Causes and types of synaesthesia

Synaesthesia is a hereditary and neurological condition (Roberston & Sagiv, 2005, pp. 12) whereby senses that are normally experienced separately, such as taste, smell, sound and so on, become ‘ fused’ together and are experienced at the same time. For example, someone may experience a taste in their mouth whilst saying certain words (lexical-gustatory synaesthesia) (Ward & Simner, 2003), or experience seeing colour when looking at different letters and numbers (grapheme-colour synaesthesia) (Ward, Li, Salih & Sagiv, 2007, Simner et al., 2005). Recent research has estimated that synaesthesia affects 4. 4 percent of people (Simner et al., 2006), known as synaesthetes, and has been proven to be a genuine phenomenon (Ramachandran & Hubbard, 2001b); not just a memory association between two senses. The neurological basis of this atypical condition and possible explanations will be explored, and finally what this information can tell us about the organisation of the human brain.

There are many different types of synaesthesia and research has looked into a large number of different cases. For example, a synaesthete experiencing vision-touch synaesthesia genuinely experiences the feeling of being touched just by looking at someone else being touched (Blakemore, Bristow, Bird, Frith & Ward, 2005). A synaesthete who has emotion-colour synaesthesia may see different colours for different people, depending on how well they know them (Ward, 2004). Synaesthetes with sound-colour synaesthesia see a colour when they hear a particular musical note (Ward, Huckstep & Tsakanikos, 2006). There is a vast array of different types of synaesthesia although grapheme-colour synaesthesia is reported to be the most common form of the condition (Day, 2001, cited in Ramachandran &

Hubbard, 2001b, pp. 6) and thus a lot of research is focused on this type of synaesthesia. However, whilst this grapheme-colour synaesthesia is the most common form, the numerous different types of synaesthesia can provide an insight into the different explanations and neurological causes of the condition.

As previously stated, synaesthesia is widely considered to be a genetic condition. Recent research by Barnett et al., 2008, has shown that forty-two percent of synaesthetes studied reported having a family member with synaesthesia, although results also showed that related synaesthetes are no more likely to share the same type of synaesthesia than unrelated synaesthetes are (pp. 883). A case study conducted by Smilek et al., 2001, demonstrated that monozygotic (identical) twins do not necessarily share the condition of synaesthesia which shows that whilst it is a genetic condition, identical genes do not necessarily mean shared synaesthesia.

But what is the cause of synaesthesia? There have been two major theories regarding the neurological causes of the condition and one theory that has been put forward is that of a cross-activation hypothesis. Ramachandran and Hubbard (2001b) propose that synaesthesia is caused by a ‘ cross-wiring’ between two areas in the brain (pp. 9). Ramachandran and Hubbard (2001b), focusing on grapheme-colour synaesthesia, noticed that the processing of colour information and the processing of graphemes occur in the same area, the fusiform gyrus (located in the temporal lobe). Ramachandran and Hubbard (2001b) suggest that considering the genetic component of synaesthesia, ‘ a single gene mutation causes an excess of cross-connections or defective pruning of connections between different brain

areas’ (pp. 9). Subsequently, when neurons associated with graphemes are activated, neurons associated with colours may also be activated at the same time, leading to the synaesthetic experience.

Ramachandran and Hubbard (2001b) have used their hypothesis to explain several phenomena regarding synaesthesia. One trend of synaesthesia is that it is thought to be ‘ more common in creative artists – poets, musicians, visual artists’ and so on (Ward, Thompson-Lake, Ely, & Kaminski, 2008, pp. 128). Ramachandran and Hubbard (2001b) suggest that cross-wiring could be the cause of this, arguing that if cross-wiring as a result of mutation causes synaesthesia, then a more diffused mutation will produce a more generally cross-wired brain. This would then create a greater tendency and more chances for ‘ creative mapping from one concept to another’ (pp. 17), which Ramachandran and Hubbard (2001b) suggest is the cause of higher numbers of synaesthetes among creative artists.

Ramachandran and Hubbard also suggest that a cross-activation hypothesis can explain why many synaesthetes report feeling certain emotions when perceiving ‘ multi-sensory’ stimuli, e. g. showing a grapheme-colour synaesthete graphemes in the colours different to the ones they claim they experience when looking at graphemes normally. Callejas, Acosta and Lupi áñez (2007) state that ‘ a feeling of discomfort’ (pp. 100) is connected with inconsistent coloured graphemes, that is, graphemes that are coloured differently to the synaesthete’s associated colours. Similarly, positive emotions are associated with consistently coloured graphemes. Ramachandran and Hubbard (2001b) propose that hyperconnectivity (cross-wiring) between the fusiform gyrus (the area that processes graphemes and

colour) and the limbic system (part of which is responsible for emotions) could mean that an inconsistent grapheme produces a ‘ disproportionately large emotional aversion’ (pp. 24).

This cross-activation theory is supported by Ramachandran and Hubbard (2001a), although it is important to point out that there is little evidence from other researchers other than Ramachandran and Hubbard (2001b, 2001a) confirming the hypothesis. In addition, Ramachandran and Hubbard’s (2001) cross-activation theory is based upon grapheme-colour synaesthesia which would question whether or not this theory could explain other types of synaesthesia. In the case of lexical-gustatory synaesthesia, Ward and Simner (2003) state that the areas in the brain responsible for taste and phonemes are ‘ geographically close’ to each other and so ‘ could promote direct connectivity between these regions’ (pp. 256), seemingly in support of a cross-activation hypothesis put forward by Ramachandran and Hubbard (2001b). However, it is also stated that a second neurological theory for synaesthesia is a better explanation for lexical-gustatory synaesthesia.

This second hypothesis of the neural basis of synaesthesia referred to in Ward and Simner (2003, pp. 256) is put forward by Grossenbacher and Lovelace (2001). Grossenbacher and Lovelace (2001) put forward an alternative theory to the hyper-connectivity (cross-activation) hypothesis; the disinhibited feedback theory. It is suggested that the neural connections within the brain of a synaesthete are not ‘ hyper-connected’ or ‘ cross-wired’ but are in fact the same as a non-synaesthete. The theory assumes that the neural connections in a synaesthete’s brain are ‘ connections that exist in normal adult human brains’ (pp. 40). Instead, it is suggested that feedback

from information sent back and forth from areas in the brain is not sufficiently inhibited, as would be in a normal brain (pp. 40). This causes ‘ interference’ between the processing stages when perceiving a stimulus, which in turn causes the synaesthetic experience. Grossenbacher and Lovelace (2001) argue that because non-synaesthetes are able to experience synaesthesia by using hallucinogenic drugs, it is evidence that the condition of synaesthesia uses ‘ normally existing adult networks’ (pp. 40) rather than the atypical cross-wired networks suggested by Ramachandran and Hubbard (2001b).

Whilst these two theories differ regarding the neural networks inside the brain of a synaesthete, both agree in the sense that a synaesthete’s brain operates differently to the ‘ normal’ brain of a non-synaesthete.

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