Neurological Changes in People with PTSD Following Terrorist Attacks

Nicky Jonkhout

Abstract
Terrorist attacks on the Western world in the last decade have made this topic a pressing issue for social scientists. The aim of this article is to explore the neurological impact of terrorism on people who, following exposure to terrorist attacks, develop post-traumatic stress disorder (PTSD), compare this with other traumas, and see if this information could be used to the benefit of victims of terrorism. Three important brain areas seem to be involved in the development and maintenance of PTSD in terrorism victims. These are the amygdala, the frontal cortex, and the hippocampus. Differences between PTSD resulting from exposure to terrorist attacks and other traumas can relate to the variables of chronicity, proximity, and context. These differences, in combination with knowledge of fear conditioning and the consolidation process, can be examined in both experimental and clinical settings.

Keywords: terrorist attacks, neurological changes, amygdala, hippocampus, frontal cortex, PTSD.

Introduction

Terrorism has been around for a long time. It can be defined as the commission of violent acts or issuing of threats without any legal justification, or for a political or religious purpose. Before 9/11, most people in Western civilizations knew about terrorism, but for most it typically seemed to be a phenomenon remote from their own lives. The attacks on the twin towers in America and the killings of Theo van Gogh and Pim Fortuyn in the Netherlands were events that seemed to bring terrorism closer to home in America and Holland respectively. Both the fear generated by terrorism in the general population and the media coverage of terrorism have risen ever since. There is also a group of people who, directly or indirectly, have become victims of terrorism. The size of this group depends on the type of terrorist attack. In the case of 9/11, an extensive large number of people needed both physical and psychological care in the aftermath of the tragedy. The psychological impact on these people can be substantial. Psychological disorders that may develop among victims of terrorism include posttraumatic stress disorder (PTSD), generalized anxiety disorder, and depression (Oorsouw, 2012). This article will be limited to a discussion of PTSD.

PTSD is an anxiety disorder which is caused by a traumatic event. Symptoms can be categorized into three groups: recurrent trauma-related memories, avoidance, and hyperarousal. These symptoms can be either acute or chronic. There has been a great deal of research on the diagnosis, prevention and treatment of PTSD. Interventions like Critical Incident Stress Debriefing and treatments like Cognitive Behavioral Therapy (CBT) have been used to treat PTSD (Voerman, 2012). Additionally, guidelines have been developed to improve the process of clinical treatment. However, a better understanding of PTSD could lead to improvements in these areas. Recent technologies like Functional Magnetic Resonance Imaging (FMRI) give us a powerful tool to better understand the neurological mechanisms behind PTSD, which in part could be used to improve the diagnosis, prevention and treatment of the disorder. This article will examine the

neurological impact of terrorism on people who develop PTSD in comparison with people who do not, what different parts of the brain are involved, and the interaction of the affected brain areas in a person with PTSD. The neurological effects of PTSD due to a terrorist attack will be compared to effects resulting from other types of traumas that can cause PTSD. The last question that will be addressed is how knowing about the neurological impact of terrorism on PTSD can improve diagnosis, prevention, and treatment.

Exaggerated amygdala response

As stated earlier, PTSD is caused by a traumatic event. A psychological trauma can be defined as an event that threatens injury or death to others or self and that causes feelings of fear, horror or helplessness (APA, 2000). Two critical variables are important in developing PTSD: whether the trauma is experienced as acute or chronic, and whether it results from a controllable or uncontrollable stressor. Animal studies show that exposure to acute and uncontrollable stress produces extended hyper-excitability of the amygdala, which in turn causes the amygdala and related structures to be easily activated (Adamec, Blundell, & Burton, 2005). This in turn is related to higher vigilance and increased fearful responses to stressors. The uncontrollable part of the stress is important, because controllable stressors do not seem to activate the amygdala enough. Exposure to a terrorist attack can usually be characterized as both an acute and uncontrollable stressor. There are other acute traumas which can cause PTSD, like rape, automobile accidents, and natural disasters. On the other hand, there are also more chronic traumas like long time physical abuse and war. These chronic traumas cause a more anxiety-like behavior instead of the kind of fearful behavior seen in acute traumas. This separates terrorism from chronic traumas, because terrorism in Western countries usually is acute. Of course in war it is also possible to experience acute stressors alongside the chronic stressor of being in a war. The difference between terrorism and war is the absence of a chronic stressor in the former.

Amygdala experiments have been conducted in order to evaluate the effects of PTSD. In one study, survivors of 9/11 who developed PTSD were compared to 9/11 survivors who did not develop the disorder (Ganzel, Casey, & Glover, 2007). Both groups where shown fearful and calm faces while their amygdala responses were measured. Results showed that the PTSD group had higher bilateral amygdala activity compared to the group without PTSD. Results also showed that, within the PTSD group, proximity to the trauma affected the amygdala response. The closer people had been to the terrorist attack, the higher the amygdala response to the fearful faces. This separates terrorism from traumas like rape, car accidents and long time physical abuse, because it is possible to be far away from a terrorist attack and yet still develop PTSD. In contrast, rape, car accidents and long time physical abuse are by nature events that involve the “close proximity” of the individual who later develops PTSD. War, on the other hand, bears a higher degree of resemblance to terrorism with regard to proximity.

Barlow (2002) created a model of possible causes of PTSD in which different kinds of triggers constitute the true alarm (trauma), but through conditioning, there is also a learned alarm or false alarm (conditioned stimulus). This is what causes the recurrent trauma-related memories. The amygdala has been shown to be involved in the conditioning of the stimulus in an air puff (unconditioned stimulus) experiment. More research is needed to figure out the exact role of the amygdala in fear

conditioning. In addition, there can be no clear distinction made in conditioning in different types of traumas. The only difference is in the kind of false alarms that are conditioned with the trauma.

Deficient frontal cortex functioning

Besides the amygdala, another brain area plays an important role in the development and maintenance of PTSD following a terrorist attack. Shin et al. (2001) used FMRI to compare trauma victims with and without PTSD. While in the scanner, subjects had to perform the "Emotional Counting Stroop." In this test, subjects must count the number of words on the screen that are either neutral, generally negative, or combat-related. Subjects with PTSD exhibited no increase in anterior cingulate cortex activity (which is part of the frontal cortex) in the combat-related condition, while subjects without PTSD did show increased activity. The neutral and generally negative condition showed no differences in either group. Deficient frontal cortex activity has been linked to exaggerated amygdala activity. In another fearful versus happy faces study, Shin et al (2005) found a negative correlation between frontal cortex activity and amygdala activity. It has been proposed that the frontal cortex has an inhibiting function on the amygdala. However, more research is needed to determine the exact direction of causality between the frontal cortex and amygdala activity.

As noted above, Shin et al (2001) showed that the frontal cortex is important for inhibiting the amygdala according to context. In victims of a terrorist attack with PTSD, the frontal cortex would only inhibit response in the Emotional Counting Stroop which is not related to the terrorist attack. This means there is a difference between PTSD caused by different traumas in relation to stimuli used in experiments and diagnosis. For terrorist trauma, victims with PTSD, only stimuli related to terrorist attacks can be used to provoke an emotional response.

In addition to showing the role of the frontal cortex in the inhibition of the response to emotional stimuli of the amygdala, new research has also identified a role of the frontal cortex in the extinction process of conditioning. As stated earlier, the amygdala has been shown to be involved in fear conditioning. Frontal cortex activity, on the other hand, and especially activity in the ventro-medial prefrontal cortex (vmPFC), was found to predict extinction success after a 24-hour delay, and was negatively correlated with amygdala activity (Gottfried & Dolan, 2004). This information is consistent with animal studies, suggesting that the vmPFC may inhibit the amygdala during the recall of extinction. As appears to be the case with other traumas, deficient extinction may make people more vulnerable to the development of PTSD following a terrorist attack.

Deficient hippocampus functioning

A third brain area that plays an important role in the development and maintenance of PTSD is the hippocampus. In order to understand the role of the hippocampus in the stress system, it is important to appreciate the importance of the hypothalamic-pituitary-adrenocortical (HPA) axis. When people are under stress, the hypothalamus activates the anterior pituitary gland, which releases adrenocorticotropic hormorne (ACTH) into the bloodstream. This ACTH reaches the adrenal cortex, which releases cortisol in reaction. Coritsol is a stress hormone that enables the body to fight infections, sustain alertness, and heal wounds. When cortisol reaches the hippocampus, it inhibits the hypothalamus from releasing more cortisol. This is why the hippocampus has an

important role in regulating the stress response. Without the hippocampus, the production of cortisol by the HPA axis would proceed unabated.

Research on animals has shown that, through prolonged exposure to cortisol, severe and chronic stress can damage the hippocampus (Gilbertson et al., 2002). More recent studies on humans have found smaller hippocampus volume in trauma victims who developed PTSD in comparison to trauma victims who did not developed the disorder. To find out whether this smaller hippocampal volume is caused by PTSD or a pre-existing condition, Gilbertson et al. (2002) used monozygotic twins. They found that the smaller hippocampus was not only present in the twins with PTSD, but also in the twins without PTSD who, unlike their siblings, had not been exposed a traumatic event. This means that the smaller hippocampus is not caused by PTSD, but is a pre-existing condition which makes people more vulnerable to PTSD. So, before a terrorist attack, some people are already pre-disposed to develop PTSD as a result of having a smaller hippocampus.

Like the amygdala and frontal cortex, the hippocampus also has a role to play in fear conditioning. As stated earlier, the amygdala is involved with generalized fear conditioning. In contrast, the hippocampus seems to be more involved in contextually based fear conditioning. The hippocampus is also involved in memory retrieval of context-dependent extinction. Patients with amnesia resulting from hippocampus damage were unable to recover fear responses in the same context following reinstatement, despite showing initial fear acquisition (LaBar & Phelps, 2005). This means that the hippocampus is necessary for eliciting contextually based fear responses.

Plasticity of the brain and consolidation of memory

Plasticity of the brain, especially in the hippocampus, may be important in the chronic development of PTSD following an acute trauma. Researchers were successful in erasing memory of a traumatic event in animals with a lesion to the hippocampus (Bremner, Southwick, & Charney, 1999). However, this effect only occurred during the first month after the trauma. After the first month, the memory is stored in the cortex and becomes more resistant to modification. This means that there is a critical period during which the traumatic memories are stored in the hippocampus, and during which they are susceptible to consolidation. Early intervention in this first month after a terrorist attack, according to these findings, could be important to prevent the chronic development of PTSD. The time of this early intervention, in combination with the kind of intervention, is very important because the wrong combination can actually worsen the severity of an individual's PTSD. Administering the medication benzodiazepine to acute trauma victims increased the long-term severity of PTSD to a much greaer extent than when they were administered placebos (Gelpin, Bonne, Peri, & Brandes, 1996). Moreover, some early interventions like Critical Incident Stress Debriefing and Psychological Debriefing are not only useless for most trauma victims, but in some instances, can actually impede recovery from a traumatic event (Gist & Devilly, 2010).

Discussion

The aim of this article was to explore the neurological impact of terrorism on people who develop PTSD, compare this with other traumas, and see if this information could be used to the benefit of victims of terrorism. Three important brain areas seem to be

involved in the development and maintenance of PTSD in victims of terrorism. These are the amygdala, the frontal cortex, and the hippocampus.

The amygdala shows exaggerated fear responses. Terrorism is different from other traumas in that it is usually acute instead of chronic. This causes responses that are more fearful than anxious. What is more, the proximity to a terrorist attack can vary, whereas proximity to other traumas is inherently fixed. The closer in proximity the terrorist attack is, the greater the probability that an individual will develop PTSD. This information should be kept in mind when dealing with victims of a terrorist attack. No studies were found that revealed whether these differences could be used in prevention and treatment. It is possible that different interventions are indicated for the fearful and anxiety subtypes. Proximity to the attack could be an indication of the intensity and duration of the treatment. More research on these topics is needed in order to answer these questions.

The frontal cortex shows deficient activity among survivors of terrorist attacks who develop PTSD. This brain area has an inhibition function on the fear responses of the amygdala, and deficient activity in that area means that it is not adequately inhibiting undesirable fear responses. It is important to note that the frontal cortex and the hippocampus are responsive to context. The context is what makes exposure to terrorist attacks different from other traumas, and it is important to take context into account in treating PTSD survivors of terrorism in an experimental or clinical setting. Virtual reality in combination with function neuroimaging has been used in experimental setting (Maguire et al., 1998). This could be a powerful tool in research that seeks to differentiate the kinds of traumas that cause PTSD. This technology could perhaps also be useful in screening, diagnosis, prevention and treatment. Psychologists could use virtual reality to simulate the trauma the victim was exposed to, thereby enabling an accurate diagnosis and perhaps also treating the patient by mere exposure. More research is needed to explore and validate these possibilities.

The hippocampus, like the frontal cortex, shows deficient activity and was found to be smaller. The smaller volume is a pre-existing condition and makes people more vulnerable to developing PTSD. It might be possible for clinicians and researchers to use this phenomenon to their advantage. After a large-scale terrorist attack, it could be possible to predict which people are more likely to develop PTSD on the basis of the volume of their hippocampus. With current imaging techniques, it would be far too expensive to scan all victims, but in the future it might be possible to scan large groups of people in a more cost-effective manner.

All three of these brain areas were linked to the fear conditioning and extinction process. The amygdala is linked to conditioning, the cortex to extinction, and the hippocampus to context-dependent conditioning and retrieval of extinction. More research is needed to understand the complex interplay in fear conditioning and extinction among these three brain areas. However, this information could be important for the prevention and treatment of PTSD. By influencing these areas through medication or transcranial magnetic stimulation, during conditioning or retrieval of extinction, the conditioned fear might be influenced.

Plasticity of the brain makes it possible to eliminate the traumatic memory in the first month by making a lesion in the hippocampus. After this first month, the memory is more strongly consolidated in the frontal cortex, and will be more resistant to modification. This effect could be used in

early interventions to extinguish or modify memories stored in the hippocampus, but it is important to conduct further research, because past interventions have in some cases made the PTSD worse instead of better. It is important to take these findings into account in order to critically evaluate current psychological interventions following a terrorist attack. Immediately after 9/11, many psychologists were rushed to the site of the attacks, to provide psychological support. What we do not know is how many of the treated victims were actually worse off due to this intervention.

In conclusion, three important brain areas are involved in the development and maintenance of PTSD in terrorist victims. These are the amygdala, the frontal cortex and hippocampus. Differences in the response of PTSD sufferers exposed to terrorist attacks as opposed to other traumas can be observed in relation to the dimensions of acuity/chronicity and proximity. These differences, in combination with knowledge of fear conditioning and the consolidation process, can be used in the experimental and clinical setting.

Reflective paragraph

This article was written from a psychological viewpoint, and has focused on the cognitions and neurobiology of individuals diagnosed with PTSD as a result of exposure to terrorism. Cognitive neuroscience is a field concerned with the scientific study of biological substrates underlying behavior and cognition. The question addressed in this paper concerns how behavior and cognitive functions are carried out by the brain. Due to the multidisciplinary nature of cognitive neuroscience, its practitioners come from very different backgrounds. Disciplines represented in this field are neurobiology, bioengineering, psychology, psychiatry, neurology, physics and philosophy. Other disciplines like sociology, anthropology and pedagogy could also make contributions to this field. A topic of interest in sociology is the possible impact of terrorism on the larger society through brain changes in individuals. The current research was mainly done in Western societies. An anthropologist could perhaps address whether PTSD results in the same neurological changes in people from different countries. Those in more individualistic countries could perhaps experience different changes. A pediatrician could perhaps look at the neurological changes in very young PTSD victims , evaluating impact on the child's family over time. Many different disciplines and viewpoints would likely contribute to increased understanding, and the development of effective treatments of PTSD.

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