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COGNITIVE MODELLING OF FACE PROCESSING: EVIDENCE FROM ALZHEIMER PATIENTS

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Abstract—This is a prospective neuropsychological study on face processing in Alzheimer's disease (AD). The aim was to assess the prevalence and the nature of face processing disorders in AD, and at investigating possible inter-test dissociations within the framework of currently used face processing models. A standardized four-test battery of unknown face discrimination and familiar face recognition was given to 30 mildly deteriorated patients with AD. Half of the patients performed below the cut-off in at least one of the tests. Deficits in familiar face recognition tests were more frequently observed than deficits in unknown face discrimination tests. There was no correlation between impairment of face processing and overall cognitive impairment or visual disorders. A multiple single case approach allowed us to elicit statistically warranted double dissociations between tasks assessing unknown face discrimination and tasks assessing familiar face recognition. Moreover, the ability to decide whether or not a stimulus is a face or a non-face has proven to be a non-mandatory step to further process the face stimuli. All together, these findings support the hypothesis that distinct pathways are involved in the processing of unknown and familiar faces, as posited by Bruce and Young [*Br. J. Psychol.* 77, 305-327, 1986].

Key Words: face processing; Alzheimer's disease.

INTRODUCTION

Alzheimer's patients (AD/pts) often experience difficulties in identifying people, failing to correctly assign the right set of memories to a given face, and relying on the interlocutor's prompts for recognition [2, 46]. Several processing defects can be held responsible for AD/pts failure to recognize faces [15]. This combination of defects is rather difficult to tackle solely from a clinical perspective. Very few studies have investigated experimentally the issue of face processing in AD [15]. Moreover, these studies have concentrated on a given aspect of facial stimuli process, such as memory for faces [19, 67], recognition of facial expressions [2, 37, 38], visual discrimination of faces [27, 66] and poor familiarity judgement for famous faces [6, 36, 47, 48].

Although interesting in their attempts to quantify different aspects of facial processing deficit in AD/pts, these studies make no effort to disentangle each specific deficit of face recognition using a cognitive frame of reference. Thus, the question remains as to whether or not AD/pts show patterns of dissociation similar to those reported in focal brain damaged

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subjects [68]. To this end, it is necessary to rely on a range of tasks testing different aspects of facial processing.

Recently Young *et al.* [70] drew attention to some of the problems embedded in the interpretation of the data gleaned from the face perception literature. Our experimental design seeks to eliminate some of the risks they identified: we tested each one of our subjects within a single testing session, using the same testing material and the same procedures for all, therefore eliminating the problem of dissociations deriving from different reports. In order to reduce the danger of equating tests and abilities, we tested each ability with two different testing devices. Lastly, we employed a multiple single-case approach to account for the risk of a dissociation occurring by chance.

Given the importance of inter-test associations and dissociations in verifying hypotheses embraced by a cognitive model, we maintain that AD/pts are particularly promising candidates for investigations of this kind [5, 7, 59]. In fact, heterogeneity characterises the pathological, metabolic and neuropsychological features of the degenerative process in AD: neuroanatomical lesions can vary between the two hemispheres, across the different cortical layers and regions at given times, and, more relevant to our issue, the involvement differs among different patients [12, 65]. Degeneration consists of a progressive heterogeneous neuronal thinning-out. The biological evidence of this type of involvement is still far from understood. However, such a feature fits in with the typical behavioural decay in AD [44]: a progressive convergence of neither 'pure' nor complete defects in several cognitive domains. In AD, contrary to what happens with focal lesions, the incomplete encroachment on regional neuronal networks might facilitate the emergence of fine-grained dissociations. This was already pointed out by Pick in 1908 [53] when he speculated that "... the particularly slow course of the senile atrophy and the evidence that it encroaches upon single areas of the brain, and even sections of them, allow to understand why the consequences of this process (i.e. senile atrophy) are so selective. ... One can assume that the atrophy systematically destroys organized groups of neurons and, as a consequence, selectively impairs the relevant functional system".* Moreover, in a previous study with AD/pts, it has been established that the combination of cognitive symptoms appears to be non-random, since no examples of 'nonsense syndromes' were found with respect to cognitive theory and modelling [5]. The dissociations within our group of AD/pts will allow us to discuss which among the available cognitive models better accounts for our data. In searching for possible dissociations, we fulfilled Shallice's criteria [57] by means of a sound statistical design [28].

Aims of this study are (i) to assess the prevalence of early AD/pts showing psychometric face processing impairments and to try to qualify them. Having employed a battery of tests tackling different aspects of the process and given the well known disproportioned impairment of the mnemonic vs the perceptual domain in early AD [49], we predicted that the deficit would emerge more frequently in familiar face recognition than in unknown face discrimination tasks.

(ii) We also aimed at ascertaining the relationship between dementia severity, basic visual abilities and the performance on face tests. This latter approach aims at contributing to the

*"... der äusserst langsame Verlauf der senilen Atrophie, der Umstand, daß diese sichtlich, ... die einzelnen Organen des Gehirns und gewiss auch die Teile dieser Organe angreift, lassen es verständlich erscheinen, wenn die Wirkungen dieses Prozesses sich oft als solche von elektiver Anordnung darstellen. (...) Kann man annehmen daß eine systematisch gleichgeartete Neuronengruppe, ... früher als die übrigen der Atrophie verfällt, und dadurch ganz isoliert die Funktion dieses Systems ausfällt; es wäre dadurch zu einer, ... funktionell umschriebenen Ausschaltung ... kommen."

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