Face recognition: impairments in prosopagnosia

Environment, Air

Face Recognition: Impairments in Prosopagnosia Prosopagnosia, also called face blindness, is a neuropsychological condition that refers to impairment in the recognition of faces. Although prosopagnosic patients suffer from other types of recognition impairments (place recognition, car recognition, facial expression of emotion, est.), they experience face recognition problems above or over other types impairments. Prosopagnosia occurs without intellectual, sensory or cognitive impairments; in other words, people with prosopagnosia can still recognize people from non-facial cues.

They cannot recognize familiar people by their faces alone, and often use alternative routes to alleviate the effects of this impairment. These routes include using voice, gait, clothing, hairstyle, and other information rather than faces. Not surprisingly, prosopagnosia can be socially crippling. In absence of these non-facial cues failures of recognizing familiar faces reveal; in fact, patients are unable to recognize famous people, close friends, family members, and even their images in the mirror. Bodamer, a German neurologist, coined the term prosopagnosia in 1947 (Ellis & Florence, 1990).

The word prosopagnosia is a combination of Greek word for face (prosopon) and the medical term for recognition impairment (agnosia). Bodamer also stated prosopagnosia was related to brain injury (head trauma, stroke, and degenerative disease), which refers to acquired prosopagnosia. People with acquired prosopagnosia had normal face recognition ability and then that was impaired. In contrast, prosopagnosia can occur from birth with no medical record of brain damage, which refers to pure developmental or congenital prosopagnosia.

Prosopagnosia is classified as face recognition impairment and differentiated from other types of impairments that can compromise face recognition (Young, 1992). People with prosopagnosia can achieve recognition using non-facial cues. In contrast, Young (1992) described patient K. S. was poor at recognizing people from faces and names. Her problem was considered as impairments of retrieval semantic information about the identities of individuals. Prosopagnosia is not a unitary syndrome, and different patients may show different types of recognition impairments.

Bruce & Young (1986) claimed there were correspondences between these different types of recognition impairments and breakdown at different stages or levels of recognition. Neural correlates of prosopagnosia In the original view, prosopagnosia is acquired during adulthood or more rarely during childhood development, and it underlies brain injury. In general, prosopagnosia is thought to be associated with lesions of ventral occipitotemporal regions (Damasio et al. , 1982). It has been suggested that unilateral lesions of the right cerebral hemisphere is more associated with prosopagnosia than the left side (Damasio et al. 1982; Farah, 1990). An earlier opinion is that relatively small numbers of prosopagnosics have bilateral lesions that are from autopsy reports (Damasio et al. , 1982). However, later attitude is that lesion extent is probably underestimated, and more cases may have bilateral lesions because of limited resolution of brain images techniques (Farah, 1990). Although these two points of view are different, some cases show people with bilateral lesions were diagnosed as prosopagnosia. For example, patient FE (Bobes et al. , 2004) suffered a close head trauma that produced bilateral lesions.

He recovered but lost the ability to recognize familiar people by their faces alone. The extent of his brain lesions was identified by magnetic resonance imaging (MRI). The extensive bilateral damage in ventral occipitotemporal area was more severe on the right side. This case suggests that brain is an interconnected organ, and the right part of brain may play a more important role in prosopagnosia. Recently developmental prosopagnosia is given more attention. In developmental prosopagnosia selective face recognition impairment exists throughout life with little evidence of a structural brain deficit (Bentin et al. 1999). Like acquired prosopagnosia developmental prosopagnosia appears functional deficits on the basis of brain imaging studies. For instance, Hadjikhani and de Gelder (2002) noted that two regions in human extra striate are essential to normal face recognition. One of these is located in midfusiform gyrus known as the fusiform face area (FFA), the other in inferior occipital gyrus (IOG). In their study (Hadjikhani & de Gelder, 2002) there were 3 patients (one is a " pure" developmental prosopagnosic, while the other two suffered from close head injury in childhood) with severely impaired face recognition.

None of these patients showed structural abnormalities on the basis of brain scans. However, they showed functional deficits of FFA and IOG; that is, these two regions did not evince stronger responses to faces than objects like normal subjects. In additional, patients showed a partly normal pattern of activation during objects viewing, and they have no difficulties discriminating faces from objects. Thus, these finding suggests that their lack of normal FFA and IOG involves inability for face recognition but not for face detection. Prosopagnosia and a functional model

Prosopagnosia is not simply a mild agnosia; in fact, people with prosopagnosia lose the ability to recognize faces but relatively preserve the intact ability to recognize objects. In contrast, McCarthy and Warrington (as cited in Farah & Ratcliff, 1994) described a patient were unable to recognize pictures of common objects but performed normally with pictures of familiar faces. This double dissociation therefore suggests that recognition of faces and common objects is served by different mechanisms that are independent for each other.

Although brain cells of face recognition have not been located, some functional models could help us interpret face recognition and prosopagnosia. One of the most influential models was proposed by Bruce and Young (1986). In this model face recognition involves several steps, and three steps more relate to prosopagnosia. Firstly, structural encoding includes view-centred descriptions and expressionindependent descriptions. View-centred descriptions derive from visual input and provide information for expression analysis, facial speech analysis, and directed visual processing.

To recognize an individual by the face, view-centred descriptions have to translate into expressionindependent descriptions those in turn active face recognition units. Some people with prosopagnosia may be due to impaired structural encoding for faces, and they show failure in perceptual face processing tasks. In fact, they are unable to make any sense of faces (eg. age or gender) and judge whether two faces are same. Secondly, face recognition units contain stored descriptions of known faces. When a familiar

face is seen, face recognition units send signals to cognitive system and active person identity nodes.

Person identity nodes allow access to semantic information about the person. The impairment of face recognition units is not necessary for inability to recognize familiar people because person identity nodes may obtain information from non-facial cues. Some people with prosopagnosia may be due to connection problem between face recognition units and person identity nodes. For instance, Patient P. H. (Young, 1992) evinces impairment on tests

requiring links between face perception processes and face semantic information. Indeed, P. H. cannot recognize familiar people from their face alone but can relatively recognize them from their names.

Finally, face identity nodes contact name generation that can be disassociated from semantic information about the person. Face recognition units may be directly linked with name generation. Citing the same patient P. H. (Humphreys & Bruce, 1989) as an instance is helpful to understand this assumption; he also shows implicit recognition of faces. He could learn to associate names with appropriate faces faster than learn to associate wrong names with familiar faces. On the other hand, he couldn't show this improvement for learning to associate names with a person's semantic information such as his occupation.

It seems that there is intact access to face recognition units with impaired access to person identity nodes. Bruce & Young (1986) gave us a functional framework for face recognition, in which a number of stages were distinguished. The breakdown of one stage or the disconnection of two stages underlies prosopagnosia. Moreover, the collapse affects overt recognition, but it seems to be less effective in covert recognition. The deficit of configural processing in prosopagnosia Prosopagnosia has been associated with deficits in face configural processing that refer to inability for perceiving relations among facial features.

People with prosopagnosia may insensitively detect the spatial relations between facial features, and they tend to use featurebased strategy to recognize faces. One study concerns this spatially relational detection of

facial features in one prosopagnosic patient (as cited in Humphreys & Bruce, 1989). Patient R. B. was presented with face patterns (usually configurable features) or non-face patterns (jumbling and symmetrical features). R. B. had to decide whether the presented pattern was a face or a " nonface". Normal subjects responded faster to faces than non-faces. In contrast, R. B. esponded faster to non-faces than faces. R. B. 's slower response for faces suggests that he fails to take the spatial relations between facial features into account, and he need check each facial features serially to decide whether the given image is a face. Therefore, at least in this study prosopagnosia can be related to impairment of using configural processing. The other study (Farah at al. , 1995) compared processing of upright versus inverted faces in prosopagnosic patient LH and normal subjects using face-matching task. LH was remarkably accurate and faster at matching inverted face than upright faces.

On the contrary, normal subjects performed notably better for upright than for inverted faces. Also, normal subjects showed the impact of ' face inversion effect'; they could not process the an inverted face as a whole, they used feature-based strategy that leads to longer reaction time and less accurate for inverted than for upright faces. In contrast, patient LH showed ' inversion superiority effect'; that is, he performed better on inverted than upright faces. It can be assumed LH's better performance on inverted faces is due to invalid configural processing or effective features-based strategy.

Configural processing gives us an ides that is processing a visual item as a whole. In addition, configural processing may explain why face matching is

intact in some people with prosopagosia. Overview There are two functional explanations for prosopagnosia. These explanations propose that the procedures necessary for normal face recognition is not working properly, but the two explanations differ in their viewpoints. It appears that

prosopagnosia actually refers to a number of different types of impairments, so neither of the two explanations will account for all cases of prosopagnosia.

Moreover, neuropsychological explanations relate brain dysfunctions with prosopagnosia. References Bentin, S. , Deouell, L. Y. , & Soroker, N. (1999). Selective visual streaming in face recognition: evidence from developmental prosopagnosia. Neuroreport: An International Journal for the Rapid Communication of Research in Neuroscience, 10, 823-827. Bobes, M. A. , Lopera, F. , Coma, L. D. , Galan, L. , Carbonell, F. , Bringas, M. L. , & Valdes-Sosa, M. (2004). Brain potentials reflect residual face processing in a case of prosopagnosia. Cognitive Neuropsychology, 21(7), 691-718. Bruce, V. & Young, A. (1986).

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