21.4 ~~5.2.1.4~~ ~~E~~Aetiology and ~~p~~Pathogenesis of ~~a~~Aphasia

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Comment from the lector for English language

l.21 what determines the occlusion: CE alone? Or both haemorrhage and CE? I've assumed the former; is it OK?

Formattato: Inglese (Stati Uniti)

Aphasia is frequent in acute and subacute stroke situations, affecting from~~approximately~~ 21% to 38% of patients who survive stroke (Hoffmann and Chen 2013)~~[6]~~. Factors that increase an individual's likelihood of experiencing stroke and aphasia are hypertension, diabetes, high cholesterol, a smoking habit, stress, inactivity, excessive consumption of alcohol, and dietary intake high in fat and sodium (Yusuf et al. 1998)~~[7]~~.

Aphasia syndromes are predictive of lesion location~~localization~~, stroke pathophysiology and recovery (Croquelois and Bogousslavsky 2011)~~[3]~~. Most patients with aphasia undergo multimodality magnetic resonance imaging (MRI) or computed tomography (CT) in the course of their stroke workup. Consequently the location~~localization~~ of the damaged brain area is usually not unknown~~an issue~~ (Hills et al. 2004)~~[5]~~. Nowadays the detection of the mechanism that finally leads to language impairment is the most important factor for preventing recurrence or worsening of an aphasia syndrome. Aphasia subtypes and aetiologies, in fact, have some significant associations. In particular, anomic aphasia appears significantly associated with small-ve~~a~~ssel disease (SVD), while global aphasia patients mostly have cardioembolic (CE) causes that lead to the occlusion of the middle cerebral artery before~~prior to its~~ it branches~~ing~~. Wernicke's aphasia is frequently related to haemorrhage or CE that determines the occlusion of the posterior temporal and parietal branches of the middle cerebral artery (Hoffmann and Chen 2013)~~[6]~~. Broca aphasia can be caused by SVD, haemorrhage, large vessel disease or CE that causes an occlusion of the anterior branches of the middle cerebral artery.

As far as ~~it is concerned~~ the lesion location ~~lization~~ (which~~that~~ finally determines the characteristic of aphasia)~~,~~ is concerned, in long-lasting language deficits conforming to the characteristics of a Broca aphasia, ~~a~~ damage to~~of~~ a sizable area of the inferior frontal gyrus is usually present. In other cases, the infarct may be in the territory of most of the anterior branches of the middle cerebral artery and will have damaged not just the cortex of the frontal operculum (Brodmann's areas 44 and 45), but also have extended into the underlying white matter, and involve the insula and the basal ganglia, as well as the inferior sector of the pre-central gyrus. In the acute stage~~epoch~~ of this condition~~,~~ a small and circumscribed infarct of the frontal operculum can also produce a non-fluent language deficit with all the characteristics of Broca aphasia.

In Wernicke's aphasia the lesion is usually located~~localized~~ in the posterior sector of the superior temporal gyrus (Brodmann's area 22) with extension in the middle and inferior temporal gyri (Brodmann's areas 37, 20 and 21) and into part of the inferior parietal lobule, destroying the lower sector of the supramarginal and the angular gyri (Brodmann's areas 40 and 39). Conduction aphasia is~~if~~ more often caused by ~~a~~ damage to the cortex of the supramarginal gyrus and to its underlying white matter, with a compromise of the arcuate fasciculus, the pathway that~~which~~ connects the posterior and anterior language areas. The reporting of conduction aphasia in patients with lesions in the left auditory cortex and insula suggests that the course of the arcuate fasciculus~~'s course may be~~ is a large sheath of white matter whose lower segments course~~s~~ under the insula.

Global aphasia is related to ~~an~~ extensive damage to the frontal, parietal and temporal regions. In transcortical aphasia the damage may involve the angular gyrus (Brodmann's area 39) and the posterior sector of the middle temporal gyrus (Brodmann's area 37). On occasion the lesions may extend into the lateral aspect of the occipital lobe (Brodmann's areas 18 and 19). Finally, anomic aphasia is frequently related to lesions located exclusively in the left temporal pole.

Although aphasia subtypes and their relationship to stroke aetiology are useful (they~~ and~~ aid~~ can assist~~ the clinician in~~with~~ the prognosis and may influence aphasia therapy), aphasia can also be characteri~~z~~sed in terms of disruption of specific cognitive processes underlying language tasks, as~~keeping in mind that~~ the mirror neuron network is the likely neurobiological substrate of language by making cross-modal cognition possible (Arbib 2011, Hoffmann and Chen 2013)~~[2, 6]~~.

Other causes of aphasia besides stroke include brain tumour, traumatic brain injury, cerebral hypoxia, large cerebral haemorrhage, cerebral infections, as in herpes temporal lobe encephalitis or HIV~~-~~-related leucoencephalopathy~~haty,~~ and dementia. For brain tumours, as for stroke aphasia, is related to the pathological involvement of brain language areas. For the remaining cases~~causes~~, aphasia may due to damage of the brain language areas ~~and/~~or to general cognitive impairment due to diffuse brain damage (Al-Khindi et al. 2010, Fitzgerald et al. 2010)~~[1, 4]~~.

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