actor, Leopoldo Frégiol, who was famous for his ability to impersonate people.

In their original description of this delusion, Courbon and Fail noted that their patient experienced other delusions whose contents were mainly erotic. The association of Frégiol delusion and erotomania has only been occasionally reported since, though in some Frégiol cases erotomania forms an obvious background possibility.1 A review of misidentification syndromes and sexuality by Barton and Barton noted that erotomania has been found to be a feature in various forms of delusional misidentification, and included an additional description of a case of a Frégiol-like delusion and erotomania, in which a female patient claimed that another patient was her ex-boyfriend, going under an assumed name, and that they were in love.4 Although this patient had other delusions, the Frégiol and de Cérambault delusions dominated the clinical picture, and were strongly held and persistent.

The patient was a 35-year-old, unemployed woman who lived on her own. She had a psychiatric history from the age of 16, and was diagnosed as suffering from chronic paranoid schizophrenia. She stopped medication 6 weeks before admission.

She was agitated and verbally hostile, and reported auditory hallucinations of famous actors who she said were her friends. She claimed to be telepathic, saying her actor friends put their thoughts into her head, and that her thoughts were broadcast to them. She showed grandiose delusions, believing she could arrange to stop all television and radio communications by telling her actor friends to go on strike using their 'telepathic' abilities. The patient believed that she was the girlfriend of Erik Estrada (an American actor and pin-up), with whom she communicted almost daily via telepathy. She also believed that Erik Estrada visited her home city regularly, disguised as acquaintances or her current boyfriend. She stated that she knew her actual boyfriend was Erik Estrada in disguise due to the absence of a previous scar on his face. She was convinced that Erik Estrada was in love with her and planned to marry her one day.

Past medical history revealed childhood epilepsy until the age of 9 years, phenobarbital therapy, and a recent history of childhood migraines. Although the patient was still in frequent contact with her ex-boyfriend, she was consumed by thoughts of marrying her new love, who she described as 'Erik' and that he continues to visit her regularly, albeit in disguise.

Her pattern of impairment on face processing tests was comparable to that found in another patient investigated, in which the Frégiol delusion arose in the context of cerebral infarction of the right hemisphere.17 That case did not show our patient's flagrant erotomania, but there was a definite possibility of an erotomanic element in her delusion. She thought that she was being pursued by her cousin and a female accomplice, both of whom adopted different disguises. It was later found that some years previously the patient had a long love affair with this cousin (lasting over 20 years, and leading to the birth of her only child).

Data for this previous case are also presented in the table, for comparison with the patient we describe here. Both patients were impaired at recognising familiar faces, matching disguised faces, and showed much poorer recognition memory for faces than controls. Although an EEG suggested bilateral abnormalities for our present patient, these face processing impairments point toward involvement of the right cerebral hemisphere, which has been noted as a feature in other cases of erotomania and delusional misidentification.23

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Bilateral crossed optic ataxia in a corpus callosum lesion

Optic ataxia is a disorder of visually-guided hand movements, usually resulting from a lesion affecting the posterior parietal cortex.1 We recently observed a patient with bilateral crossed optic ataxia and a disconnection syndrome. MRI showed a large corpus callosum lesion without any other visible lesion.

A 37-year-old right-handed man was admitted to intensive care with an acute respiratory distress syndrome due to severe lung disease. The patient had a long history of alcoholism. One month later, the patient's condition had improved and he was alert and cooperative. On neurological examination, there were no sensory loss or motor weakness, no cranial nerve abnormalities, cerebellar syndrome or gait disturbance. Visual acuity was 10/10, bilaterally. The visual fields (Goldmann perimetry) and visual evoked responses were normal. Eye movements were recorded using electro-oculography. Horizontal smooth pursuit gain was normal, and horizontal visually-guided saccades had normal accuracy and latency. Higher cortical function testing showed a slight impairment of recent memory, but normal verbal comprehension, speech and reading. However, left ideomotor apraxia and left hand agraphia were present, suggesting the existence of a disconnection syndrome.

There was also left asteagnosia: the patient correctly named only 2 objects out of 12 when they were placed in his left hand, but made no errors when they were
Figure Sagittal MRI of the brain, T1 weighted image (TR 600, TE 15, 1-0 Tesla) shows an extensive lesion (hypointensal) involving the whole corpus callosum. The anterior commissure was spared (arrow).

placed in his right hand. Similarly, left pseudo-hemianopia was present: the patient correctly named 2 objects out of 15 presented tachistoscopically in his left visual field, but 15 out of 15 when they were presented in his right visual field. The dichotic listening test also revealed total extinction on the left side. The patient obviously experienced difficulty in reaching objects located in the hemispace contralateral to his hand. While the patient was looking at a central visual fixation point, a lateral target (pencil) was presented in his peripheral visual field, either right or left. The patient was instructed to reach out and take this lateral target (right or left) with one hand (right or left), without moving his eyes from the central fixation point. Twenty trials were made for each of the four combinations. When using the hand ipsilateral to the lateral target, the patient easily and accurately reached this target: 20/20 correct responses were obtained for the right hand in the right hemispace, and 19/20 for the left hand in the left hemispace. However, when he had to reach a target located contralaterally to the hand used, he experienced marked difficulty: the direction of the arm movement was grossly inaccurate and the target was missed. Only 5/20 correct responses were obtained with the right hand in the left hemispace, and 9/20 with the left hand in the right hemispace. MRI showed a lesion involving the whole extent of the corpus callosum (figure). The anterior commissure and the cerebellar peduncles were spared, and there were no visible lesions in the region of the floor of the third ventricle. There were no abnormalities in the cerebral cortex, in particular in the parietal lobes or the corona radiata.

The presence of a disconnection syndrome, associated with a large lesion affecting the corpus callosum was, in the context of severe alcoholism, compatible with Marchiafava-Bignami disease. The most interesting finding was the impairment of visually-guided reaching movements, in the absence of motor weakness and somatosensory or visual field defects. This bilateral crossed visuo-motor impairment was consistent with bilateral crossed optic ataxia, that is, a specific impairment of visuo-motor coordination.1 Left ideomotor apraxia is a different entity from bilateral crossed optic ataxia as: 1) They are different types of movements (imitative gestures vs visually guided reaching movements); 2) The movement disorders are not observed in the same movement fields (the whole movement field vs the contralateral movement field), and 3) With the same arm (left arm vs both arms). The crucial role played by the posterior parietal cortex in this function has been established,14 and 5 number of cases of optic ataxia following posterior parietal lesions have been published. However, optic ataxia may be observed in the absence of a parietal lesion. The posterior parietal cortex is connected to the motor areas of the frontal lobes, ipsilaterally through parieto-frontal association fibres and contralaterally through the corpus callosum.5 Thus a lesion affecting one of these fascicles could theoretically result in optic ataxia. A lesion affecting the intrahemispheric association fibres could result in ipsilateral optic ataxia, but as such a lesion probably also partly involves the primary motor cortex region, the ensuing motor deficit interferes with the demonstration of optic ataxia. A corpus callosum lesion could result in bilateral crossed optic ataxia. This syndrome was reported in one case of a split brain.6 To our knowledge, no other case of bilateral crossed optic ataxia following a lesion restricted to the corpus callosum has been reported. Our case confirms that bilateral crossed optic ataxia should be included in the classic signs of the disconnection syndrome.

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Figure 1 A train of responses recorded from the adductor digiti minimi with electrical stimulation of various frequency in a healthy subject. Smooth increment in amplitude is noted at higher frequency stimulation. As the duration became concomitantly shorter, the negative area remained relatively unchanged.
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