exposure to drugs or toxins.12 We report a patient, repeatedly exposed to the pesticide benzene hexachloride (lindane), who developed the syndrome of hypertensive hydrocephalus.

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 a right field defect 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 to 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 findings: his right eye was 2/66 OD and 6/9 OS. He had a right relative afferent pupillary defect. Ocular motility and slit lamp examinations were normal. Intracranial pressures were 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 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 bacteria or 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; 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 bilateral oedema from nephritis 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 lipohilic tissues.6 It is 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 depressant known to cause headache, nausea, vomiting, diarrhoea, convulsions, muscle spasms, respiratory failure with cyanosis, coma, and death.34 7 Optic neuritis after "improper use" of lindane powder has also been reported.8 Heuser and Heuer 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 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 (GABA) receptor antagonist to produce convulsive effects and interference with the production and utilisation of free ammonia in the brain.9 10 Chlordecone, a cyclodiene 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.11 Lindane may have similar effects on the arachnoidal 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 lindane caused permanent or prolonged alteration of the arachnoid villi. Alternatively, 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 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 epiduritis or spinal metas- tasis. We report a rare case of radiation induced motor neuron syndrome that developed three years after local radiotherapy in which spinal cord magnetic resonance imaging (MRI) showed a central 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-
pigian carcinoma invading the thyroid and the right wall of the trachea. He was treated with ablation of the right thyroid lobe, tracheotomy, and three courses of chemotherapy (CCDP, 5FU) followed by radiation therapy. The thickness of the thyroid was 4 cm, Sagittal and axial CT scans showed a cystic lesion with well defined borders and a signal isointense with respect to CSF on T1 weighted images (TR = 500 ms, TE = 15 ms) and T2 weighted images (F1S P = 10°, TR = 100 ms, TE = 27 ms) within the spinal cord from C4 to C6 and a smaller cavity behind the body of the atlas (figure). There was no enhancement of this image after IV injection of gadolinium and the cerebellar tonsils were within the normal range. The fatty replacement of the spinal bone marrow due to radiation therapy accounted for the high signal intensity of the cervical spine on the same level. The magnetic MRI was normal. There was no change in neurological condition after eight months.

This case fulfils the strict criteria established for an accurate diagnosis of radiation myelopathy. The authors preferred the term "radiation myelopathy" to "post-radiation myelopathy" because it is a well known term used to describe lesions following radiation therapy. The clinical presentation was consistent with a compression due to metasases excluded by neurological investigations; and a latent period of at least nine months before the development of symptoms. Radiation myelopathies may be divided into five main types: 1) transient myelopathy (the more common and generally marked only by a Lhermitte sign); 2) chronic progressive myelopathy; 3) radiation myelopathy; 4) radiation myelopathy without significant sensory change, spheincter disturbance, or clinical involvement of corticospinal tract. There was electrophysiological evidence of an involvement of the anterior horn: giant potentials and normal motor and sensory conduction velocities. MRI data agree with this location. A coincident, however, cannot be excluded but the only alternative diagnosis would be idiopathic syringomyelia for which the clinical picture, without a considerable sensory problem, the rapid progression of symptoms, the absence of Chiarl or kyphoscoliosis would be atypical. There was no evidence for a syringa secondary to an intraspinal tumour (thoracic MRI normal). Furthermore, before the wasting of the hands, a Lhermitte sign occurred, which is a common symptom of radiation myelopathies.

Nevertheless, to our knowledge such a clinical presentation has not previously been described. All reports of delayed lower motor neuron syndrome after radiation concern the legs, generally after irradiation for lymphomas or testicular malignant tumours. Most of these motor neuron syndromes usually present as a progressive weakness of the legs without sensory or sphincter disturbance. Development stops after some months or years and so far the prognosis is better than in other forms of myelopathies. Signs suggestive of a cervical anterior horn involvement (muscle wasting, fasciculations, tendon reflex loss) have been mentioned in some cases but they were associated with corticospinal and long sensory tract lesions (mainly Brown-Sequard syndrome).

The pathogenesis of radiation myelopathies remains controversial, but vascular changes are almost constant and are considered as the primary damage rather than neuronal or immunological disorders. As the grey matter is classically considered less vulnerable to radiation than white matter, the anterior horn is presumed to be less or not involved, although in several anatomical cases of radiation myelopathies an extension of lesions towards the anterior horns has been reported. 1, 9 There are no well documented anatomical reports of motor neuron syndromes after radiation and some controversy exists as to the site of lesions (anterior horn, motor anterior roots, or plexus), but clinical and electrophysiological data from most cases suggest an involvement of the anterior horns. 1

We know of only one report of MRI data from a patient with motor neuron syndrome of the legs, and the transversal TI weighted images were normal. 10 In a case reported by Lechevalier et al a patient had Brown-Sequard syndrome after radiation with proximal wasting and weakness of the left arm. Histological examination of the cervical spinal cord showed a centrospherical necrosis with complete neuronal loss of the left anterior horn. Perhaps in our case the spinal cavity on MRI was secondary to necrosis involving the cervical anterior horns.

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The incidence of muscle cramp

In true muscle cramp there is sudden involuntary and painfull shortening of muscle and visible or palpable knotting of muscle often with abnormal posture of the affected joint which is relieved by stretching or massage. 1 In myelopathic lesions on data on muscle cramp are sparse and based upon empiricism. Wilder in 1940 found that 19% of young soldiers, 33% of workers in a silk mill, and 67% of medical students had experienced muscle cramp. The incidence in a group of internal, neurological, and psychiatric patients was 40%. 2 In a study of 1947 in 100 men and 100 women aged 15-80 years, Hall found that 56% had suffered from cramp; of those aged 50 years or more, 70% had suffered. 3 Of 121 college students questioned in 1956, Norris found that 115 had experienced spontaneous muscle cramp at least once, 18

Figure Sagittal T1 weighted SE (500 ms) 15 ms) image showing cystic hypertensive cavity affecting spinal cord from C4 to C6 without ectopic tonsile. Note fatty replacement of cervical spine bone marrow from C2 to C6, responsible for hypointense on T1 weighted images.

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