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

In their original description of this delusion, Courbon and Fail¹ noted that their patient experienced other delusions whose content was mainly erotic. The association of Frégoli delusion and erotomania has only been occasionally reported since, though in some Frégoli cases erotomania forms an obvious background possibility.^{2,3} A review of misidentification syndromes and sexuality by Barton and Barton² 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 Frégoli-like delusion and erotomania, in which a female patient claimed that another patient was an ex-boyfriend, going under an assumed name, and that they were still deeply in love.

We describe a further case for whom the Frégoli delusion arose in the context of the form of erotomania known as de Clérambault's syndrome, in which patients suddenly arrive at the delusional belief that someone (usually of higher social standing) is in love with them.⁴ Although this patient had other delusions, the Frégoli and de Clérambault delusions dominated the clinical picture, and were strongly held and persistent.

The patient was a 35 year old, divorced, 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 her "telepathic powers".

The patient believed that she was the girlfriend of Erik Estrada (an American actor and pin-up), with whom she communicated across the Atlantic 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, phenobarbitone being stopped at the age of 11 years. There was no family history of mental disorder. Routine haematological, biochemical and serological examinations were normal. Physical examination revealed no abnormality. An EEG showed a moderate excess of mixed irregular and rhythmic slow activity at 2–6 Hz and 10–30 µV in the central and post-central regions. She refused neuroimaging.

Neuropsychological tests of face processing were also carried out. Details of the tests used are given in Young, *et al.*,⁵ which includes data from 10 male controls aged 25–35 years. Results are summarised in the table. The patient was able to recognise photographs of emotional expressions (happy, angry, sad, etc) without significant difficulty. She was impaired at recognising photographs of familiar faces, but showed no tendency to misidentify unfamiliar faces as familiar (20/20 correct rejections of unfamiliar faces). In this face recognition test, she did not claim that any of the photographs showed Erik Estrada, in disguise or otherwise. She performed at the borderline of the impaired range on the Benton Test (which requires matching of unfamiliar faces) and was very poor at matching unfamiliar faces when they were masked by various disguises. On the Warrington Recognition Memory Test, she showed normal recognition memory for words but severely impaired recognition memory for faces.

The patient was started on a fluphenazine depot and her mental state improved considerably. Twelve months later, however, she still believes she is to marry "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 for another case we investigated, in which the Frégoli delusion arose in the context of cerebral infarction of the right hemisphere.^{3,5} That case did not show our patient's flagrant erotomania, but there was a definite possibility of an erotomaniac 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 words. 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.^{2,3,5}

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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 caused by 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 agnosia were present, suggesting the existence of a disconnection syndrome.

There was also left astereognosis: 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

Table Performance of face processing tasks, and means and standard deviations for control subjects of comparable age. Data from another Frégoli patient,^{3,5} are presented for comparison (this patient was aged 68, so her performance has been compared to a different set of controls).

	Previous case	Present case	Controls Mean (SD)
FACIAL EXPRESSIONS			
Labelling:	20/24	20/24	22.00 (1.24)
IDENTIFICATION OF FAMILIAR FACES			
High familiarity faces			
Occupation:	15/20***	12/20***	17.58 (1.08)
Name:	8/20***	12/20**	16.17 (1.53)
Unfamiliar faces			
Correct rejections:	20/20	20/20	
UNFAMILIAR FACE MATCHING			
Benton test:	42/54	39/54 ^b	
"Disguise" task:	13/24***	16/24**	21.60 (1.90)
RECOGNITION MEMORY			
Warrington RMT: Faces:	36/50 ^{d1*}	33/50 ^{d2**}	43.90 (3.65)
Warrington RMT: Words:	47/50 ^{d1}	47/50 ^{d2}	45.71 (4.76)

[Asterisked scores are significantly impaired in comparison to the performance of controls:

* $z > 1.65$, $p < 0.05$; ** $z > 2.33$, $p < 0.01$; *** $z > 3.10$, $p < 0.001$. ^b=borderline of impaired range on test's norms. ^{d1 d2}=significant discrepancy between faces and words scores].

Figure Sagittal MRI of the brain, T1 weighted image (TR 600, TE 15, 1.0 Tesla) shows an extensive lesion (hyposignal) 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,^{1,2} and a 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.³ 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.⁴ 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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MATTERS ARISING

False facilitation to repetitive stimulation

We have read with interest a letter from Drs Pullicino and Beck on incremental response to repetitive nerve stimulation in Guillain-Barré syndrome (GBS).¹ They suggested that acetylcholine (ACh) release from motor nerve terminals might be impaired in acute GBS. In motor neuron disease, abnormal decrement such as myasthenia gravis has now been well documented. However, the facilitation they showed seems to be a false positive result.

In neuromuscular block in Lambert-Eaton syndrome, compound muscle action potential (CMAP) to the first stimulus is of very low amplitude. At low frequency stimulation, a waning pattern similar to that seen in myasthenia gravis is a common finding.² At high frequency repetitive stimulation, marked facilitation more than 200 to 300% is thought to be confirmatory. The true facilitation results from an increase in the number of ACh quanta released, which give rise to a larger end-plate potential. Electromyographers, however, have paid attention to the false facilitation erroneously seen without true neuromuscular block.

Figure 1 is an example of false facilitation, recorded from a healthy normal subject aged 30. At 2 Hertz stimulation little change was noted. At higher frequency stimulation, successive increase in amplitude was remarkable. The rate of increment was highest at 50 Hertz stimulation, up to 180% of the initial amplitude. The critical finding is that duration of the negative phase became concomitantly shorter when the amplitude increased. As a result, the area of negative phase remained relatively unchanged, which is a typical finding for false positive CMAP changes, either increment or decrement. In true facilitation in

Figure 1 A train of responses recorded from the abductor 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.