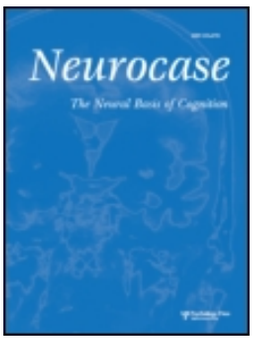

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"Blue is music to my ears": Multimodal synesthesias after a thalamic stroke

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Acquired synesthesias have been reported in association with deafferentation of the visual system, temporal lobe seizures, and the use of psychedelics. Based on our review of the literature, the appearance of synesthesias after a thalamic stroke has been reported only once. We present the case of a 45-year-old hypertensive male who, 9 months after a hemorrhagic stroke involving the left lateral posterior nucleus of the thalamus developed persistent sound–tactile, sound–color, and grapheme–gustatory synesthesias. Moreover, the patient noted that even thinking about a sensory stimulus could trigger the experience of another sensory modality, a conceptual type of synesthesia.

Keywords: Synesthesias; Multimodal; Thalamic; Stroke; Posterior lateral thalamus.

The neurocognitive perceptual experience in which a sensory stimulus triggers responses in brain systems that are not being stimulated is defined as "synesthesia". With more than 100 years of research in the scientific literature (Galton, 1883) and even longer in art, literature and music (Brougher, Strick, Wiseman, & Zilczer, 2005) it has only been recently, with the input of neuroimaging (Nunn et al., 2002) and detailed clinical descriptions (Ward & Simner, 2007) that the brain mechanisms involved in synesthesias have become clearer (Ramchandran & Hubbard, 2001). The prevalence of synesthesia is estimated at 0.05% in the general population (Baron-Cohen, Burt, Smitt-Laittan, Harrison, & Bolton, 1996) but there have been suggestions that it may be more common among painters, musicians, and writers (Cytowic, 2002). Synesthesias are currently classified as developmental and acquired.

While developmental synesthesias do not constitute a neurological disorder but rather a different way of experiencing one's environment (Afra, Funke, &Matsuo, 2009), they have specific characteristics. They appear after one year of age (Maurer, 1997), are lifelong, idiosyncratic, and synesthetes of the same family may even experience different forms. They are also automatic, involuntary, and consistent during the lifespan (Hubbard, Annan, Ramachandran, & Boyton, 2005). On the contrary, acquired synesthesias are usually caused by neuropathological, neurophysiological, or neurochemical insults. Consequently it has been reported in association with deafferentation of the visual system (Bender, 1977), partial complex seizures of temporal lobe origin (Jacome & Gumnit, 1979), ventrolateral thalamic stroke (Ro et al., 2007), and psychedelic drugs such as LSD (Hollister, 1968), mescaline (Simpson & Mckellar, 1955), ayahuasca (Shanon, 2002), sativa divinorum (Babu, McCurdy, & Boyer, 2008), and hashish (Marks, 1975). We present the case of a 45-year-old male who, 9 months after having a left posterior lateral thalamic hemorrhagic stroke, developed persistent multimodal acquired synesthesias of the sound–color, sound–tactile, and visual–gustatory type. In addition he developed a conceptual type

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of synesthesia which was elicited by just thinking about a concept, rather than seeing, hearing, or touching any kind of stimulus (Dixon, Smilek, Cuhady, & Merikle, 2000).

CASE REPORT

A 45-year-old, bilingual, right-handed male Registered Nurse with a past history of hypertension was referred to our Memory Clinic by his primary neurologist for investigation of subjective memory impairment following a hemorrhagic stroke that he experienced 14 months previously. At that time, he was admitted to the emergency department (ER) with a hypertensive crisis, likely triggered or aggravated by the use of cocaine the night before. He was conscious on and off for 4 days. When fully conscious, he had a right hemiparesis, right hemisensory deficit, right hemianopsia, slurred speech with normal comprehension, and a transient right alien limb phenomenon described as "scared of my own limb". Brain CT scan demonstrated a left thalamic hematoma with intraventricular blood extension (Figure 1). An angiogram CT scan, thoracic CT scan, and echocardiogram were all normal. He lost the sense of smell. He had not experienced a head injury. His subjective memory impairment could not be confirmed by our neuropsychological tests performed 14 months after the acute thalamic hemorrhage. Six months after the stroke he started to develop frequent déjà vu episodes and petite Madeleine phenomenon triggered in his case by visual stimuli. For example, the sight of a ginger cookie opened a full childhood scene in his grandmother's kitchen with visual, auditory and olfactory sensations. He could have been 4 or 5 years of age, because he saw himself unable to reach the kitchen counter. He developed sensitivity to some auditory stimuli; for example, the sound of an ambulance's siren triggered a pleasant warm feeling all over his body. He was always able to control these sensations, however, and they were not associated with changes in consciousness. Furthermore, an EEG done at the time was normal. The anosmia recovered 6 months later, first in his left nostril and then in the other.

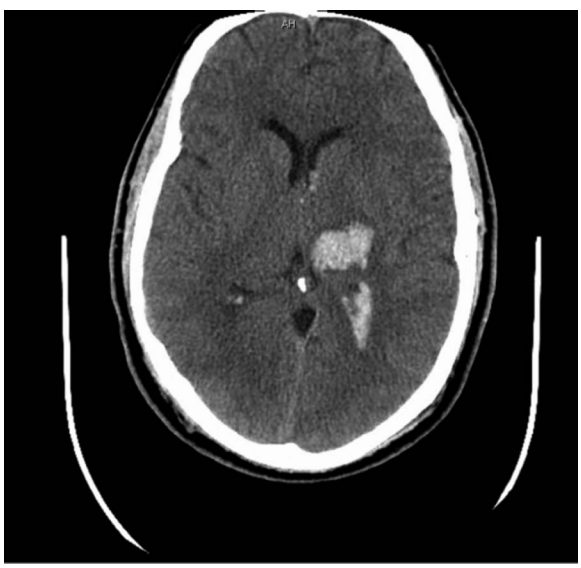

Figure 1. Left thalamic hematoma with intra ventricular blood extension.

Nine months after the hemorrhagic stroke he started to have sound–color, sound–touch, and grapheme–taste synesthesias. Listening to high tone sounds, such as Hindi, Inuit, or Chinese music, would trigger an extracorporeal sensation described as "riding the music", "surfing the music", or "flying on top of the music". He noted at the same time the appearance of light blue colors occupying the left halves of both visual fields. When reading a shopping list he was able to smell and taste the foods. He would read advertisements for some food items such as chicken and he would feel the chicken "did not smell right" based on the color of the letters. Black and white letters were right. Blue letters were always wrong. In his own words, when he was answering the questions of the Synesthesia Battery, "the word chicken printed in black ink is just fine: I can buy or eat the chicken. The exact same words printed with blue ink makes me nauseated and I won't purchase nor order chicken from the menu just because of the color of the printed word". Moreover, even thinking about the shopping list could elicit the taste of the foods. After 3 years of close follow-up he continues to experience the synesthesias in a remarkably consistent way: the same modalities are triggering the same responses. Furthermore, his scores in the chord color picker test and in the instrument color picker test of the Synesthesia Battery (Eagleman, Kagan, Nelson, Segaram, & Sarma, 2007) are in the synesthetic range of minus 1: 0.62 and 0.83, respectively. His score of 1.66 on the piano scale color picker test of the same battery leaves him closer to the synesthete rather than the score of 2.0, typical of non-synesthetes who are using memory or free association to answer the test. He could voluntarily suppress all these synesthesias;

they are not experienced when he is cold or when lyrics are in French and/or English. Currently he is walking with a right hemiparetic spastic gait; he has right hemisensory deficits for touch, pinprick, joint position, and vibration sense, which are more marked on his face and arm, and normal sensations in his right lower limb. Visual fields are full. He can detect odors but can correctly identify only 50% of them. Taste is normal, and the sensation inside his mouth for touch is normal. Touching his right upper limb triggers a very unpleasant sensation, difficult for him to describe, but which makes him nauseated or "sick". He did not experience extinction (inability to detect and report a contralesional event when simultaneously presented with an ipsilesional one) and/or allesthesia (the experience where unilateral tactile stimulus delivered to the impaired side is felt in a corresponding area on the intact side.) He is starting to write and type using his left hand and is improving his computer skills. Scores on the Mini-mental state examination (Folstein, Folstein, & McHugh, 1975) and the Montreal Cognitive Assessment (Nasreddine et al., 2005) were both 30/30. On the Behavioural Neurology Assessment (BNA: Darvesh, Leach, Black, Kaplan, & Freedman, 2005), a standardized and validated test that measures different forms of attention, memory, naming, visuospatial, and executive functions, he scored 114/114. A SPECT Scan was normal, and his recent head MRI, 24 months after the stroke, revealed the left thalamic stroke with a lesion involving the posterior part of the lateral thalamus, including pulvinar, lateral posterior, and ventral posterior lateral nuclei (Figure 2).

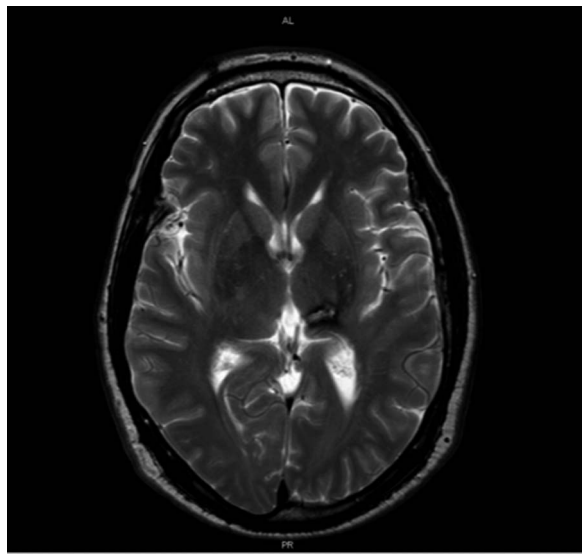

Figure 2. Left thalamic stroke with a lesion involving the posterior part of the lateral thalamus, including pulvinar, lateral posterior, and ventral posterior lateral nucleus.

DISCUSSION

The thalamus, with its 14 different myeloarchitectonic nuclei and some more nucleus subdivisions, constitutes the central relay station for the brain. Accurate clinic pathological syndromes are emerging from studies of strokes affecting the thalamus in humans (Bogousslavsky, Regli, & Uske, 1988) and from studies after tract tracing investigations in non-human primates (Schmahmann & Pandya, 1990). As a consequence, the communication between the thalamus and the cerebral and cerebellar cortex is so rich that we should no longer consider the operations of either structure separately from the other (Schmahmann & Pandya, 2008). These lesions are mimicking many cortical deficits that were previously cited as the sole site of cognition and behavior. The appearance of synesthesia – the confusion of different sensory modalities – occurring some time after an acute thalamic hemorrhagic stroke has been reported to our knowledge only once (Beauchamp & Ro, 2008). In this case, a 40-year-old hypertensive patient experienced auditory tactile synesthesia 18 months after a small ischemic stroke in the right ventrolateral nucleus of the thalamus. The authors suggested that altered connections from the ventrolateral nucleus of the thalamus to the cerebral cortex could be the cause for these sensory changes. In our hypertensive patient, we describe the occurrence of different types of synesthesias 9 months after a left hemorrhagic thalamic stroke, when the initial clinical symptoms of hemiparesis, hemisensory deficit, and hemivisual, olfactory and taste deficits were in frank recovery. The lesion likely responsible for the synesthesia is located in the left posterior part of the lateral thalamus, including the pulvinar lateral posterior, posterior nuclei, and ventral posterior lateral nuclei of the thalamus. All these are relay nuclei for the posterior parietal cortex and also multimodal integrative areas from the posterior insula to the orbito frontal cortex and even areas 18 and 19 of Brodman responsible for visual inputs (Schmahmann & Pandya, 2008).

The patient's complex multimodal perceptual sound–color, grapheme–gustatory, and sound–tactile synesthesias and even conceptual synesthesia

(perception in a modality by thinking about a particular concept in another modality), suggest that aberrant activation of anomalous sensory regions in response to stimulation (Baron-Cohen, Harrison, Goldstein, & Wyke, 1993) and anomalous cortical connections (Paulesu et al., 1995) may underlie the synesthesias in our patient. Neural plasticity, a well-known mechanism of restoration of function after cerebral insult can also be responsible for the abnormal thalamic–cortical connections and the late development of perceptual and conceptual multimodal synesthesias. The fact that our patient can suppress these post-stoke acquired synesthesias is a marked difference from developmental synesthesias which are involuntary. We are closely following our patient with further functional neuroimaging studies.

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