Left hemianomia of musical symbols caused by callosal infarction

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We are indebted to the patient’s wife for successfully capturing the wasps and thereby enabling their identification.

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Musical scores consist of two components, musical symbols and pitch notations. Musical symbols are graphic depictions that do not denote pitch. Examples include time symbols (for example, “4/4”, “.cres.”) and dynamic marks (“f”, “piano”) as indicated by roman letters. Pitch may be defined as the quality of a sound that fixes its position on a scale, indicated by “notes” written on, between, above, or below the five lines comprising the music staff. Case studies suggest that the left hemisphere is dominant in reading pitch notations, but opinions are divided on the left hemisphere’s superiority for naming musical symbols. In order to investigate this problem, we assessed hemispheric function in a patient with a calllosal lesion.

In November 1997, a 69 year old, right handed businessman who suffered from hypertension and diabetes mellitus suddenly developed impairment of the movement of his left hand. When opening a desk drawer with his right hand, his left hand involuntarily caught in the drawer as the result of a cerebrovascular event. He was an amateur violinist and had been active in concerts as a soloist, or as a member in an ensemble. For example, at the opening ceremony of the concert hall of our city, he performed Beethoven’s Romance in F major accompanied by the piano. He had been a member of a semiprofessional orchestra for 30 years. He met criteria for Gríson’s sixth level of musical culture.4 After this cerebrovascular event, he was unable to play the violin as well as before. Even with familiar musical pieces, his left fingers could not move with their previous accuracy. Left fingers require finer movements than the right, as they press the violin strings. He could read musical scores, and movement of the bow with his right hand was unimpaired. Brain magnetic resonance imaging (MRI) showed an infarct of his corpus callosum (fig 1) which affected the whole of the body and the anterior half of the splenium.

In April 2001, we undertook neurological and neuropsychological examinations. For the statistical analysis, we used Fisher’s exact test. A score of 24 points on a revision of Annett’s hand preference questionnaire4 confirmed that he was right handed. He was fully conscious and attained a normal score on the Mini-Mental State Examination (28/30) and Raven Coloured Progressive Matrices (27/36). He clearly remembered daily and social events. A Japanese version of the Rivermead Behavioural Memory Test yielded a normal score; a standard profile score of 18 (mean (SD), 19.73 (2.93)), and a screening score of 9 (mean (SD), 9.15 (1.78)). He had no weakness in any of his limbs, and no sensory disturbances, aphasia, dyscalculia, or visual discrimination difficulties were observed.

Examination of calllosal functions revealed left unilateral ideomotor apraxia. To investigate somatotactic transfer between cerebral hemispheres, the examiner touched a point on the patient’s hand with a pen while the patient’s eyes were closed, and then asked him to touch the corresponding place on the ipsilateral and contralateral hand using his thumb. When the examiner touched the patient’s left hand, the patient correctly identified 16/16 on the left hand and 6/16 on the right hand (p = 0.0002); when his right hand was touched, he correctly identified 4/16 on the left hand and 16/16 on the right hand (p = 0.00002). Thus our patient

Figure 1 Brain magnetic resonance imaging showed an infarct of the corpus callosum affecting the whole of the body and the anterior half of the splenium.
showed a somesthetic transfer defect. A dichotic listening test revealed auditory suppression (left 14/120, right 68/120; p = 0.01). tone audiometry was within normal limits and showed no difference in threshold between right and left ears. Using a tachistoscope, visual stimuli were presented for 70 ms in each visual hemifield, beside a yellow spot 3° in visual angle. The presence of left hemianopia for musical stimuli was identified (left 16/30, right 25/30, p = 0.025). Some graphically simple kanji are learned by the end of the first grade of elementary school, and many of them are pictographic (for example, "枠") for the mouth). Recently, such Japanese kanji revealed no significant differences between left and right (left 20/30, right 23/30, p = 0.57). The following callosal disconnection syndromes were absent: left visual anosia (left 10/10, right 10/10, p = 1.0); left agraphia of kanji (left 44/46, right 46/46, p = 0.49) and kana (left 43/46, right 46/46, p = 0.24); left tactile anosia (left 20/20, right 20/20, p = 1.0); and right constructional apraxia, which was examined using the block design test of WAIS-R (Wechsler Adult Intelligence Scale–Revised) (left 8/10, right 9/10, p = 1.0).

At the time of the tachistoscope, we also assessed hemicerebral superiority for the naming of musical symbols. Thirty musical symbols (G, F, and C clefs, up and down bow, accent marks, repeat marks, 16th-, 8th-, quarter-, half, whole notes and rest, a couple of eight notes, sharp, double sharp, flat, double flat, natural, 7, segno, tenuto, flageolet) were randomly ordered and briefly presented in each visual hemifield. Time and dynamic symbols were not included because they were composed of actual letters, the reading of which involves the left hemisphere. The patient showed left hemianopia for musical symbols (three times examined: left 22, 20, 20/30; right 28, 27, 28/30, total left 62/90, right 83/90, p = 0.0001). He mistook up bow, accent, 16th-note and rest, 8th-note and rest, sharp, double sharp, natural, segno, and flageolet in the left visual hemifield, and, in the right, flageolet, double flat, and tenuto. It was possible that the patient could not represent verbal answers even if his right hemisphere was able to understand the meaning of those musical symbols, because of the disconnection between the two hemispheres. In order to clarify this, a way of answering that did not require verbal processing was recommended. We undertook the same hemispheric superiority test for naming of musical symbols described above. Only 13 symbols were chosen this time (up and down bow, 16th-, 8th-, quarter-, and whole notes and rest, sharp, flat), because the other symbols are hard to express in a non-verbal way. Each symbol was presented twice (26 trials in all). The patient was asked to answer by pointing to a symbolic representation of the musical symbol with the hand that was ipsilateral to the represented visual hemifield. These representations consisted of figures which did not contain any words. For example, for the up and down bow, an upward and a downward arrow was used. For the quarter tone or rest, four columns with Japanese kanji were characterised each column having one to four painted rooms. On this test, the patient showed no significant differences between the two visual hemifields (left 21/26 and 22/26, right 24/26 and 24/26, total left 45/52, right 48/52, p = 0.23).

The results of our examination showed left hemisphere dominance in naming musical symbols. This result is in agreement with reports showing dominance of the left hemisphere in musicians when dealing with musical stimuli. Owing to the callosal lesion, the right hemisphere had lost access to the contralateral speech control centres, so reading musical symbols aloud was impaired. It is widely accepted that visual information is delivered through the splenium of corpus callosum. The only hemisphere our patient affected the anterior half of the splenium of the corpse callosum, suggesting that information contained in musical symbols is mediated there. Our patient also showed left alexia of kana, but not of kanji. The kanji used in our experiment convey pictographic meaning, whereas kana are purely symbolic. Musical scores, except for time and dynamic symbols, are also symbolic. It seems reasonable to conclude that similar neuro-pathological mechanisms are at work in the patient’s alexia of both kana and musical symbols.

References


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Leucocytoclastic vasculitic neuropathy diagnosed by biopsy of normal appearing skin

Leucocytoclastic vasculitis (LCV) is a clinicopathological entity that preferentially involves capillaries or small vessels rather than the medium sized or large arteries typical of polyarteritis nodosa. Its histopathological features are characterised by the presence of perivascular polymorphonuclear leucocytes with fragmented nuclear debris (leucocytocisis). It may be limited to skin lesions such as erythematous macules, purpuric papules, and haemorrhagic vesiculobul- lous lesions. Although LCV often involves skin lesions. Although LCV often involves skin lesions.