# LETTERS TO THE EDITOR

## A case of music imperception

Defective perception of music due to an altered capacity to discriminate the elementary components of musical stimuli (rhythm, pitch, timbre, intensity and duration) produces an alteration in the aesthetic enjoyment of and the emotional involvement in music.[1] To our knowledge, there is no previous evidence of the existence of a condition in which the primary disruption is to the capacity to process the musical stimulus as a whole. We report a case of a young musician who, as a result of a right temporo-parietal lesion, presented with loss of the gestalt capacity to process music, with the consequent loss of aesthetic pleasure.

Our patient was a 24 year old male, who was an amateur musician and a skilful guitar player. He reported an early interest in music: during primary school he played keyboard instruments and during secondary school, the flute. He later started playing the guitar, attending private lessons for two years.

On 31 December 1987, after a muscular strain (jumping over a gate), he noticed he had difficulty in understanding "the nuances of words and inflection of sentences". The same day he realised that he had difficulties in understanding music: he could not perceive the structure of musical pieces clearly; the relationship between the accompaniment and the soloist was indiscernable, and aesthetic pleasure for the musical world had completely vanished. At the same time he complained of generalised headache, described as pressure, mostly on the right side.

On 5 January 1988, he came under our observation. He still complained of a receptive musical impairment, although less severe than at onset, while his previous prosodic difficulties and headache had disappeared. Neurological examination, tonal audiometry and standard neuropsychological examination were normal. In particular, he showed no language impairment; oral and written comprehension of complex passages was good. He was able to recognise simple and complex figures and recognise faces, both familiar and unknown, on comparison tasks. On a meaningful sound recognition test, he recognised animal, human and environmental sounds and voices, effortlessly. The patient was ambidextrous; there was no family history of left handedness.

Examination of musical abilities (recognition and production of physical features of musical sounds, plus identification and/or reproduction of rhythm, melody and harmony; vocal and instrumental performance, and listening to musical compositions) were normal. However, he complained of difficulties from the very beginning for the listening task. On hearing the pieces played on the piano, he complained, "my perception is changed... it's flat, it's no longer 3-dimensional; it's only on two planes... there's no emotion...". His difficulties increased as the presented compositions became more complex: "... this is even worse: I can distinguish the different instruments, but I can't perceive the whole... in jazz pieces, the relationships between the accompaniment and the soloist escape me."

An EEG showed slight right temporal abnormalities. A CT scan was scheduled, but the patient did not keep the appointment.

On 31 April 1988 he had a generalised tonic-clonic seizure: a CT scan (figure, left) and MRI (figure, right) showed the presence of a right temporo-parietal malacic area, involving the plica curva and supramarginal gyrus. This area was post-haemorrhagic, with peripheral haemosiderin deposits, and was surrounded by serpiginous hypointense images. A DSA showed an arterio-venous malformation (AVM), fed by right sylvian branches.

On 16 June 1990, he developed left hemianopia and headache due to a haemorrhage involving the right temporo-parietal region. A month later, the AVM was surgically removed. Since that time, the patient has experienced normal health.

The correct characterisation of this patient's disturbances poses considerable difficulties. It is neither auditory agnosia nor word deafness, given his correct performance on neuropsychological tests. The complexity of his deficits, on the other hand, rule out their being due to "paracousia", that is, simple distortion in the perception of sound.

This impairment could instead be classified as a type of receptive amusia, which can manifest itself not only as the incapacity to discriminate between melodic patterns, timbre and pitch, but also as qualitative alterations of the acoustic experience, including an emotional involvement in the music.[1] The patient may have lost the ability to convert musical perception into something emotionally or intellectually meaningful (the third fundamental ability of musical function[2]). Disturbances of this type are, unfortunately, difficult to view objectively because of their highly subjective nature. These deficits are perhaps undervalued and overlooked, and as a result, there are only two cases in which the loss of aesthetic enjoyment in listening to music[3] or problems in "conceiving of a whole piece" and "conjuring up the appropriate atmosphere for composition"[4] are mentioned in the literature.

Even though much of our case is based on the patient's subjective reports, it seems convincing for the selectivity of the disturbance (altered perception of the whole and of the emotional component of music, without compromise to analytical perception) and for the strict focal nature of the lesion (AVM involving the right plica curva and the supramarginal gyrus). This supports the hypothesis that the right hemisphere, or at least some of its supramodal areas, is better than the left hemisphere[5] at performing the process of appreciation of the entire sound.

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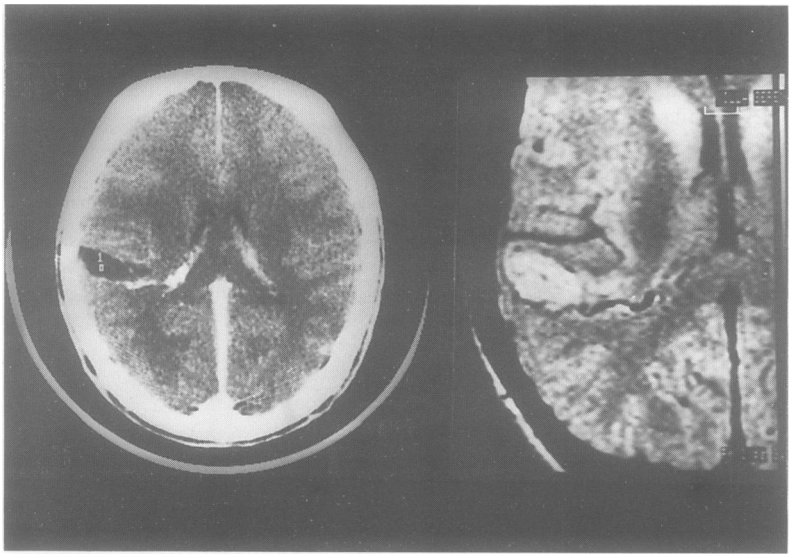

*Figure Left: CT scan (2 May 1988) showing a ribbon-shaped malacic area in the right temporo-parietal region, the posterior part of which has absorbed contrast agent Right: MRI (5 May 1988) showing the malacic area involving the plica curva and the supramarginal gyrus.*

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## Frégoli delusion and erotomania

The Frégoli delusion involves the belief that a familiar person disguises himself or herself as others. It was named after an Italian

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

In their original description of this delusion, Courbon and Fail[1] 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 Barton[2] 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 10–30uV 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 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 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]

*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).*

| | | | Controls |
|---|---|---|---|
| | *Previous case* | *Present case* | *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: * $z > 1{\cdot}65$, $p < 0{\cdot}05$; ** $z > 2{\cdot}33$, $p < 0{\cdot}01$; *** $z > 3{\cdot}10$, $p < 0{\cdot}001$. [b]=borderline of impaired range on test's norms. [d1 d2]=significant discrepancy between faces and words scores].

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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 agraphia 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