

BRAIN DAMAGE-INDUCED DOG PHOBIA

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Martin was a healthy and active 41 year old male who suddenly developed problems with simple, everyday motor tasks such as taking a walk. The severity of these problems seemed to fluctuate, but when he also started to experience repeated strong headaches centred at the back of his head, he decided to see a specialist.

Martin went to a hospital where several tests were run in order to determine the nature of his problem. These tests showed that in addition to his headaches and difficulties walking, Martin had developed a range of cognitive, perceptual and motor problems. Some of these included an inability to carry out simple mathematical calculations involving addition and subtraction, and difficulties performing tasks such as verbally identifying which finger was being touched by the physician (digital agnosia). In addition, it appeared Martin had poor motor control on the right side of his body.

After conducting these behavioural tests, doctors decided to scan Martin's brain using magnetic resonance imaging (MRI). MRI scanning provides a detailed three-dimensional photograph of the brain from which structural problems can often be identified. Photographs of Martin's brain showed that he had suffered a stroke. The stroke had caused bilateral damage to the posterior part of Martin's brain, particularly around the border between the parietal and occipital lobes. The most extensive damage was mainly in Martin's left posterior parietal cortex, a cortical area of the brain that plays an important role in processing visual input.

After a period of treatment and recuperation in the hospital Martin was released. He had to return to the hospital to attend physiotherapy sessions three days per week, but otherwise he resumed to a large extent the life he had led prior to hospitalisation. Martin was particularly pleased to once again be able to take up his habit of going for long walks around the streets of the city's old town, an activity in which he had previously engaged a number of times per week. To all extents and purposes it appeared Martin's recuperation was going well.

However, in the months following his release from hospital Martin began to experience some strange problems. While taking his walks, he reported becoming increasingly aware of a feeling of discomfort, almost fear, when he found himself in crowded streets and public areas in which objects (such as buses and people) were moving. This fear was greatly magnified in the case of any dogs he happened to encounter: he reported that the movement of a dog (and thus the dog itself) led him to experience bouts of excessive fear. This fear quickly developed in extremity to the point where Martin's response to the sight of a dog could be considered phobic. Indeed, he eventually gave up walking altogether in order to avoid any chance encounters with the animals.

As a consequence of these problems, and in spite of the fact that he did not initially relate them to the damage caused by his stroke, Martin decided to return to the hospital for further testing. Results from these tests showed that while he was still able to successfully identify visual objects such as a friend's face, a car or an animal, Martin's ability to see motion had been severely impaired: he found it very difficult to judge the direction, relative position and speed of moving objects. Interestingly, however, and in spite of the generality of his motion blindness, Martin continued to report that only moving dogs elicited his extreme fear, other objects causing him only discomfort or low levels of fear.

The development of this specific dog phobia was particularly surprising because prior to the stroke, Martin reported that he was completely unafraid of the animals and was in fact very fond of dogs. Although he did say that as a child he had been bitten, he pointed out that in subsequent years he had enjoyed being the owner of not just one but two dogs. On the basis of these past experiences, Martin acknowledged that his excessive fear of dogs was irrational. Even so, the fear did not dissipate during the year following his stroke. Whilst he reported that his discomfort in crowded places and in the presence of other types of moving objects became less acute with the passage of time, the same was not the case for Martin's dog phobia.

Conceptualisation

Phobias as a model of fear processing

Phobia case studies such as Martin's are important clinical tools because of their potential for providing insight into the neural mechanisms on which such phobias are based. Specific phobias are defined in the clinical diagnostic manual, the DSM IV (APA, 2000), as a marked and persistent fear which is excessive or unreasonable and is brought about only by the presence or anticipation of a specific object or situation. As such, the study of their neural correlates provides an opportunity both to identify the mechanisms underlying specific phobias and to better understand fear-processing mechanisms in the human brain more generally.

Relationship between the phobia and motion blindness

At a conceptual level, the visual processing deficits uncovered during the second round of hospital tests go some way to explaining the development of Martin's fears. They at least explain Martin's feelings of discomfort in the presence of moving objects: while he was still able to recognise and identify a bus, person or even a dog, his judgements regarding the movement of such things were severely impaired. On this basis it may be supposed that the very real danger of incorrectly planning his own movements in relation to the movement of other objects, thereby possibly resulting in injury or even death, formed the basis for Martin's fears of the objects. However, the existence of motion blindness does not explain the development of the specific dog phobia reported by Martin.

Neurophysiological models of phobia generation

In 1996 LeDoux proposed a model of phobia generation that helps understand, at a neurophysiological level, the onset of Martin's phobia. This model focuses on the role of the amygdala. The model was constructed on the basis of the important role known to be played by the almond-shaped neural area in generating fear responses, and on evidence that it has multiple connections to both subcortical and cortical neural sites involved in processing external stimuli (Amaral, Price, Pitkanen, & Carmichael, 1992).

In the model proposed by LeDoux (1996), phobias arise through a process in which automatic or 'unaware' fear responses are uncoupled from more controlled or 'aware' fear responses. LeDoux (1996) suggests that unaware responses arise as a consequence of processing in subcortical neural loops that incorporate the amygdala (thalamus-amygdala), while aware responses arise through processing in cortical neural loops into which the amygdala is integrated (thalamus-cortex-amygdala). According to the model

(LeDoux, 1996), phobias arise when the faster, subcortical responses are no longer mediated by the slower, cortical responses on which comparisons with previous experience are based.

Martin and the LeDoux model

Martin's case provides support for the LeDoux model. According to the model, phobias arise when subcortical unaware responses to external objects are no longer mediated by cortical aware responses to the objects. In Martin's case, damage suffered as a result of his stroke was focused largely on his posterior parietal cortex. Critically, this cortical area has been shown to play an important role in 'aware' processing of moving external objects such as buses and dogs. In a famous case study reported by Zihl and his colleagues (1983), a female patient with damage to her posterior parietal cortex was rendered effectively blind to motion. Indeed, on the basis of this type of evidence the posterior parietal cortex has been identified as being a part of the 'where' or motion processing visual pathway (see Mishkin & Ungerleider, 1982). Damage to this specific area in the case of Martin, therefore, provides a way to understand not only his general motion blindness, but also the onset of his phobia: if the stroke damaged the cortical aware response pathway then Martin's responses after the stroke are being mediated largely by the subcortical neural loop.

The reason that this neural uncoupling generated a phobia towards dogs in particular remains unknown. It is possible that Martin's early aversive encounter with a dog played a role (see Fyer, 1998). LeDoux (1996) suggested that ongoing non-fearful encounters with an object can extinguish an initial fear response. It is therefore possible that in the case of Martin, uncoupling of cortical and subcortical response loops served to reinstate an earlier fear response that had been overcome.

Application Questions

1. What difficulties did Martin experience that caused him to seek professional help?
2. To which specific neural locus did Martin suffer focal damage?
3. Describe in detail and with reference to other case studies the role played by this area in sensory processing.
4. What is the definition of a phobia?
5. Describe in detail a neurophysiological account of how phobic behaviours arise.
6. Describe how Martin's case is consistent or inconsistent with this account.

Resources

Blanke, O., Vavilav, V., Landis, T., & Safran, A.B. (2003), Dog phobia in a motion-blind patient, *Cognitive Neuropsychiatry*, 8, 211–221.

Gives the initial, more detailed account of Martin's perceptual deficits and brain damage, and provides a thorough discussion of the LeDoux (1996) model and other neurophysiological data.

References

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Anna Brooks and Ricky van der Zwan work at Southern Cross University, and Olaf Blanke is at the Ecole Polytechnique Federale de Lausanne. Together they are investigating the cortical processes involved in higher-order motion processing in normal and clinical observers.