Brain damage



Essay cover sheet Essay Title: What does the study of brain injury and disease tell us about normal brain functioning? Word count (Excluding title and references section): 829 What does the study of brain injury and disease tell us about normal brain functioning? To understand atypical brain function, it is important to distinguish the expectations for a typical brain function. It is true that many diseases or injuries result in impairments in cognition; as different areas of the brain is designed to control specific cognition and processes.

For example the hemispheres are known to control different functions such as language, spatial judgements, reasoning and abstract notions (Martin, 2003). Whilst, the frontal lobe is famous for processing memory, attention, personality, and behaviour (Martin, 2003). Parietal lobe tends to control spatial and sensory information; whereas occipital lobe processes visual stimulus. Language, retrieval of memory and behaviour is administrated through temporal lobe (Martin, 2003). Finally, the limbic system tends to control emotion as well as short term memory (Martin, 2003).

Brain damage is the degeneration or abnormal growth of brain cells, which can be the result of outer (injury) or inner (disease) influences. Therefore, in cases of brain disease there are biological and psychological impairment that causes abnormality in the brain such as Alzheimer's disease, Dementia, Amnesia and Aphasia; which some may be genetically inherited. Brain disease such as Alzheimer's help us to understand the processes of the central executive function, which assists in producing controlled and flexible responses (Groome, 2006).

In Alzheimer's Disease (AD), this process is replaced by automatic and stereotyped responses; thus, it results in a dysexecutive syndrome (Groome, 2006; Baddely & Wilson, 1988). Conditions such as AD, symptoms like amnesia and dementia are known to involve damage to frontal lobes (Groome, 2006); therefore, there are impairments in abstract and conceptual thinking, attention, behaviour and memory. For example, lesions to frontal lobes result in difficulty in retrieving contextual information (Parkin, Walter & Hunkin. 1995); thus, when presented with series of items, the frontal lobe patients are likely to remember the item shortly after, however, are not able to specify the order in which they were presented in (Swain, Polkey, Bullock & Morris., 1998). Additionally, patient H. M had part of his medial temporal lobes removed due to his epilepsy; however most of his hippocampus were also removed to reduce seizures (Groome, 2006). Although his condition improved, he developed amnesia, which affected his short term memory (Groome, 2006).

This meant he was no longer able to form newmemoriesdue to the lesion made to his hippocampus. Schizophrenia is a psychiatric disorder that results in several cognition impairments such as: deficit in memory and learning, poor abstract thinking and problem solving, difficulty in sustaining attention. Studies suggest that such patients are likely to suffer from dysfunction in areas such as: frontal lobe, temporal lobe, left or right hemisphere and basal ganglia (Blanchard & Neale, 1994). Heinrichs & Zakzanis (1998) illustrated how schizophrenic patients tend to have impaired verbal memory.

Furthermore, injuries or lesions to frontal and temporal lobes of the cerebral cortex can result in language deficiencies, such as Wernicke and Broca's

aphasia (Groome, 2006). For example, patient Phineas Gage suffered an injury to the Broca's area (damage to frontal lobe), showed inability to produce language, as there was no sentence structure and the language was just string of disjointed words (Fleischman, 2002; Groome, 2006). Gage also showed emotional inbalance, where he was described to have acted out of character and was more aggressive, which was the result of damage to amygdale (Fleischman, 2002; Groome, 2006).

Whilst, Wernicke's aphasia (damage to temporal lobe) results in meaningless production of language; the patient is able to produce sentences but it does not convey information (Groome, 2006). Moreover, studies on blindsight suggest that patients such as DB have no conscious experience of perceived surroundings, however they manage to use the visual information at some other level to guide them through the surrounding world (Groome, 2006). It is believed that this neglect is the result of damage to the contralateral hemisphere.

For example patients who have lesions to the right hemisphere have left spatial neglect, thus will fail to notice the left side of space (Groome, 2006). Such studies, highlight that spatial neglect is not a unitary disorder but a cohort of deficits. Thus, it allows us to distinguish between conscious experiences and the ability to respond appropriately to stimulus (Groome, 2006). The brain is a major organ that executes functions and vital processes essential to human activity; for example thinking, memory, language and emotions.

The use of brain injuries and diseases, enables us to identify better models to comprehend cognition; as these areas will create a natural lesion in the

processing mechanism. Thus, it identifies specific elements that play a major role in cognition. The mentioned studies show specific cognitions can be used to process information in a particular way. To ensure that specific cognitive model is processing properly, it is important to look at the neural activity of that region. Lack of activity in the interested area shows impairment in the model and that region.

Therefore, by studying that specific area further, we can highlight its activity and information processing. This will allow us to understand normal functioning of the brain further. However, it is important to note that these theories might not be fully supported. For example, not much is known about the central executive system; thus, the vagueness only allows to corroborate processes that are not fully understood. Therefore it is vital to critically analyse theories before applying them. References Baddeley, AD. Kopelman, MD., and Wilson, BA. (2004). The Essential Handbook of Memory Disorders for Clinicians. John Wiley & Sons, Ltd Blanchard, J. J. & Neale, J. M. (1994) The neuropsychological signature of schizophrenia: generalized or differential deficit? American Journal of Psychiatry, 151, 40-48. Fleischman, J. (2002). Phineas Gage: A gruesome but true story about brainscience. Boston: Houghton Mifflin. Groome, D. (2006). An introduction to cognitive psychology. Hove: England. Heinrichs, R. W. & Zakzanis, K. K. 1998) Neurocognitive schizophrenia: A quantitative review of the evidence. deficit in Neuropsychology, 12, 426-445. Martin, G. N. (2003). Essential biological psychology. London: Arnold. Parkin, Al., Walter, BM., and Hunkin, MM. (1995). Relationships between normal aging frontal lobe function, and memory for temporal spatial information. Neuropsychology, 9, 304-312.

Swain, SA., Polkey, CE., Bullock, P. & Morris, RB. (1998). Recognition memory and memory for order in script-based stories following frontal lobe excisions. Cortex. 34, 25-45.