A 54 year old farmer, while bailing hay, recalled a sudden headache, “as if struck by lightning” before he lost consciousness. Brain CT showed widespread subarachnoid and intraventricular blood and acute obstructive hydrocephalus (figure top row).

The visual acuity was limited to detection of motion in both eyes. The red reflex and pupillary responses were absent. The posterior pole of the eye could not be visualised due to extensive vitreous haemorrhage. (Figure middle row: absent red reflex with appearance of “black
pupils” after maximal dilatation; bottom row: red reflex in a normal subject for comparison.) Retinal haemorrhage in subarachnoid haemorrhage may rupture the hyaloid membrane to enter the vitreous compartment (Terson’s syndrome). Although the prognosis for spontaneous visual recovery is good, vitrectomy should be considered in those patients with bilateral vitreous haemorrhage who have not improved after 6 to 12 months.1

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NEUROLOGICAL PICTURE

Chronic misuse of paint thinners

A 17 year old man who had chronically misused paint thinners for 3 years was examined for occasional occipital headache after an abstinence of 4 months. He was neurologically normal without any cognitive or psychological impairment. Fluid attenuated inversion recovery images showed multiple high intensity lesions in the subcortical and deep white matter (figure, A, B).

The major constituent of thinner is toluene, which is a neurotoxic solvent. Brain MRI in chronic thinner or toluene misuse may show cerebral and cerebellar atrophy, atrophy of the corpus callosum, and loss of grey-white matter contrast. T2 weighted images show scattered high signal lesions in the white matter and brainstem due to demyelination or gliosis, and low intensity lesions in the basal ganglia, thalami, and subcortical white matter, which are postulated to be caused by iron deposition or toluene accumulation into the cell membrane due to its lipophilicity.1–3

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