toxic to the CNS and cause especially reversible brainstem damage.

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References

3 Hallowell M. Acute haemolytic anemia following the ingestion of paradichlorobenzene. Arch Dis Child 1959;34: 74–75.

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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. Being rare, they have been reported only with psychiatric conditions such as hysteria or under hypnosis. 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 man began experiencing brief episodes of unawareness without loss of consciousness or unusual motor activity. Cerebral 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 attention. 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 object and they differed from hallucinations, in the normal sense, because they did not involve perception in the absence of a stimulus. 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. 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. Prosopagnosia is a specific failure to recognize familiar faces and appears to result from bilateral lesions in the inferolateral occipital cortex. In addition, we have considered those reported cases in which visual field defects occurred solely as an ictal phenomenon.

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, and such a phenomenon reported in association with neurologic disease.

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References

Herpes simplex type II encephalitis in a non-immunocompromised adult

Sir: At least five of the double stranded DNA herpes family of viruses are known to infect the human nervous system, (HSVII, EBV, CMV, VZV).1-3 The clinical and pathological involvement is relatively specific for each individual virus species, but is also significantly influenced by age and immune status of the host.

A 19 year old, previously normal, male presented with a 4 day history of generalised headache, fever, anorexia and vomiting. His parents reported that he had appeared slightly disorientated the day prior to admission. Further questioning revealed no history of head trauma, epilepsy, travel, drug intake or recent sexual contact.

On initial examination he was fully conscious but appeared agitated with slowed mentation. He was pyrexial (rectal temperature 39°C). There was no meningism nor signs of increased intracranial pressure. Further systemic examination revealed nil of note. Initial laboratory investigations including full blood count, erythrocyte sedimentation rate, serum urea and electrolytes, serum glucose and urine microscopy were normal. Cerebrospinal fluid obtained on admission revealed an opening pressure of 20 cm H2O, raised protein (131 mg/100 ml) and normal glucose level. There were 42 neutrophils and 244 lymphocytes per mm3. Gram stain and bacteriological culture were negative.

Over the next 12 hours he deteriorated markedly, becoming stuporose with neck stiffness and myoclonic jerking of his left arm as well as twitching of the right side of his face. At this stage computed tomography of the brain showed an area of low density in the right temporal lobe. An electroencephalogram showed diffuse background slowing with high amplitude sharp waves in the left fronto-temporal area. A tentative diagnosis of herpes encephalitis was made and therapy with intravenous acyclovir was commenced. There was further deterioration in the following 24 hours and a right temporal lobe biopsy was performed.

Histological examination and electron microscopy revealed inclusion bodies typical of Herpes simplex encephalitis. However, no virus could be isolated in tissue culture or demonstrated by immunofluorescence. Thereafter followed a prolonged course of gradual recovery, complicated by episodes of pyrexia, bradycardia, hypothermia, manic psychosis and an episode of malignant neuroleptic syndrome (produced by haloperidol).

He has subsequently made a remarkable recovery. He is presently on anti-epileptic therapy and has had no further convulsions. Except for an apparent long-term memory deficit there is no clinical neurological deficit. Relevant serological investigations included those for Herpes simplex virus. Antibodies (Ab) for Herpes simplex virus were determined in blood and cerebrospinal fluid (CSF) by the indirect immunofluorescence (IFA) method.4 On admission the HSVII IgM (IFA) titre was 1:20 and 2 weeks later 1:640, that for HSVII IgG 1:40 on admission and 1:160 2 weeks later. The HSVI IgM (IFA) titre remained negative. Complement fixation revealed a 1:16 titre for HSVI on admission as well as 2 weeks later.

The use of serological virus identification and antigenic typing techniques has enabled the delineation of specific illness profiles associated with each herpes virus type.4-6 Several methods are available for detecting Ab to HSV in body fluids. The complement fixation test (CFT), formerly in wide use, has largely been supplanted by more sensitive and convenient techniques. Disadvantages of the CFT are that it cannot differentiate Ab to HSV-I from HSV-II, rises in Ab titre may be obtained in recurrent as well as acute infections, and IgM cannot be determined. More sensitive and specific techniques include the indirect haemagglutination test,7 immunoassay,8 neutralisation test9 and IFA test.8 In this patient the clear seroconversion around the 14th day of illness, the presence of the rising titre of specific IgM and the temporal relationship of these with the patient's illness are highly suggestive of an acute HSVII encephalitis.

Of the group of herpes virusiae, the Herpes simplex viruses are probably the most common cause of severe nervous system infection. However, the two types produce fairly distinct clinico-pathological disease entities. According to Adams,10 three central nervous system pathological entities can be distinguished. These are acute necrotising encephalitis, encephalitis associated with widespread virus dissemination, and aseptic meningitis.

Acute necrotising encephalitis occurs at any age except the neonatal period and very early childhood. The cause is HSVII,11 which is believed to gain access to the brain either by the olfactory nerve or from a reactivated latent viral focus in sensory ganglia.11-12 Characteristically the infection produces a widespread but asymmetrical necrotising encephalitis mainly involving the temporal lobes.11-13 Clinical features include herpes labialis, fever, headache, photophobia, meningism, obtundation, seizure, focal neurological deficit, coma and death.12,13 Without early recognition and treatment the prognosis is grave. Diagnostic modalities include serological investigation, cerebrospinal fluid analysis, electroencephalography and computed tomography of the brain.5,12,14,15 Brain biopsy remains the most reliable investigation for early diagnosis.16 High degrees of sensitivity and specificity have been reported,16 especially when involved brain sites can be located by EEG and or computed tomography.16,17 The current drug of choice is acyclovir, which has been reported to lower dramatically both morbidity and mortality rates.18,19

Encephalitis associated with widespread viral dissemination is seen typically in neonates and is usually caused by HSVII.1 The commonest presumed source of infection is from an infected genital tract, the virus being transmitted to the neonate at birth.1 Postnatal transmission from another infected infant or personnel may occur. Typically multiple organ systems are involved, including the central nervous system, eye, skin, reticuloendothelial and respiratory systems.1 Both diffuse and focal brain involvement have been described. The prognosis of this form of infection is also extremely poor.

Aseptic meningitis due to HSV usually occurs in young adults and is due to venereally acquired HSV Type II. The prognosis is excellent, almost all cases recovering spontaneously.