Sir: Paradichlorobenzene (PDCB) is mainly used as a moth repellent, mildew control agent and as a space deodorant. In spite of the broad spectrum of uses there are few reports of oral, inhalation or skin contact toxicity of this compound to man. Cataract formation, hepatic damage, anaemia, thrombocytopenia and lymphocytopenia have been reported following exposure to PDCB. However, no toxicity to the central nervous system (CNS) had been reported. We report a patient who developed cerebellar ataxia and speech disturbance following 6 years exposure to PDCB.

A 25 year old woman was admitted to our hospital complaining of gait disturbance and dysarthria. She had been diagnosed as suffering from neurosis for 6 years. When she was 19 years old (1980) the unusual idea that there were many ticks in her room occurred to her and she learned to use moth balls of PDCB in her bedclothes and pillow as well as in her wardrobes. Moreover she began to grind them into powder and scattered it everywhere in her room. She put on her underwear which had been kept in bags filled with PDCB powder. Her mother says that there had been a strong odour of PDCB in her room for the last few years. In January 1986, clumsiness of both hands became apparent. She could not use chopsticks. She also found difficulty in gait and speech. Gradually her signs and symptoms became worse and she could not stand without support after 2 months. There was no fever. She was admitted to a hospital where neurological examination revealed severe cerebellar ataxia, dysarthria, moderate weakness of all limbs and hyporeflexia. Computed tomography of the brain, four vessel angiography and CSF findings were normal. Her symptoms and signs gradually improved after admission. A month later she was admitted to our hospital for further evaluation.

She was alert and well-nourished; neurological examination revealed moderate limb and truncal ataxia, dysarthria, hyporeflexia, hypotonia and mild proximal weakness of the four limbs. Blood pressure was 120/80 mmHg with normal cardiac rhythm and there was no orthostatic hypotension. Normal laboratory data included blood count, electrolytes, liver and kidney function, serum creatine kinase, fast blood glucose, lactate, pyruvate, serologic reaction for syphilis and SLE, and thyroid function. Serum antibody titres to herpes, mumps, ECHO, varicella, Coxackie, polio, influenza, mycoplasma and EB virus were within normal limits. CSF findings were normal and oligoclonal bands were not detected. CSF culture and stains for bacteria including anti-fast bacilli and fungi were negative. Magnetic resonance imaging of the brain was normal. EEG, nerve conduction velocity, visual evoked potentials, blink reflexes, somatosensory evoked potentials to median nerve and posterior tibial nerve stimulation were also normal. Brainstem auditory evoked potentials (BAEPs) showed a marked delay of waves III, IV and V and elongation of II-V interval while latencies of waves I and II and I-II interval were normal (fig A). Her symptoms gradually improved and became minimal in 6 months after onset. BAEPs re-examined 8 months later were normal (fig B).

PDCB is a white crystalline compound which has been used for several decades as a repellent, space deodorant and fungicide (mildew-control agent). However, toxicity to the CNS of this substance had not been reported. In our case, continuous long-term exposure for 6 years is characteristic, and inhalation and local contact are considered to be the predominant routes of exposure. In experimental animals subjected to PDCB, intense eye and nose irritation, tremors and twitches of the extremities, a "mark time" reflex, a loss of the righting reflex, a definite nyctagmus, rapid but laboured respiration, reversible granulocytopenia, kidney and lung injury, and some deaths have been reported.\(^1\)\(^2\) Hollingsworth \etal also observed marked tremors, weakness, loss of weight, eye irritation, unkempt appearance, and unconsciousness in rats, guinea pigs, and rabbits subjected to repeated 8-hour exposures, 5 days a week, to the high concentration of PDCB vapour. Two of the rabbits survived 62 exposures and recovered completely.\(^3\) These facts suggest toxicity of PDCB to the CNS, although the result of microscopic examination of the CNS of exposed animals was not mentioned. Reversibility of symptoms seen in the experimental models reported by Hollingsworth \etal and our case may be partially attributed to rapid elimination of this substance.\(^4\) Acute cerebellar ataxia and brainstem encephalitis should be ruled out in our case. However, there was no preceding infection and no fever, speed of worsening was rather slow, and symptoms improved after cessation of exposure. Abnormalities of BAEPs have not been reported in acute cerebellar ataxia. Therefore long-term exposure of PDCB may be
toxic to the CNS and cause especially reversible brainstem damage.

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Negative hallucinations: an ictal phenomenon of partial complex seizures

Sir: Negative hallucinations can be defined as non-perceptions of a stimulus placed in the field of an intact sensory modality.1 Being rare, they have been reported only with psychiatric conditions such as hysteria or under hypnosis.2–4 In the following case, negative hallucinations occurred as an ictal phenomenon of partial complex seizures.

Eighteen months prior to his admission, a 76 year old male began experiencing brief lapses of awareness without loss of consciousness or unusual motor activity. cranial computed tomography (CT) elsewhere revealed a left occipital infarct and an electroencephalogram (EEG) exhibited paroxysmal bitemporal slowing with occasional left hemispheric spike discharges. Two days prior to admission, he experienced the sudden disappearance of his wife from his visual field. One day later, a waitress suddenly disappeared from his view. In both cases, the disappearing subject was in the centre of his visual field and nothing in the background was changed. All other elements of the scene were intact, undistorted, and normally illuminated. Even the chair in which the patient’s wife sat and the counter behind the waitress remained unchanged. Within 10–15 seconds, both subjects had reappeared as suddenly as they had disappeared. The patient denied awareness of anything else unusual during these episodes.

One day after admission to our hospital, the patient suddenly indicated that his examiner had just disappeared. His eyes and then head deviated to the left and he was unresponsive to questions for approximately 15 seconds. He then stated that he had just had an experience similar to the previous two, in which the person in front of him had briefly disappeared, while everything else in the room appeared to be normal. He was amnestic for events occurring between the disappearance of the examiner and his resumption of responsiveness to questions.

Past medical history, physical examination, and routine laboratory studies were unremarkable. Contrast enhanced CT revealed bilateral occipital low-density lesions surrounded by areas of luxury perfusion and mild ventriculomegaly. During an EEG, sudden spontaneous eye opening, left versive eye and head movement, and unresponsiveness occurred. The EEG revealed paroxysmal fast activity in the right temporal region which evolved into 3 Hz polyspike and wave discharges remaining focally prominent in the right hemisphere and lasting approximately one minute. Two similar episodes were subsequently recorded.

Comprehensive neuropsychological testing revealed mild auditory discrimination impairment and slight difficulty with attentiveness. There were no significant histrionic features. Goldmann visual field testing was normal. The patient experienced no further seizures or unusual psychosensory disturbances after achievement of therapeutic serum levels of carbamazepine.

This patient’s experiences may be distinguished from several other well known psychosensory phenomena. Unlike illusions, they involved something other than a mere distortion of the stimulus object5 and they differed from hallucinations, in the normal sense, because they did not involve perception in the absence of a stimulus.6 Rather, our patient perceived the absence of a stimulus which was present (that is, a negative hallucination).

Other similar but distinct psychosensory experiences are well known to occur with structural lesions or seizure foci in brain regions close to those found in our patient. Anton’s syndrome involves the patient’s denial of total blindness and results from bilateral lesions in the calcarine cortex or parieto-occipital cortex.7 Balint’s syndrome involves failure to appreciate a whole scene, despite recognition of its parts, and occurs with bilateral lesions of the angular gyrus and adjacent occipital cortex.8 Prosopagnosia is a specific failure to recognize familiar faces and appears to result from bilateral lesions in the inferomedial occipital cortex.9 In addition, we have considered those reported cases in which visual field defects occurred solely as an ictal phenomenon.10 In our patient’s case, this would require bilateral ictal macular scotomas, since the subjects disappeared from the centre of his visual field. This would not be inconsistent with this man’s experiences since his seizures had a right unilateral onset.

We conclude that our patient experienced true negative hallucinations: failure to perceive a sensory stimulus despite intact functioning of the appropriate sensory modality. To our knowledge, this is the first case of such a phenomenon reported in association with neurologic disease.

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