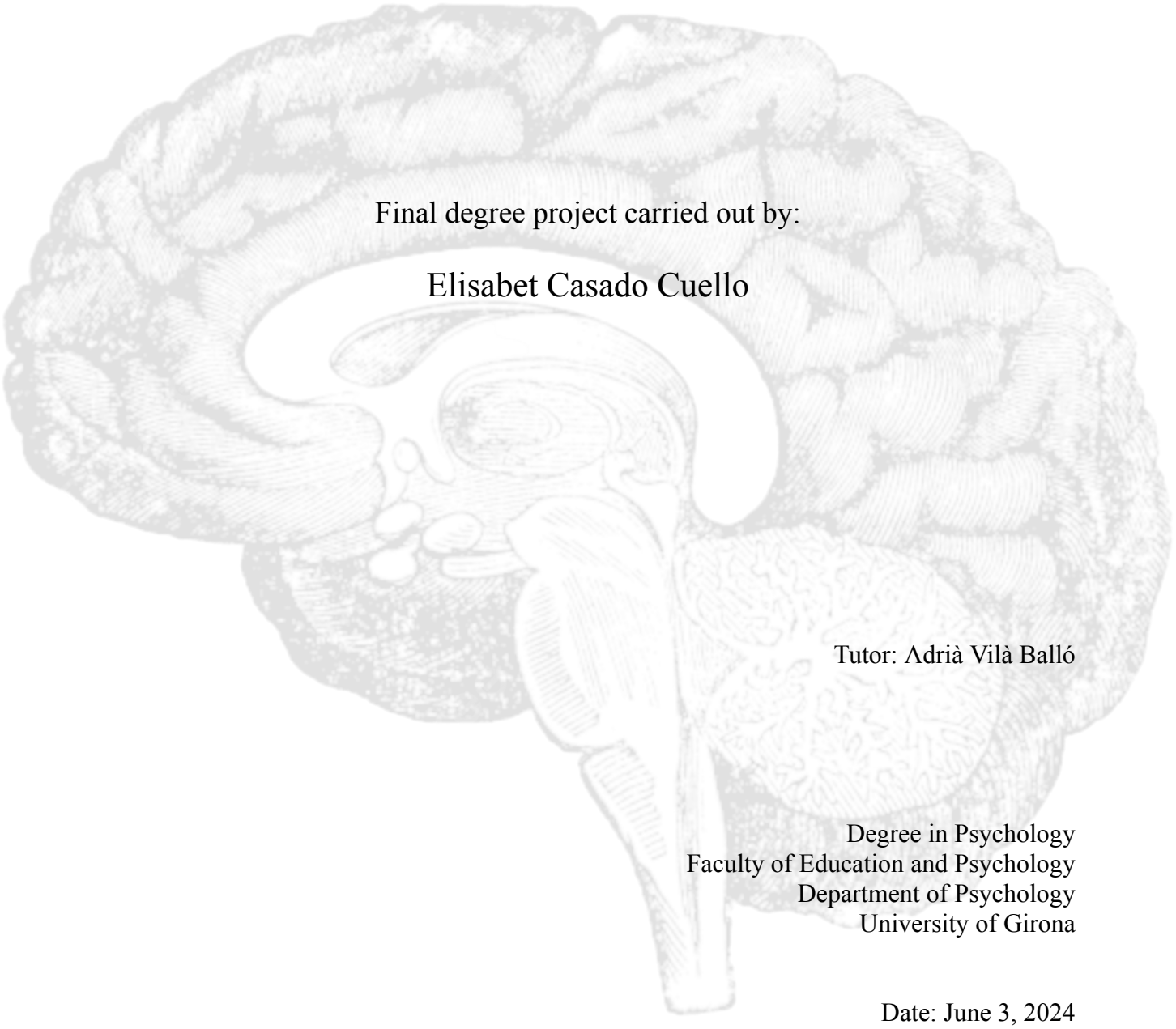
Universitat de Girona

FACIAL PERCEPTION IN ALZHEIMER'S DISEASE:

Investigating the gnosic line of facial recognition



Final degree project carried out by:

Elisabet Casado Cuello

Tutor: Adrià Vilà Balló

Degree in Psychology
Faculty of Education and Psychology
Department of Psychology
University of Girona

Date: June 3, 2024

ACKNOWLEDGEMENTS

A en Pere Combis i l'Aroa García; aquest treball porta la vostra essència, al igual que jo.

Gràcies a tots aquells que, conscient o inconscientment, heu afegit el vostre granet de sorra; els amics, la família, i les grans professionals que m'he trobat en aquests últims anys de carrera que m'han recordat la meva passió per la psicologia i m'han fet seguir endavant sense mirar enrere ni penedir-me de cap decisió professional.

Gràcies a totes les grans científiques que sense saber-ho es converteixen en les nostres referents.

INDEX:

ABSTRACT

Background. Deficits in facial recognition in people with Alzheimer's disease (AD) is a highly attributed and studied aspect in relation to memory problems. Although the reality of memory deficits in the clinic of the disease is a fact, this is not the only explanatory aspect for facial recognition problems; this can also be explained by gnosic problems. Evidence literature exists for this line but it is highly understudied.

Objective. To study whether there is a specific deficit in facial visual perception in patients with AD, contrasting the results with a control group.

Method. A sample of 10 participants (5 clinical and 5 controls) were evaluated in a cognitive screening test (MMSE), one of facial visuoperceptive skills (the central paradigm of the study, the BFRT-r), and two of generic visuoperceptive skills (RBANS line orientation test and VOSP number location test).

Results. The analyses of the data collected in the tasks showed significant differences between groups in the facial perception test with a lower performance by the clinical group, while not in the generic visuoperceptives.

Conclusions. The study suggests significant evidence on the deficit in facial perception on the part of Alzheimer's patients in the sample compared to the control group.

Keywords: Alzheimer's disease (AD), facial perception, face recognition, visuoperception, Benton’s Face Recognition Test revised (BFRT-r)

1. INTRODUCTION

1.1. JUSTIFICATION, INTEREST AND RELEVANCE OF THE TOPIC

This work aims to increase the bulk of the existing literature with respect to what is known about how the deficit in the recognition of family members works in people with Alzheimer's disease (AD onwards). This deficit could be given by two aspects: on the one hand, mnesic -memory problems-, and on the other hand, gnosics -perceptual problems-. It is a fact that people with AD present memory deficits as a symptom par excellence, but is the lack of recognition of people in their known circle given solely because of this? The fact is that the Gnosic aspect is still poorly studied, as explained by Lavallée et al. (2016), and there is also a lot of literature that talks about faces as a very special visual stimulus (for example, Werheid & Clare (2007)) and that must be studied in isolation from other stimuli. All this is what led me to the question that, if I evaluated this visual perceptual capacity specifically facial, in a group with AD and a control group, would I find significant differences? In this sense, it is essential to emphasize the importance that studies should be aimed at diagnosing the disease as early as possible, either through biomarkers and/or identification of cognitive profiles (Quintana, 2009). This research will provide information to many fields of research, such as psychology, neuropsychology, neuroscience, clinical (evaluation, diagnosis and intervention) and at the social level (improvement of quality of life).

The reason for choosing AD as the study pathology is because it is the leading cause of neurodegenerative dementia worldwide (Manzano et al., 2018), and one of the great health challenges of the twenty-first century (Scheltens et al., 2016). It is fast becoming one of the most expensive, lethal and heavy diseases of this century (Scheltens et al., 2021). The most recent data indicate that, by 2050, the prevalence of this dementia will double in Europe and triple globally (Scheltens et al., 2021). Therefore, all the information that can be obtained to thicken the literature of this disease and thus, with the perspective of improving the quality of life of these people, is important and we must try to investigate it.

As for studying faces specifically as visual stimuli, it is important to understand that, apart from their own patients, the people who suffer most from the effects of the disease are family members or people from the close circle seeing how that person they love is not able to recognize them. Certainly, due to the mnesiac nature of the disease itself, this rote role has a great weight, but if it is not unique but there are more factors, it is necessary to study them to try to provide literature in research against Alzheimer's. In addition to this, in a certain way

this study will also provide general information on how the perceptual system of people works, since a source of great wealth of information to study the human brain and its processes, is through pathologies -and what better than one of such scope as this-. Therefore, research on facial recognition in Alzheimer's disease can improve our knowledge of both neurotypical facial recognition and help discover new ways to help patients cope with their symptoms (Gregory, 2018).

On the other hand, the choice of topic is also justified by my interest in neuropsychology, in research, and because in my 4th year career internship I was able to deal with people with different dementias -mostly cases of AD-, something that aroused even more interest and desire to develop this work.

Thus, this work is aimed at deepening my knowledge in psychology and neuropsychology, in AD and what implications it has, and in how the facial perceptual process really works in this population. From the previous relevant bibliographic research, and then with the subsequent practical part, passing a battery of tests to two populations -clinical with AD and non-clinical control- to study this facial perceptual factor, a discussion is raised with the consequent conclusions according to the results obtained.

1.2. NEUROPSYCHOLOGY: WHAT IS IT ABOUT?

According to Gich (n.d.) neuropsychology is the science that studies cognitive, behavioral and emotional alterations caused by brain injuries, whether focal or diffuse, stable or degenerative. According to Portellano (2011) it falls within the field of neuroscience, unifying the knowledge of neurobiological and psychobiological processes combining the study of the brain and higher mental activity.

Depending on the field of work, this can have several objectives: within clinical practice there is the diagnostic objective -through neuropsychological explorations- and the therapeutic objective -through cognitive rehabilitation-. Within the field of research are the scientific objectives -as is the case of this work- and finally within the field of teaching are the teaching tasks.

Regarding the research aspect, which is the case that occupies this work, these scientific objectives are translated into the generation of protocols and research projects, and in the publication of results. In this case it is a research project carried out and with a possible final publication of the results obtained.

Among the disorders that neuropsychology deals with, in neurological / neurosurgical pathology there is a large list: cerebral vascular pathologies (ischiemic and haemorrhagic stroke, cerebral aneurysms, cerebral arteriovenous malformations, etc.), extrapyramidal pathologies (such as Parkinson's disease and similar), neurodegenerative pathologies, infectious pathologies, brain tumors and traumatic brain injuries (Gich. n.d.). Then, within all this, neuropsychology is concerned with studying all kinds of disorders such as language (aphasia), gestures (apraxias), reading and writing (dyslexia and dysgraphia), recognition disorders (agnosia) and, among others, memory disorders (amnesias).

AD, being a neurodegenerative disease, could be found, consequently due to its symptoms, in several of these groups (disorders of memory, language, gestures and gnosics). In the case of this work, it will be approached from a global perspective as a neurodegenerative disease, paying special attention to the part of mnesic and gnosic symptoms.

2. THEORETICAL FRAMEWORK

2.1. MILD COGNITIVE IMPAIRMENT: BEFORE THE AD

Mild cognitive impairment (MCI) is widely recognized as a public health problem, as it is associated with the development of AD (Quintana, 2009). The concept of mild cognitive impairment has been proposed as a clinical situation involving elderly people with cognitive and functional impairments (Petersen, 1999; Quintana, 2009), in this way, an attempt is made to identify subjects with objective abnormal cognitive loss, beyond those of healthy elderly people (Quintana, 2009).

The first approach to this concept dates from 1962 defined as "benign forgetfulness of aging" by Kral, through "age-related memory impairment" in 1986 by Crook et al., to 89 to "age-compatible memory impairment" by Blackford and La Rue, later in 1994 defined as "age-related cognitive impairment" by Levy, and in the same year in DSM-IV it is introduced as "mild neurocognitive impairment" understood as an alteration of two or more cognitive areas, one of which must be memory. Thus, later in 1997, in the Canadian Study of Health and Aging by Graham et al., the term "cognitive impairment-non-dementia" was developed to describe all these subjects who present cognitive alterations in language, visuospatial abilities and attention, but without reaching the diagnosis of dementia.

Finally, the most popular and used term is the one initially proposed by Petersen et al. (1999) of "mild cognitive impairment", specifically defined in 2003 by the same author as "a state between normal cognition and dementia, characterized by a deficit that is not explicable by age, educational level or medical diseases".

In 1982, two global scales of dementia were published: Hughes' CDR (*Clinical Dementia Rating*) and Reisberg's GDS (*Global Derterioration Scale*). These were intended to be able to classify subjects who are at some point in the continuum that is the threshold of dementia (from normality to the most advanced stages of it). Mild cognitive impairment is identified with a clinical stage of the GDS scale (Reisberg et al., 2008; Quintana, 2009), but a score of 0.5 in the CDR corresponds to "questionable dementia" or "very mild impairment". However, these tools are not used as diagnostic instruments, but as a stratification of the severity of possible cognitive impairment; a patient with GDS3 or CDR 0.5 can meet both criteria for MCI, mild dementia or AD (Quintana, 2009).

There are different types of MCI diagnostic criteria, but all of them have in common that patients with MCI present an objectifiable cognitive impairment and the absence of dementia (Quintana, 2009). All of them are included in table 1 of the annexes.

Thus, according to Quintana (2009) the International Group on Mild Cognitive Impairment (Winblad et al., 2004; Quintana, 2009) proposed a series of recommendations in the general criteria:

- ➢ The patient is not cognitively normal or has dementia
- ➢ There is an obvious cognitive impairment, referred by the patient and/or an informant, together with a deficit in objective tasks and/or impairment in neuropsychological tests
- ➢ The activities of daily living are globally preserved with minimal alterations

These criteria, therefore, highlight the importance of clinical evaluation with a good neuropsychological examination and personal and family anamnesis.

In terms of MCI prevalence, epidemiological and longitudinal studies place MCI at between 3 and 42%, depending on the criteria used (Busse et al., 2006; Ganguli et al., 2004; López et al., 2003; Ritchie, 2004; Quintana, 2009). According to the criteria of the *May Clinics* it is 12-18% in people aged 65 years or older (Petersen, 2007; Quintana, 2009), and the incidence stands at 8-58 per thousand per year (Ritchie, 2004; Quintana 2009).

Finally, after the definition of MCI, the concept of "prodromal Alzheimer's" appeared with the following diagnostic criteria: According to Quintana (2009) 1) Memory complaints referring to the patient or family member. 2) Progressive evolution. 3) Normality or slight alteration in the advanced activities of daily life. 4) Amnesiac syndrome of hippocampal type defined by: poor free recall despite adequate and controlled coding, decrease in total memory due to insufficient input effect or recognition deficit; numerous intrusions. 5) Persistence of

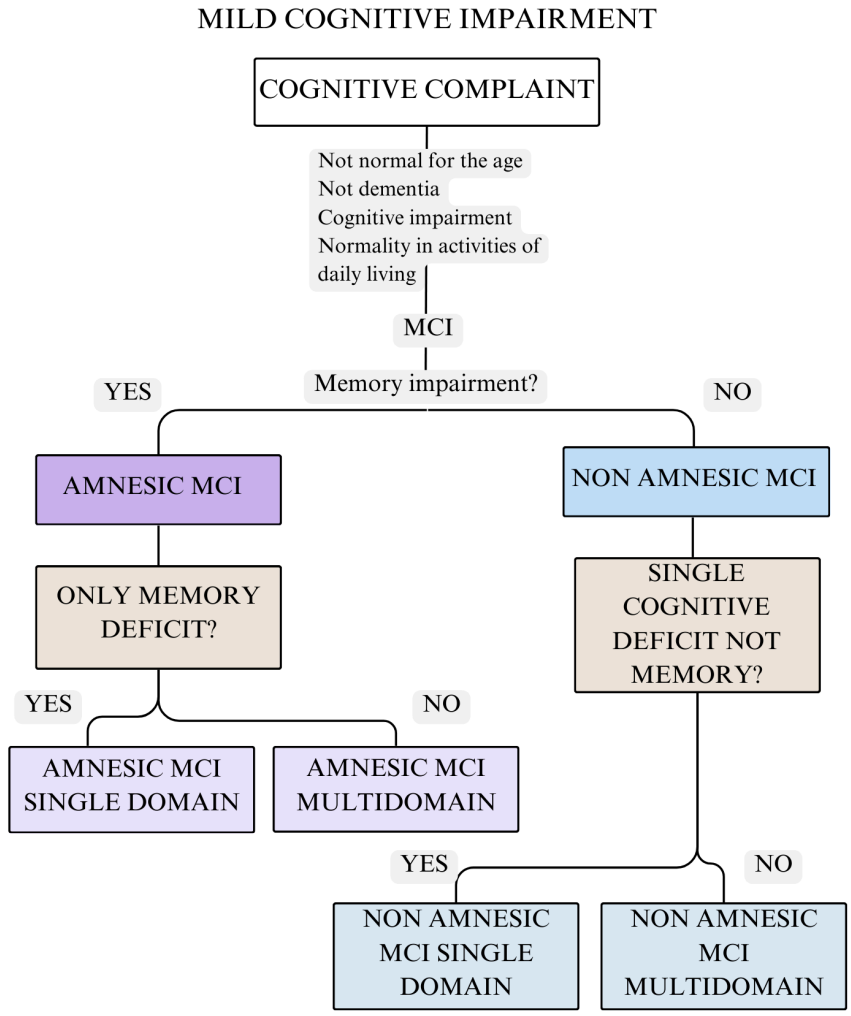
mnesic deficits in subsequent evaluations. 6) Absence of dementia. 7) Exclusion of any alteration that may be the cause of MCI, such as an adequate diagnosis through neuropsychological tests, neuroimaging and biomarkers.

2.1.1. SUBTYPES OF MCI:

Four subtypes of MCI have been described: amnesic MCI, non-amnesic MCI, amnesic MCI affecting multiple areas, and non-amnesic MCI affecting multiple areas (or multiple domain) (Petersen, 2001a, 2004; Quintana, 2009). The formulations to reach each of them are presented in Figure 1.

Figure 1

MCI type circuit



Note. Adapted from *Mild Cognitive Impairment,* by Petersen (2004); Quintana (2009).

➢ Amnesic MCI:

According to Petersen et al. (1999), cited by Quintana (2009), subjects report memory losses -preferably corroborated by an informant- and this deficit is objectified by a neuropsychological evaluation (memory at 1.5 typical deviations below normal). The rest of the cognitive functions are preserved and do not present alterations in the activities of daily living (ADL).

➢ Amnesic MCI multidomain:

The subject may have impairment in various cognitive functions in addition to amnesic deficiency, and it has been considered that this can evolve into DTA (Alzheimer's-type dementia).

➢ Non-amnesic MCI:

The cognitive function that is altered is usually language or executive functions. Thus, there are as many types of non-amnesic MCI as there are cognitive functions (other than memory): language MCI, executive MCI, visuoperceptive-MCI and praxic-MCI, fundamentally (Quintana, 2009).

➢ Multidomain non-amnesic MCI:

Patients present at least two altered cognitive functions, none of them being memory (Sánchez-Valle, 2007; Quintana, 2009).

2.1.2. CLINICAL MANIFESTATIONS OF MCI:

Usually episodic memory impairment is the typical sign of amnesic MCI and early AD (Quintana, 2009). However, as has been explained, patients with MCI sometimes present not only alterations in memory (in the case of the amnesic type) but also in other cognitive functions (Quintana, 2009): verbal fluency, language and processing speed, verbal comprehension, semantic memory, attention, executive functions and visuospatial and visuoperceptive skills. Even so, in the case of the visuospatial deficit, it has been seen that the performance deficit is only in especially complex tasks, but outside of these performance is within normality.

In addition to cognitive alterations, MCI can also lead to neuropsychiatric symptoms (Hwang et al., 2004; Quintana, 2009) appearing in 35-75% of patients (Apostolova & Cummings, 2008; Palmer et al., 2007; Quintana 2009). Non-psychotic symptoms such as apathy, depression, anxiety, agitation, and irritability are usually the most common (Apostolova & Cummings, 2008; Rozzini et al., 2008; Quintana 2009).

2.1.3. NEUROPATHOLOGY OF MCI:

Studies of pathological anatomy have shown that patients with MCI present alterations typical of AD, although insufficient to constitute the neuropathological diagnosis of this disease (Petersen, 2009; Quintana 2009). The greatest neuropathological change in MCI is the number of neurofibrillary tangles in the neocortex and mesial-temporal structures (Bennett et al., 2005; Hof et al., 1996; Markesbery, 2009; Mitchell et al., 2002; Morris et al., 2001; Petersen et al., 2006; Quintana, 2009).

Authors such as Jicha et al. (2006) (cited by Quintana, 2009) defined the neuropathological substrate of MCI-amnesia as pathological abnormalities in the medial temporal lobe, in most cases neurofibrillary degeneration. When compared with the neuropathological substrate of AD, differences are observed in the number of neurofibrillary tangles in the amygdala and subiculum (Markesbery et al., 2006; Quintana, 2009).

There are some studies on the synaptic and neurotransmitter changes that occur in MCIs; Mufson et al. (2000) (cited by Quintana, 2009) showed a cholinergic deficit in MCI-amnesic patients with neuronal loss in the baseline Meynert nucleus, and more recently it has also been seen that patients with MCI present synaptic loss of hippocampal neurons compared to control subjects (Scheff et al., 2007; Quintana, 2009).

In addition, studies with structural neuroimaging have shown that patients with MCI-amnesia present selective atrophy of the medial temporal lobes, especially the entorhinal cortex and hippocampus, although the degree of atrophy of these structures is lower than in patients with initial AD (Appel et al., 2009; Duara et al., 2008; Jack et al., 2005; Quintana, 2009). Finally, functional neuroimaging currently -especially in all health advances in the identification of biomarkers- is being widely used in the study of MCI; studies with PET scans have shown hypometabolism in temporoman-parietal areas and in the posterior cingulate cortex (Chételat et al., 2005; Clerici et al., 2009; Dickerson et al. 2005; Drzezga et al., 2003; Landau et al., 2009; Quintana, 2009), and in studies with PET using the B-Pittsburg component (GDP), binding PET to detect amylode plaque deposits in vivo, distributions of such a compound were found in both the frontal cortex and the anterior and posterior cingulate (Fripp et al., 2008; Kemppainen et al., 2007; Okello et al., 2009; Quintana, 2009).

2.1.4. RATES AND PREDICTORS OF CONVERSION BY AD:

Patients with MCI have a high risk of developing dementia (Busse et al. 2006; Manly et al., 2008; Morris et al., 2001; Tschanz et al., 2006; Quintana, 2009); usually MA.

Even so, of all cases of MCI, in relation to how to know which ones would evolve towards AD, there is a series of evidences of progression predictors, which must be taken into account in the diagnosis and follow-up of patients. According to Quintana (2009):

- ➢ Age: has been shown to be a risk factor for transition between MCI and AD (Amieva et al., 2004; Tyas et al., 2007; Quintana, 2009)
- ➢ Neuropsychological disorders: several studies (Chen et al., 2000; Fleisher et al., 2007; Ritchie et al., 2001; Quintana, 2009) have shown that presenting deficits in executive functions and deferred recall indicate a high risk of progression to dementia, hence the importance of an exhaustive neuropsychological examination aimed at detecting these predictive variables as soon as possible. Following this line, Bennet et al. (2002) (cited by Quintana, 2009) suggest that measurements of episodic memory, semantic memory and processing speed should be incorporated into the neuropsychological assessment of MCI, as they are associated with the development of AD. When patients, in addition to memory deficit, present another cognitive alteration, the risk of developing AD increases (Tabert et al., 2006; Quintana, 2009).
- ➢ Memory deficit: all MCI subtypes with memory deficits are more vulnerable to AD
- ➢ Atrophy in medial temporal structures (DeCarli et al., 2007; Jack et al., 2008; Visser, Verhey, Hofman, Scheltens & Jolles, 2002; Quintana, 2009): this, characterized by loss of hippocampal volume (Apostolova et al., 2006; Erten-Lyons et al., 2006; Quintana, 2009) predicts the progression towards MA.

2.2. ALZHEIMER'S DISEASE

2.2.1. CONCEPT:

Alzheimer's disease (AD) is a progressive neurodegenerative disease, with distinctive clinical and neuropathological characteristics characterized by the abnormal deposit in the brain of two proteins: the β Amyloid protein related to Amyloid plaques and the Tau protein, related to the so-called neurofibrillary change or tangle (Llibre-Rodríguez et al., 2022).

It is a syndrome of a chronic nature characterized by a deterioration in cognitive function that affects memory, thinking, orientation, understanding, calculation, learning capacity and judgment, all in the absence of alteration of consciousness (Palenzuela, 2018). This deterioration is also usually accompanied by a deterioration in emotional control, social behaviour and motivation (WHO, 2017; Palenzuela, 2018).

There are different types of dementia according to their etiology, but by far, AD-type dementia is the most common worldwide.

2.2.2. EPIDEMIOLOGY:

According to the *Guía oficial de práctica clínica en demencia* (2018), Alzheimer's disease (AD) is the leading cause of neurodegenerative dementia worldwide and is a major health problem. In addition, its prevalence is expected to increase in the coming years.

Epidemiological studies allow estimating the prevalence and incidence of AD, but there are several factors that make it difficult to carry out and/or interpret epidemiological studies: the inaccuracy of the clinical diagnosis of dementia type AD (up to 20% error), the difficulty in diagnosing in the phase of mild cognitive impairment (MCI onwards) that requires the use of biomarkers with little diffusion, among others (*Guía oficial de práctica clínica en demencia*, 2018). That is why there is some variability between the reported prevalence and incidence figures, but thanks precisely to these results it has made it possible to estimate the prevalence and incidence of dementia due to AD.

Regarding **prevalence**, according to the studies included in the systematic review by Takizawa et al. (2015) selected according to the PRISMA declaration which includes articles published between 2002 and 2012 with epidemiological data on AD dementia in Europe and the USA, it is shown that in Spain a prevalence of between 2.8 and 3.9% has been reported in the 75-79 age range. and between 7.2 and 34.1% in people over 85 years of age (*Guía oficial de práctica clínica en demencia*, 2018). Even so, this should be much higher since in all these studies only type AD dementia is usually analyzed but forgetting to study the initial phases of MCI (or prodromal AD if it has been objectified with biomarkers that it is AD), so its prevalence is unknown while we know that 15% of the population over 65 years of age has MCI and that 50% of these are due to AD (*Guía oficial de práctica clínica en demencia*, 2018). In addition to this, it is known that AD is more frequent in biological women (prevalence of 7.1% in women and 3.3% in men), since they have twice the risk of developing this type of MCI because thanks to studies such as the ADAPTED project of Fundació ACE it has been observed that the APOE4 allele of the APOE protein, The main risk factor for AD, poses an increased risk for the development of AD in women, and added to the hormonal changes associated with the female sex this seems to be decisive.

Regarding the **incidence**, in the same studies mentioned in the prevalence, in Spain cases of up to 4.3 people out of every 1000 inhabitants-year have been found in type AD dementia between 75 and 79 years.

2.2.3. SOCIAL AND HEALTH COSTS

AD has a high socio-sanitary cost at the national level and this cost is expected to skyrocket in the coming years as a result of the increase in the prevalence of the disease (*Guía oficial de práctica clínica en demencia*, 2018). As López-Pousa (2004) points out, as a result of the increase in life expectancy, the number of AD cases has also grown in recent decades, thus increasing the entire cost allocated to the social and health resources necessary to meet the needs of patients and their families.

As explained in the GERAS studies of Wimo et al. According to the study by López-Pousa et al. (2004), there is unanimity in all published studies about the considerable increase in costs as the severity of the disease increases, both in developed and developing countries (*Guía oficial de práctica clínica en demencia*, 2018).

To understand what is talked about when social and health costs are mentioned, the *Guía oficial de práctica clínica en demencia* (2018) describes them in non-institutionalized patients, dividing them into four groups:

- ➢ Costs directly related to the patient's health: medications, hospital nights, visits to emergency departments and outpatient visits
- ➢ Costs related to the patient's social environment: housing, community care, structural adaptations of the home, consumables and perceived financial support
- ➢ Costs related to the health of the caregiver: medications because they are caregivers, hospital nights, visits to emergency departments and outpatient consultations
- ➢ Costs related to the socio-occupational impact of the disease on the caregiver: time costs and loss of working hours

According to the *Guía oficial de práctica clínica en demencia* (2018), based on the report of Alzheimer's Disease International of 2015, the estimated total global cost of dementia amounts to 818 billion US dollars. In Spain, it is estimated that the average cost per patient per year ranges between €17,100 and €28,200. In addition, it is known that the social and health costs attributable to AD increase in parallel with functional and cognitive impairment, comorbidity and the presence of neuropsychiatric symptoms of patients, being maximum at the time of institutionalization. On an annual average, due to this cognitive worsening, the

cost increases by an average of €14,956 in mild, €25,562 in moderate cases and €41,669 in severe cases. Most of these costs are financed by the family, so this adds to the own suffering caused by the disease. Taking into account all levels of severity, it is estimated that the total cost in Spain of AD treatment in patients over 65 years of age is around 10,000 million euros per year, which represents 1.5% of the national gross domestic product (*Guía oficial de práctica clínica en demencia*, 2018).

2.2.4. RISK FACTORS

According to the *Guía oficial de práctica clínica en demencia* (2018), risk factors are considered to be those situations or conditions that increase the probability of developing the disease and in the case of AD clearly established factors are considered to be age (aging), sex (female) and genetic factors (APOE haplotype). In addition, vascular risk factors (middle-aged arterial hypertension, dyslipidemia, obesity, sedentary lifestyle, smoking, diabetes mellitus) individually or in groups, a history of head trauma and depression in late stages are also considered.

Apart from this, it is also necessary to consider according to the *Guía oficial de práctica clínica en demencia* (2018) that "a low and unstimulating educational level, from the cognitive point of view, would constitute a risk factor for the disease, probably linked to the advancement of the clinical expression of the disease by having a lower cognitive reserve".

On the other hand, according to a study by Suárez-Armiento et al. (2023) on the risk factors for AD and its etiology, among the main results to highlight were age in 30%, heart disease in 29%, diabetes in 20%, 17% accompanied by diseases of cognitive impairment and depression and 18% for lung diseases.

2.2.5. DIAGNOSTIC CRITERIA FOR ALZHEIMER'S DISEASE

Initially, the most commonly used diagnostic criteria for the diagnosis of Alzheimer's-type dementia (DTA) were the **NINCDS-ADRDA** criteria since 1984, with a sensitivity of 81%, but its specificity decreased to 70% and in case of including cases of possible DTA it decreased to 48%.

In addition, according to the *Guía oficial de práctica clínica en demencia* (2018), with the growing knowledge of the biological bases of the disease and the development of biomarkers, different limitations of these criteria were highlighted:

- ➢ The histopathology of the disease can be found in a wide clinical spectrum (normal [MCI] dementia).

- ➢ The absence of knowledge, at that time, of data that distinguishes the disease from other dementias.
- ➢ The non-inclusion of PET and CSF results.
- ➢ Proof that many non-amnesiac presentations are also DTA.
- ➢ Absence of information about the genetics of the disease.
- ➢ The presence of DTA both under 40 years of age and above 90 years.
- ➢ That the "possible DTA" had shown itself to be a very heterogeneous entity.
- ➢ Finally, the difficulty in differentiating the transition between MCI and dementia.

Thus, in **2007**, in order to address these limitations, the *International Working Group for new research criteria for the diagnosis of AD* (IWG) published new criteria (they will be referred to as the **IWG-1**). These focus on a clinical core of early and significant deterioration of episodic memory, which must be accompanied by at least one or more abnormal biomarkers in structural neuroimaging explorations such as magnetic resonance imaging (MRI), molecular neuroimaging explorations with PET, or the analysis of β amyloid and tau proteins in the CSF (*Guía oficial de práctica clínica en demencia*, 2018). In addition, in these updated criteria, the concepts of preclinical and prodromal AD were used for the first time.

Later, in **2011,** the *National Institute of Aging* and the *Alzheimer's Association* (**NIA-AA**) published a new update of the diagnostic criteria (see table 2 of the annexes) for Alzheimer's dementia (AD), MCI, MCI due to AD, preclinical staging of the disease and made a classification of AD biomarkers, as well as the criteria for their use (*Official guide to clinical practice in dementia*, 2018).

Finally, in **2014** the IWG published new criteria (**IWG-2**) (see table 3 of the annexes) taking into account the differences in approach, terminology and use of cognitive markers and biomarkers between the IWG-1 and NIA-AA criteria, so they proposed advances to improve the diagnosis of AD taking into account the limitations of its previous criteria (IWG-1).
Table 4 of the appendices shows the comparison between the different diagnostic criteria mentioned.

2.2.6. AD BIOMARKERS

In the framework of the NIA-AA research for the definition and staging of AD, Clifford et al. (2018) highlight and put the biomarker method on the table, establishing that AD is defined by the underlying pathological processes documented by them, rather than by the clinical consequences. Thus, the committee proposes the grouping of these into 3 groups: biomarkers

of amyloid beta, pathological fibrillar tau and markers of neurodegeneration or neuronal injury.

Thus, according to Clifford et al. (2018) the definition of AD refers to plaques of amyloid beta and pathological tau deposits, defined *in vivo* by abnormal biomarkers of pathological amyloid beta and tau (both are mandatory).

2.2.7. THE PROFESSIONAL OF PSYCHOLOGY IN AD

Psychology professionals play a very important and diverse role in working with dementia or, specifically, in Alzheimer's disease, which is the pathology with the greatest impact among the elderly population, both from a clinical branch, as well as psychosocial intervention, as well as research.

Psychological intervention provides an important value both at the neuropsychological level for the patient, in the clinical and psychosocial field with the work with caregivers, and at the level of advice and training of centers and institutions, tasks that are being especially valued from the platform of associations of affected relatives (Ruiz-Adame, 2000).

Despite the heterogeneity that the course of this disease can have in each individual case, it is certainly often described easily and quickly as a process of functional involution (like the cognitive and learning evolution of children, but vice versa). That is why it is really a very widespread field that generates the disease to intervene by psychology professionals: thus, the best known field -and with greater social recognition among those affected- is that of psychostimulation and neuropsychostimulation. In addition, also precisely because of this progressive -and chronic- cognitive impairment, the figure of the caregiver (main) is necessary, and therefore the figure of the psychologist is usual in nursing homes where profiles of this type are found, to carry out follow-up and care that at a psychological level is also appropriate.

On the other hand, below are detailed in a more specific way some of the specific functions that the professional of psychology in AD can have:

- Evaluation and diagnosis function:

In this case, from the clinical branch, the professional has the task of carrying out neuropsychological evaluations on the cognitive state of the patient in order to, together with the rest of the members that make up an interdisciplinary team (neurologists, nurses, etc.) in order to diagnose it (or, in any case, follow up if it is not the first evaluation that is made).

These functions can be performed by psychogeriatric units, day units or memory assessment and dementia units (Serra-Mestre et al., 1997, cited by Ruiz-Adame, 2000) in a work, as mentioned, usually multidisciplinary, although in this aspect, in Spain there is a wide diversity of experiences, covering situations such as those that occur in Catalonia where the figure of the psychologist has a greater integration into these teams, and in these functions consequently, until the case of Andalusia, where there is no public dementia unit that has a psychologist hired by the Administration, despite the demand supported by the rest of the professionals (Ruiz-Adame, 2000).

- Intervention function:

In addition to the evaluation function described above, psychologists can also work by intervening with patients: they will do so through cognitive-behavioural psychostimulation programmes, from psychogeriatric day units, dementia units, inpatient centres or home help services (Ruiz-Adame, 2000). Also in this line, the psychologist is also competent, in collaboration with the management of the center or unit, to contribute their knowledge in order to establish the plan of activities of the same, or on its rules of operation (Ruiz-Adame, 2000).

The importance of this task lies in the fact that Alzheimer's patients will tend to apathy and isolation, which is known to facilitate the progression of their deterioration, so it is a question of establishing programs that break this dynamic, stimulate the best preserved faculties and slow down prognosis (Kalish, 1982; Hernando, Pereda, Martín, Mauri, 1988; Tárraga, 1994, quoted by Ruiz-Adame, 2000).

Likewise, this intervention function is not limited only to working with the patient but also involves caring for the caregiver both at an individual psychotherapeutic level and group therapies, and even working on support tasks for the rest of the team of professionals to avoid the well-known *Burn-out* (Ruiz-Adame, 2000).

- Advisory function:

Similarly, according to Ruiz-Adame (2000), another function that can be performed is to provide guidance with the patient's relatives at different times during the course of the disease: advising during the diagnostic process, informing about prognosis, prophylactic measures, giving guidelines for action against behavioural, emotional and cognitive stimulation alterations to compensate, as well as referring to other social and health support networks -multidisciplinary work- and/or mutual aid organizations.

- Training and teaching function:

The teams involved in AD need a continuous flow of information and updating of perspectives that always allow us to improve the diagnosis and treatment of patients and their environment. In this way, the psychologist must participate in training activities both internally -elaboration of protocols, clinical sessions, review of cases ...- and externally exchanging knowledge with other professionals of psychology, members of other disciplines and affected (seminars, congresses, courses ...), in order to allow the continuity of these strategies.

- Resource Management Role:

According to Ruiz-Adame (2000), psychologists can also intervene as activity coordinators, directing evaluation, assessment and treatment programmes, training programmes, or care centres for Alzheimer's patients and/or caregivers. As part of this function, the psychologist is perfectly qualified to be in charge of controlling the material resources necessary for the correct performance of the planned activity plans and being the one who coordinates the human team, regardless of whether each of the members of the team has their own areas of competence depending on their specialty. Experience and suitability.

- **Research function:**

Research in AD (or dementia in general), clinical trials, everything related to research is a very important task in order to advance in medicine to be able to treat the diseases that grip such a high percentage of our population. Thanks to research and trials, new drugs can be found and tested that aim to fight, in this case, against AD. According to the *Registro Español de Estudios Clínicos* (2024), dementia is a rapidly growing public health problem that has a significant global socioeconomic impact. Clinically, the continuous process of progression of the disease from mild cognitive impairment (MCI) to mild, moderate and severe dementia is accompanied by a gradual loss of cognitive function and increasing difficulties in performing daily activities. Therefore, therapeutic interventions aimed at the early stages of cognitive impairment could have considerable clinical and economic consequences for patients, their caregivers and society. There are currently no treatments available for MCI and there are only symptomatic treatments for Alzheimer's-type dementia as mentioned, which is why research, intervention and clinical practice are extremely important to treat this health problem so evident in society that covers the entire field of neurodegenerative diseases (whether we talk about MCIs, dementias, AD specifically, etc.).

This study is contemplated within this framework of research in psychology.

2.2.8. EVALUATION TOOLS

The scales currently used to evaluate AD and its diagnosis are explained below, according to the NACC (*National Alzheimer's Coordinating Cente*r), specifically the UDS battery (*Uniform Data Set*) validated and used worldwide by NACC to perform joint AD evolution data.

NACC (National Alzheimer's Coordinating Center):

This center was born in 1999. In 1984, the US National Institutes of Health (NIH) created the Alzheimer's Disease Centers (ADC), to improve diagnostic methods and identify new treatment options (Porto et al., 2022). In order to unify these data produced by the different ADCs, a registry of the disease was established through a consortium that developed a battery of neuropsychological tests to detect cognitive impairment associated with AD, but over time it showed deficiencies for the diagnosis of mild cognitive impairment (MCI) (Porto et al., 2022). Thus, in 1997 a more comprehensive protocol was developed but the database proved to be limited in terms of information derived from neuropsychological evaluations (Morris et al., 2006; Porto et al., 2022) so finally in 1999 the NACC was born to evaluate the neuropathological changes in the brain found in the autopsies of individuals diagnosed with AD in the different ADCs (Morris et al., 2006; Porto et al., 2022). In this way, a uniform profile (medical, cognitive, behavioural and functional) can be obtained that allows further clinical studies with patients from these centres (Cronin-Stubbs et al., 2000; Porto et al., 2022).

Later the UDS was established with more detailed information about patients.

UDS (Uniform Data Set):

According to Porto et al. (2022) the UDS (version 3, UDS 3.0) contains complete information about people with AD and provides greater diagnostic accuracy; This battery of tests provides measurements of attention, information processing speed, executive functions, episodic memory, and language. The UDS makes it possible to detect differences between clinically normal people and people with dementia, therefore it favors early diagnosis. In addition, although initially designed for English speakers, it was also translated into Spanish and has been studied in the Spanish-speaking population residing in the US (Benson et al., 2014; Porto et al., 2022).

According to Porto et al. (2022) this battery of tests includes:

-The MoCa (*Montreal Cognitive Assessment*): created in 2005 by Nasreddine and colleagues, as an instrument to detect MCI and AD (Palau et al., 2018)

-The *Craft Story* 21: immediate and delayed recall: one of the many variants to evaluate verbal memory.

-The *Benson Complex Figure*: copy and memory: created by Frank Benson, a simplified version of the complex Rey-Osterrieth figure to more purely assess visuospatial characteristics (Sisakhti et al., 2024)

-*Number span test*: forward (direct) and backward (inverse): developed by Arthur Benton, the classic working memory test (Blackburn & Benton, 1957) for repetition of numbers in direct and inverse order

-Categorical fluency: animals and plants: one of many different types of tests to evaluate semantic verbal fluency, for example -in this case- using the categories of animals and plants

- The *Trail Making Test* (TMT) part A and part B: developed by Ralph M. Reitan (1971) to assess attention and executive functions

-The *Multilingual Naming Test* (MINT): developed by Gollan et al. (2012), a drawing/image naming test to assess language deficits (Ivanova et al., 2013)

-The *Verbal fluency phonemic test*: letters P and M: one of the variants of the phonemic fluency test (in this case, words beginning with the letter P and M)

2.3. VISUAL PERCEPTION

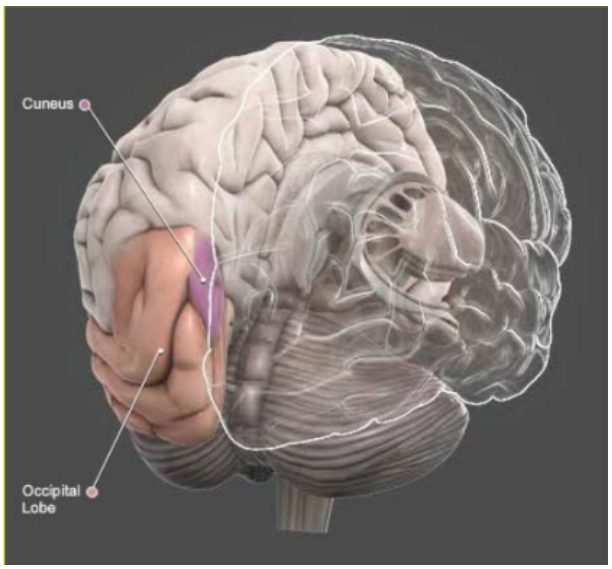
2.3.1. NEUROBIOLOGICAL AND NEUROPSYCHOLOGICAL BASES: BRIEF BRAIN BIOLOGICAL REVIEW

2.3.1.1. THE OCCIPITAL LOBE: NEUROANATOMICAL BASES:

The occipital lobe is one of the 4 lobes that make up our brain; This is located in the posterior area of the brain, behind the parietal and temporal lobes, as shown in Figures 2 and 3.

Figure 2

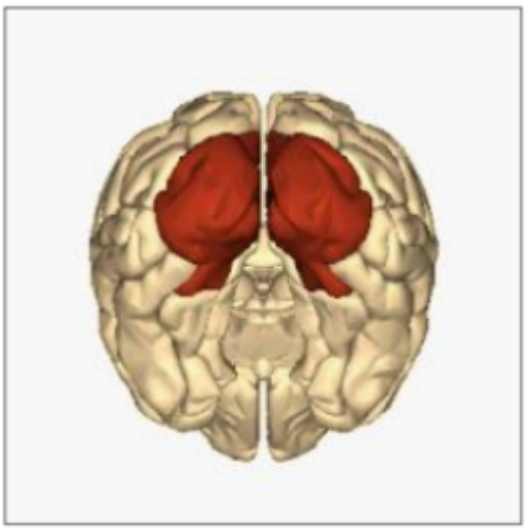
Occipital lobe



Note. Adapted from Caño, 2020.

Figure 3

Occipital lobes



Note. Adapted from Caño, 2020.

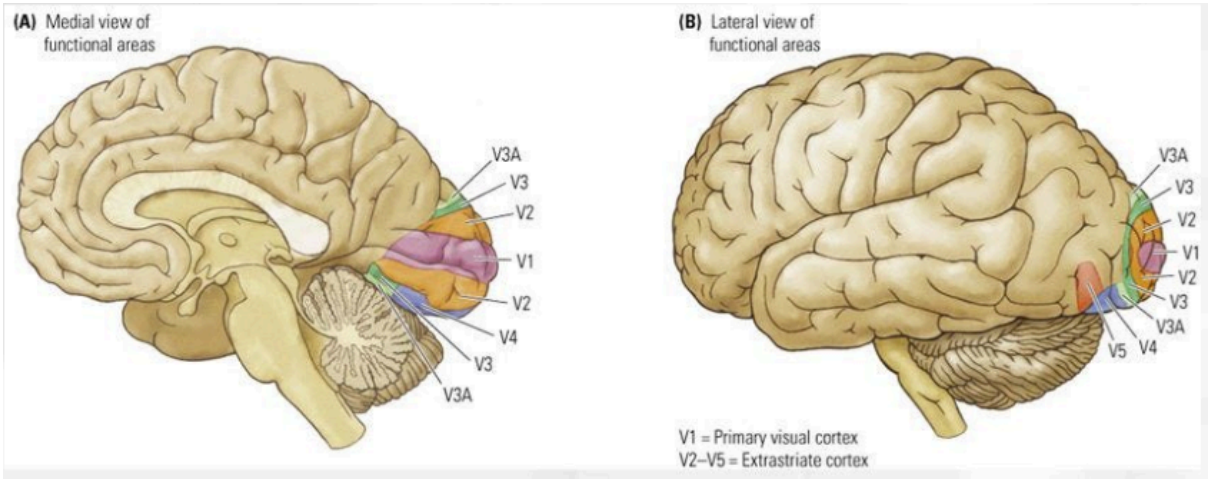
The main function of this lobe is related to all visual processing. All stimuli reach this cortex and the information begins to be analyzed in such a way that later, depending on what is done with it (interpret it, discriminate ...), it is sent to different pathways and cortices.

The visual cortex (Figure 4), which makes up this lobe, is made up of the striated area (corresponding to the primary visual area or V1) and the extra-striated area (corresponding to

areas V2, V3, V4 and V5). As the stimulus is processed, it passes from one area to another as a result.

Figure 4

Visual cortex of the occipital lobe



Note. Adapted from Caño, 2020.

➢ Primary visual area (Broadmann area 17):

It is the region that receives input from images from the retina of the eye. This area is where our brain interprets colour along with other important aspects of vision and is essential for the conscious processing of visual stimuli (Caño, 2020).

This region receives stimuli and separates them to process shape, color and movement and projects them towards the other visual areas (V2-V5).

➢ Secondary visual area (Broadmann area 18):

This is made up of two distinct regions: the prestriated cortex and the inferotemporal cortex. The prestriad is located around V1 and receives afferences from this and other cortical areas, as well as from the thalamus. Its functionality has been related to memory and association with past visual experiences (Caño, 2020). As for the inferotemporal cortex, it is located in the lower part of the temporal lobe and lesions in this area produce agnosia (lack of recognition).

In these first two visual areas, visual information of different types is gathered and then from there it is sent to the 3 remaining visual areas (V3-V5) which are more specialized.

➢ Tertiary visual area (Broadmann area 19) or V3, V4 and V5:

According to Caño (2020):

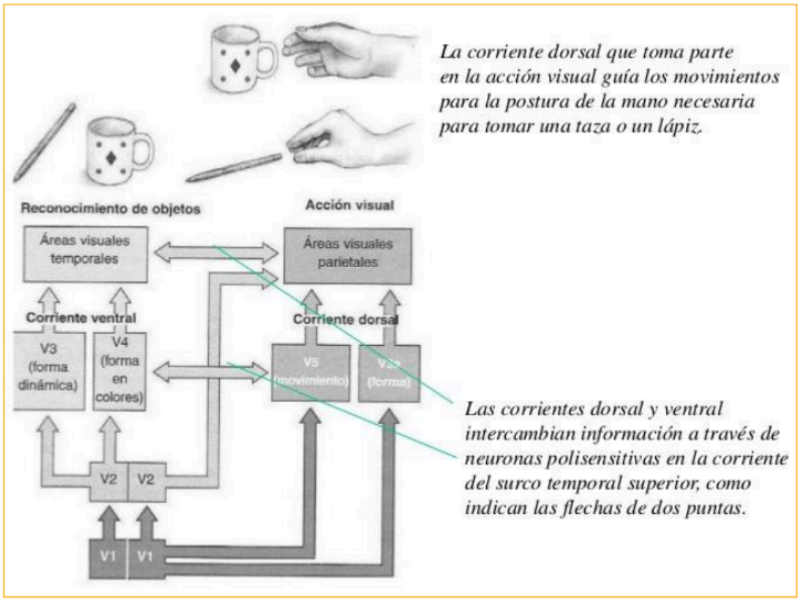
- V3: linked to dynamic form (shape of moving objects)
- V4: participates in the analysis of the colour and shape of visual stimuli
- V5: participates in the perception of the movement of objects regardless of their shape (temporoparietal region)

Once the information has passed through V1, then this torrent of data will fork following two different routes (see figures 5 and 6): the ventral pathway (temporal lobe direction) and the dorsal pathway (parietal lobe direction).

The dorsal pathway is given by the connection between V1 and V2 with the V3 zone giving this occipitoparietal pathway. Then, through the visual cortex V3 and V5 the information processed by V1 will reach the parietal lobe; It is believed that this area of visual processing is responsible for establishing the location characteristics of the movement of what is seen - it exercises a function of visual guide of it; This is why it is also called the "where" and "how" pathways.

Figure 5

Representation of action of the dorsal and ventral pathways

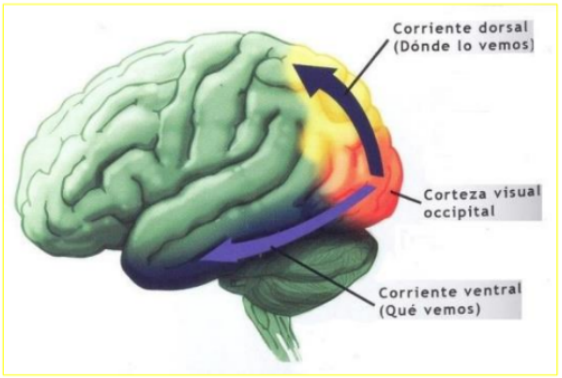


Note. Adapted from Caño, 2020.

On the other hand, the connection between V1 and V2 with V4 is what gives rise to the occipitotemporal pathway (ventral route). This pathway is responsible for the content of vision (that's why it is also called the "what" pathway), it processes the characteristics of the isolated elements that are being seen at any given time, therefore it is linked to the perception of objects -including color-.

Figure 6

Representation of dorsal and ventral current



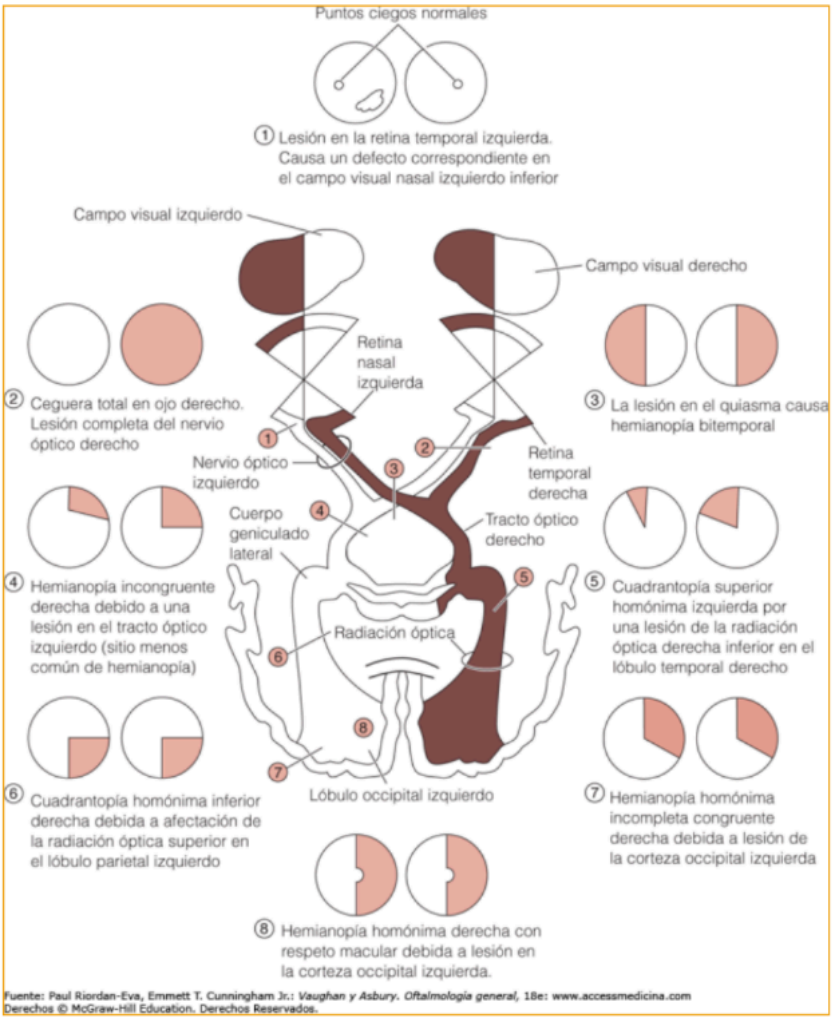
Note. Adapted from Caño, 2020.

2.3.1.2. OCCIPITAL LOBE PATHOLOGY:

- ➢ Primary visual area (V1): A lesion in one of the hemispheres in this cortex causes the appearance of scotomas in the contralateral visual field to the lesion. In the case of bilateral V1 lesions, there is talk of visual cortical blindness, in which the person will not see anything but at a given moment they can, for example, dodge an object because the thalamus has been able to carry out some small processing. Figure 7 shows examples and possibilities of injuries in this part.

Figure 7

Types of visual pathologies related to the visual system and occipital lobe



Note. Adapted from Caño, 2020.

➢ Visual area of association:

This area is composed of two regions: Broadmann area 18 and 19 (surrounding V1 in the occipital lobe) and Broadmann areas 20, 21 and 37 (inferotemporal visual area, which make up a large part of the temporal lobe). The main function of the cortex is to relate the current visual experience to the past, so that it allows us to recognize the meaning of what is seen and identify its meaning. In fact, electrical stimulation in this cortex causes hallucinations of very vivid past scenes, so this seems to indicate its role in storing or remembering visual experiences.

In case of injury in this area, it can occur: loss of sensation of movement, shape, color (injury in areas 18, 19 and 37 causes achromatopsia), spatial orientation, depth perception ..., and also complex perceptual defects -but with preservation of elementary perception- such as

metamorphopsia (ripples of straight lines are perceived), illusions (distorted perceptions) or hallucinations (visual images with no external stimulus).

Cortex injuries cause visual agnosia, or difficulties in interpreting stimuli. If this occurs for example in the medial part of the inferior temporal lobe, it causes prosopagnosia, a disorder that produces a total inability to recognize faces.

Lower bilateral lesions, according to Caño (2020) can lead to: deficits in visual imagination, spatial disorientation and simultaneous disorientation.

➢ Dorsal route:

Affectations in this pathway will give rise to problems in various processing carried out in these areas: position of the object, at what speed it moves, at what distance the observer is, etc. However, their activity does not require conscious activity, enabling a large number of automatic actions, or reflexes, such as dodging an object approaching us at too fast (Caño, 2020).

➢ Ventral route:

Injuries in this pathway will cause a deficit in object recognition.

2.3.2. RECOGNITION DISORDERS: AGNOSIA

Agnosia, according to Gich (n.d.) is a specific affectation of recognizing previously learned stimuli or recognizing stimuli that can be commonly learned after adequate exposure. This, in addition, must happen in: absence of visual disorder, absence of language disorder, absence of intellectual disorder, being a consequence of brain injury and is specific to a sensory modality (the patient who may not recognize it through a specific afference is usually able to identify it by another entry).

2.3.2.1. HISTORY - VISUAL AGNOSIA

The German physiologist Hermann Munk in 1890 coined the term **psychic blindness** by observing that dogs that had undergone bilateral ablation of the occipital lobes skilfully avoided obstacles in their path – so they preserved their sight – but did not react appropriately to the presentation of objects that had previously frightened them – lack of recognition.

Then, later that year, the German neurologist Heinrich Lissauer made a categorical distinction between two different levels of mental blindness and recognition: aperceptive and associative.

The aperceptive level corresponds to visual processing and patients, therefore, are not able to copy (draw) objects they do not recognize (Gich, n.d.).

The associative level allows us to associate meaning with perception.

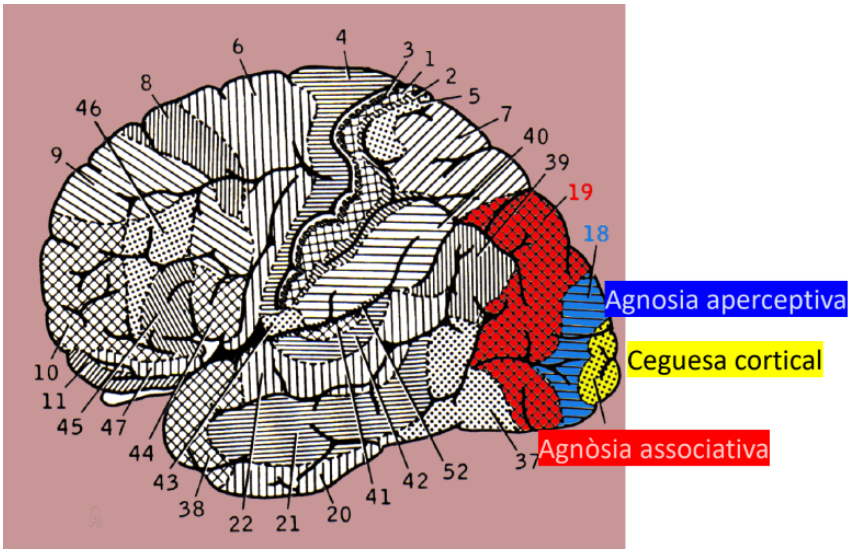
Finally, in 1891, Freud called mental blindness "agnosia" (Gich, n.d.).

2.3.2.2. SKETCHES OF NEUROANATOMY

As explained above in section 1.2.3.1, elementary visual information from the retina to the external geniculate bodies and then to the striatum area (primary visual area, Broadmann's 17) is subsequently subject to a separate treatment by shape, colour and movement (Gich, n.d.). Figure 8 shows how this process is related in the Broadmann areas with the associated pathologies.

Figure 8

Representation of Broadmann areas associated with types of psychic blindness



Note. Adapted from Gich, n.d.

Also continuing with what has been explained above about the ventral pathway *What* and the dorsal pathway *Where*, a series of differential characteristics should be highlighted as shown in table 1:

Table 1

Differences in functioning between the ventral and dorsal pathways

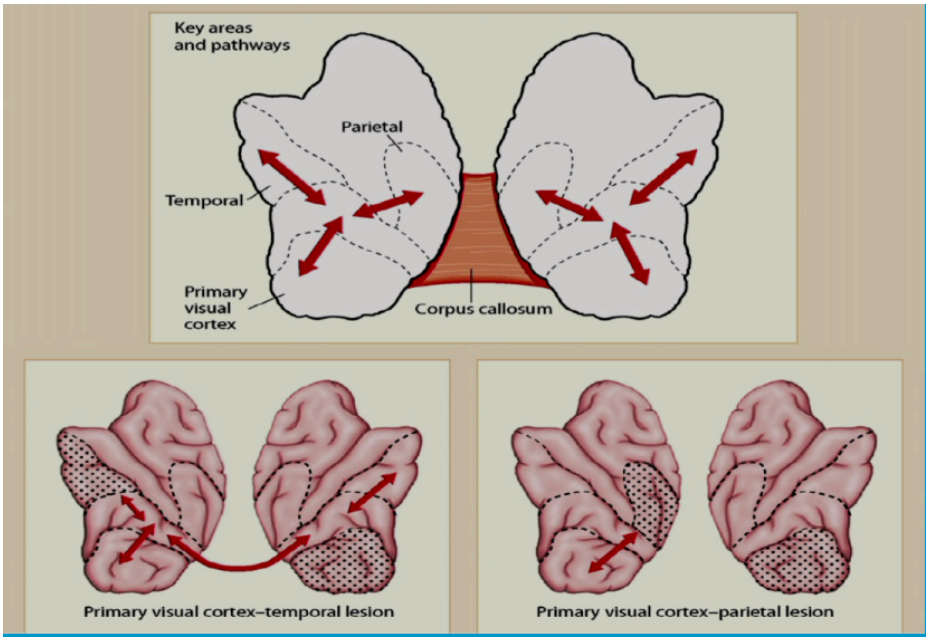
Via ventral *What*	**Via dorsal *Where***
Perception	Action
Characteristics of objects	Ability to direct movements towards objects
Conscious Information	Non-conscious information
First centre in visual recognition	Most elaborate phase in visual recognition
Visual perceptual tasks	Perceptual motor tasks

Note. Adapted from Gich, n.d.

The corpus callosum only provides an interhemispheric connection for the ventral visual pathway, but not for the dorsal, which is more lateralized at the hemispheric level (right). In this way, objects that are touched with the left hand can cross from the right hemisphere to the left hemisphere and reach the language. Figure 9 shows a representation of this.

Figure 9

Functional representation of the corpus callosum in relation to the primary visual cortex



Note. Adapted from Gich, n.d.

2.3.2.3. TYPES OF AGNOSIA:

Among the different types of agnosia are: visual (visual aperceptive and visual associative), auditory and tactile. For the study it occupies, only the visual will be discussed.

➢ Visual agnosia:

According to Junqué (2001), quoted by Gich (n.d.), alteration of the visual recognition of objects encompassing other categories such as faces or colors, with visual acuity, visual fields, visual tracking, linguistic function and higher cognitive function intact. Objects are detected, but not identified.

➢ Aperceptive visual agnosia:

Basic visual functions preserved. Damasio and Geschwind (1985) (cited by Gich, n.d.), consider that aperceptive agnosia would not be "real recognition disorders", but a consequence of perceptual alterations (pseudoagnostic syndrome).

They have difficulty matching and drawing. In general, patients explain that they fail in the proposed tasks because they do not see it well. They do not draw in the copy or discriminate in a matrix two copies of the same object.

If they are shown, for example, a watch, they will be unable to identify it as such, but if they can hold it with their hands they can name it correctly. Likewise, if they are asked to identify a clock by an oral description of it, they have no difficulty in doing so (Gich, n.d.).

Patients, too, are unable to point out the objects cited by the examiner.

Unlike cortical blindness, visual acuity is perfectly preserved in aperceptive visual agnosia; What they fail is the specific recognition of elements that require the perception of shapes and patterns (geometric figures, letters...).

➢ Associative visual agnosia

According to Gich (n.d.), normality in elementary visual processes (visual acuity) normally in copying, image pairing and visual description. This indicates indemnity in perceptual processes. The fundamental distinguishing feature of associative agnosia is that subjects do not recognize objects, but are able to describe and draw them by copying. However, there is also an absence of global mental disorders (confusional syndrome or dementia).

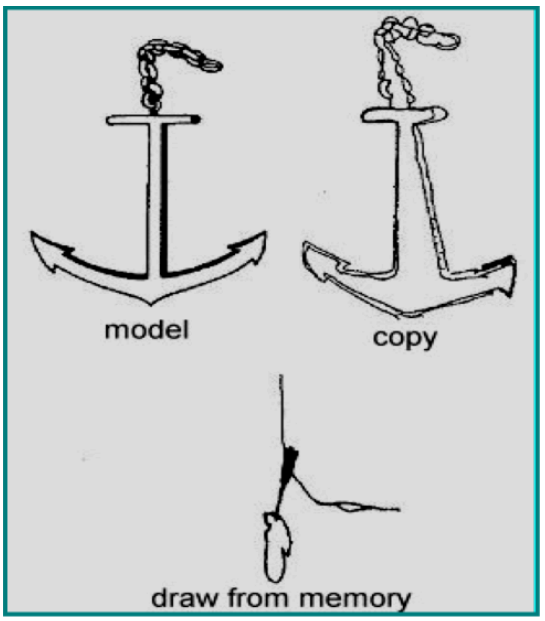
It is "real visual agnosia" and occurs as a result of injuries in the tertiary areas of the II Functional Unit of Luria (CFU) (Gich, n.d.).

Patients with associative visual agnosia, as shown in Figure 10, can copy, for example, an anchor, and also define it; but they cannot draw it without having it in front of them, having

to access the mental memory image of the object, demonstrating a deficit in this access to the mental image.

Figure 10

Example of drawings (in copy and memory) of a person with associative visual agnosia



Note. Adapted from Gich, n.d.

➢ Differences:

Table 2 shows the main differences between the two agnosias.

Table 2

Differences between aperceptive and associative agnosia

TYPE	COPY/DRAWING	PAIRING
APERCEPTIVE Perceptual defect	**NOT**	**NOT**
ASSOCIATIVE Disconnect between perceptual processes and semantic memory	**YES**	**YES**

Note. Adapted from Gich, n.d.

➢ Some disorders of visual agnosia: simultagnosia and prosopagnosia:

- SIMULTAGNOSIA (aperceptive agnosia):

Wolpert (1924) (quoted by Gich, n.d.) designates the inability to recognize complex images, while details can be perceived, without coherent synthesis being possible. Subjects can see only one object at a time.

  - Dorsal simultagnosia: bilateral parietooccipital injury, often associated with Balint's syndrome and which could be related to oculomotor disorders (object mobilization aggravates the deficit) (Gich, n.d.).
  - Ventral simultagnosia: due to injury to the left temporoccipital conjunction, usually less severe, associated with an alexia of spelling and which could follow a perceptual problem (Gich, n.d.).

- PROSOPAGNOSIA:

Described in 1860 by two ophthalmologists, Quaglino and Borelli, but not until 1947 was called prosopagnosia. It consists of a difficulty in recognizing previously learned faces and an inability to learn new ones (in severe cases, they may not recognize one's face in photographs); Patients deny knowing these faces shown to them, but if they are given other information (they hear the voice, see the person's way of walking, the clothes they wear, etc.) they can identify them.

They retain the knowledge of people who are unable to recognize, so they are unable to recognize the King, the President..., but once they manage to give the name they can give abundant information about it (Gich, n.d.). Most cases have difficulty identifying objects exactly, such as flower classes, dog breeds, car brands..., so some researchers have speculated that prosopagnosia is actually a defect in the distinction of objects within categories containing specimens that may be confused; may mistake a lion for a tiger, but never for an elephant (Gich, n.d.).

2.4. MEMORY:

2.4.1. WHAT IS IT AND HOW DOES IT WORK?

Memory is the neurocognitive process that allows information to be recorded, encoded, consolidated, stored, accessed and retrieved. According to Luria (1966) (Gich, n.d.), in fact, this would be the ability to print, retain and reproduce the traces of previous experience, which gives man the ability to accumulate information and count on the signs of previous experience after the phenomena that motivated it disappeared.

When we talk about memory, we are talking about three parameters that make it up: temporal, sequential and domain. According to Gich (n.d.):

- ➢ Sequential parameter:
    - Coding: processes by which attention is paid and new information learned is elaborated. Good coding is done carefully and associating it meaningfully and systematically with existing knowledge, integrating new information with what we already know.
    - Consolidation: includes processes that alter newly stored information (still labile) to make it more stable.
    - Storage: all the mechanisms and places by which memory is preserved in the long term in our brain.
    - Retrieval: refers to the processes that allow the recovery and use of stored information.
        - It involves gathering information that is stored in different places.
        - The most effective recovery when it takes place where the information was acquired and in the presence of the same keys
        - Recovery is actively dependent on working memory
        - The subsequent recall is not an exact copy of the information originally stored. When we remember we use strategies (comparison, skilful reasoning ...) to generate a memory congruent and coherent with our schemes
        - Recovery is a constructive process and consequently subject to distortion (Frederick Barlett)

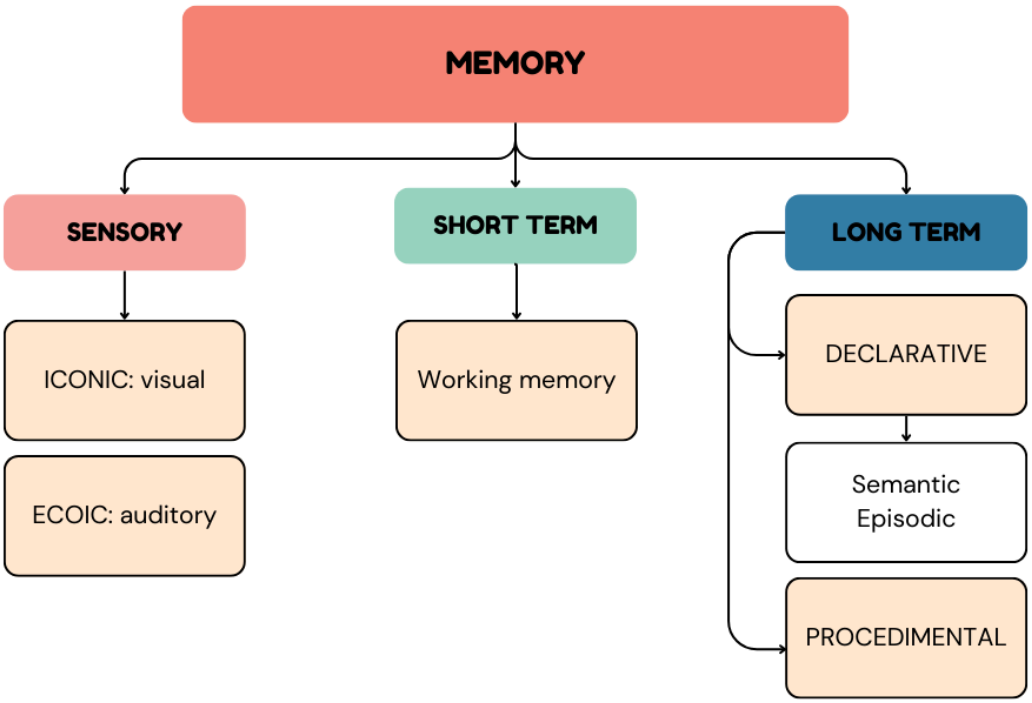
- ➢ Time parameter: memory is divided into sensory memory (a few seconds), short-term and long-term.

- ➢ Domain parameter (how is memory retrieved and evoked?): declarative/explicit memory and procedural/implicit memory exist

According to the theory of multiple stores, memory is divided into sensory memory (system that retains sensory information received unconsciously through the senses, lasts a few seconds), which is subdivided into iconic (visual) and echoic (auditory), short-term memory (composed of working memory) and long-term memory, which is subdivided into declarative (semantic and episodic) and procedural. See figure 11.

Figure 11

Schema typologies of memory



Immediate (or sensory) memory, therefore, has a direct relationship with perceptual processes. It is a set of storage systems that are modally specific, where perceptual information generated by the senses is retained for a short period of time (1-2 according to visual afferences and a few seconds auditory afferences) (Gich, n.d.).

According to Gich (n.d.), short-term memory is that of limited capacity and that supplies the substrate of conscious experiences, since it allows to maintain and manage information for a short period of time. This is composed of working memory (model of Baddeley and Hitch of 74), which is the way to operate this short-term memory, which allows us to reason, solve problems, perform mental calculations, understand language and perform executive functions. This is a tripartite system: on the one hand the central executive and two subsystems that are the articulatory loop (verbal information) and the visuospatial drawing block (visual information).

The central executive is responsible for controlling and integrating the different sources of information, directing attention.

The articulatory loop, on the other hand, is related to the linguistic, auditory-verbal aspects of working memory and is formed by two components: the phonological store (which has the function of maintaining verbal or acoustic information for brief seconds) and the articulatory control processor (inner speech) which has two functions: it can keep the material in the

phonological store thanks to subvocal repetition and can recode visual material and record it in the phonological store by subvocalization (Gich, n.d.).

The visuo-spatial notepad has the function of maintaining visuo-spatial images through covert movements of the eyes (equivalent to the subvocal repetition of the articulatory loop). It is contemplated by two different neuroanatomical pathways mentioned above: the *What* pathway (related to the integration of color, shape, size and texture of the object seen) and the *Where* pathway (related to the ability to notice spatial relationships) (Gich, n.d.).

Long-term memory (LTM), on the other hand, according to Gich (n.d.) consists of the evocation of information after an interval of time, in which the subject's attention has focused on another task. Depending on the time parameter it can be divided into recent LTM and remote LTM, and depending on the type of information in declarative memory (episodic or semantic) or procedural.

The recent LTM allows you to maintain information from minutes to days and includes the learning of new declarative information. It is justified by the process of encoding-consolidation of the information that is carried out in the medial temporal structures, diencephalic limbic and the anterior basal brain (Gich, n.d.).

Instead, remote LTM is the information we have stored for months and years. It is stored in the cerebral cortex (in posterior associative cortical areas) and there is a previous coding and consolidation process (Gich, n.d.).

Explicit or declarative memory refers to the ability to consciously remember information about specific facts, events or things. This can be subdivided into semantic memory, related to knowledge we have about reality, general knowledge, concepts (objects, facts, rules, etc.), which are acquired at specific times -but become independent of the original event or some personal experience- and is resistant to brain injuries and episodic, which is the memory of events, situations and life experiences that the person has experienced at a certain time and place. Semantic memory, for example, includes knowledge about the world, definitions, historical facts, mathematical concepts, etc. while episodic memory allows us to remember, for example, an autobiographical experience (where it happened, when, how the person felt at that moment, etc.).

On the other hand, procedural memory is that most unconscious memory of the plane related to the learning of motor, perceptual and cognitive skills that are acquired gradually and slowly. An example of procedural memory is the action of driving a vehicle.

Finally, we must also highlight the concepts of retrograde and anterograde memory; The first respectively refers to the ability to remember information from the past, while the second is

the ability to continue memorizing information (future). These two concepts, however, are used to talk about pathological cases (cases of brain injuries or degenerative pathologies, for example). When we talk about retrograde amnesia, then, we talk about the inability to recover events that happened before the injury, and when we talk about anterograde amnesia we talk about the inability to memorize after brain injury (Gich, n.d.).

2.4.2. ANATOMICAL BASES OF MEMORY:

To know memory in its entirety, it is necessary to understand it in a binomic way, in which we must understand its functioning both from a physical and a psychological aspect.

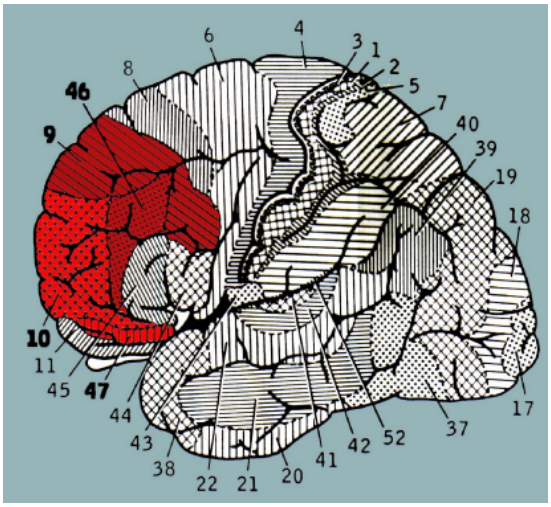
First of all, it is necessary to briefly highlight all the brain structures involved in the memory executive function: the frontal lobe (at the level of storage of short-term memories), the temporal lobe (at the level of creation and storage of both episodic and semantic long-term memories, and at the level of short-term material processing), the motor cortex (involved in procedural memories), the hippocampus (responsible for forming new long-term semantic and episodic memories), the amygdala (at the level of formation of new emotional memories) and the cerebellum (storage of procedural memories created in the motor cortex) (Bonet, 2008; García, 2014; Palenzuela, 2018).

Some of these are explained a little more in detail below:

- ➢ Central executive (figure 12): Broadmann's areas 9, 10, 46 and 47

Figure 12

Brain areas involved in the central executive



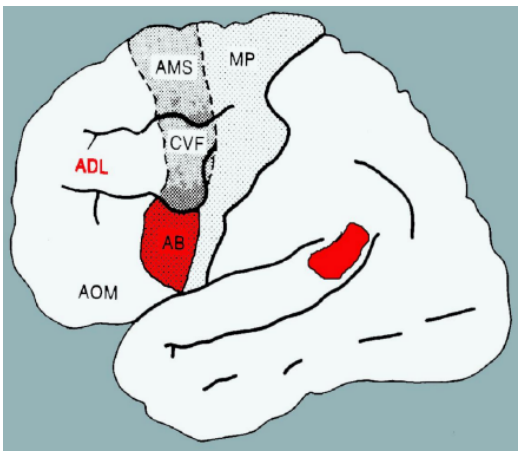
Note. Adapted from Gich, n.d.

The prefrontal cortex is associated with executive functions such as planning, decision making, impulse control, problem solving, and working memory. That is why these areas are those related to the central executive, due to the very functional nature of the system.
Area 9 of Broadmann, along with area 10, is involved in planning and organizing behavior, monitoring higher cognitive functions and working memory. Area 10 is involved in multisensory information integration, complex task planning, powerful decision-making and social cognition.
Area 46 plays a crucial role for working memory (and thus for cognitively complex tasks such as problem solving, selective attention, and decision making). And, finally, 47 is involved in the regulation of emotions (decision making based on rewards and cognitive flexibility).

- Articulatory loop (figure 13): concept of *rehearsal process*: this refers to a cognitive process in which information is actively repeated to keep it in working memory for a short period of time, a key process of the articulatory loop of the Baddeley and Hitch memory model. We also find, in this system, the structure of phonological short-term memory (the part that temporarily stores verbal information while it is actively repeated through the *rehearsal process*). Some of the brain structures involved, therefore, with all these processes and systems are Broca's area (verbal production), Wernicke's area (language comprehension and integration), primary motor area (production of articulated discourse), dorsolateral area of the prefrontal (fundamental role in the active manipulation of information from working memory, including in *rehearsal process*) and the internal capsule (important for efficient communication and transmission of signals between different areas involved in language processing), among others.

Figure 13

Brain areas involved in the articulatory loop



Note. Adapted from Gich, n.d.

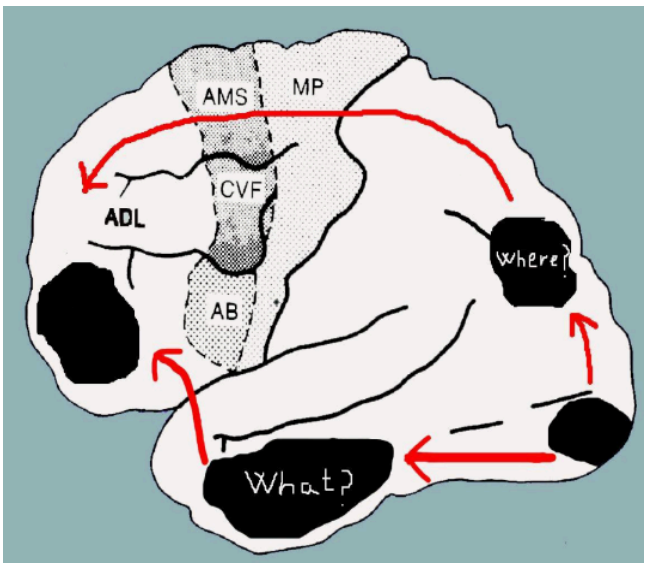
- ➢ Visuospatial notepad (Fig. 14): via *What* (ventral route, inferior temporal lobe) and via *Where* (dorsal route, posterior parietal lobe)

  The visuo-spatial notepad is related to these two visual processing pathways.

  - ➢ Via what: in the context of notepad, this pathway stores information about visual details of objects, and when a person needs to remember or manipulate this information, the visuo-spatial notepad is activated to process it.
  - ➢ Via where: also in the context of the block, it is related to the location and spatial orientation of objects when remembering the position of an object in the environment or planning a spatial path, the block is what allows storing and manipulating this information.

Figure 14

Graphic brain representation of the dorsal and ventral pathway

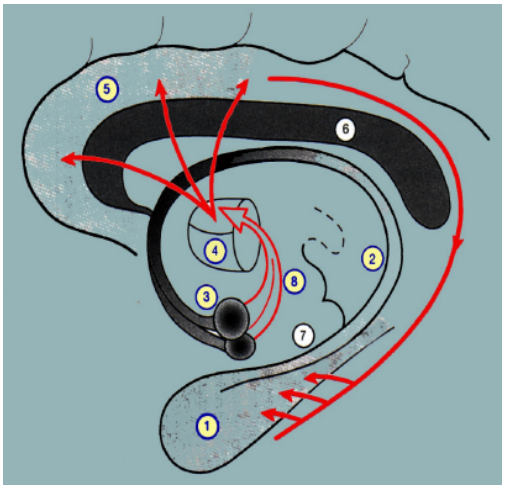


Note. Adapted from Gich, n.d.

➢ Papez circuit (figure 15): although it is a circuit originally proposed as a way to explain the functioning of emotions, its relationship with memory has also been recognized, especially emotional memory (how emotional experiences can influence the formation and recovery of memories). This circuit is mainly made up of the hippocampus, cingulate gyrus, fornix, mammillary bodies and thalamus.

Figure 15

Circuit de Papez



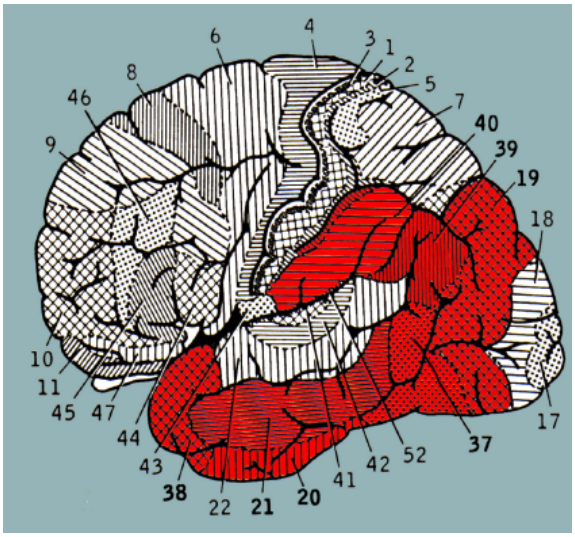
Note. Adapted from Gich, n.d.

➢ Posterior cortical areas of association (Figure 16): have an important role in memory in terms of integration of multisensory information (which can subsequently improve

the formation of memories and information retrieval), in terms of the construction of cognitive maps -of the environment and space- (which may facilitate the memory of past events and navigation in the environment) and the processing of contextual information (which will be critical for the precise retrieval of memories and their relationship to contexts).

Figure 16

Posterior cortical areas of association



Note. Adapted from Gich, n.d.

2.4.3. ALZHEIMER'S AND MEMORY

The cognitive detention that occurs in AD, although it covers practically all cognitive functions, undoubtedly the one that is most affected and is also present from the beginning of the disease, is memory (Cuetos et a., 2003).

In AD the first symptoms that usually manifest themselves are in the alteration of episodic memory, which is why neuropsychological exploration of this cognitive function is usually so important (for example, through learning word lists or text memory): small everyday forgetfulness usually occurs in these earlier stages, such as forgetting the keys, what day of the week it is, etc., all these are episodic memories and the primordial feature of AD.

However, short-term memory is the one that is mainly limited: as explained, it works by a set of systems that are proposed by Baddeley and Hitch in their model of the Working Memory of the year 74; the articulatory loop the visuospatial agenda and the central executive, each with the corresponding functions explained above. The affectation of these three components generates one of the most outstanding characteristics of AD, and that it produces the greatest

impediments, such as amnesia -partial or total loss of memory that produces an inability to retrieve information-. In addition, this memory disorder can become so severe that not only is the possibility of retrieving information lost, but all notion of having been exposed to it is lost (Ostrosky-Solís, 1998; Palenzuela,2018).

In general, people suffering from AD have anterograde amnesia, retrograde amnesia and nonspecific amnesia with confabulation (appearance of false memory traces caused because the person is unable to selectively evoke their memory fingerprints) (Palenzuela, 2018).

In short, the transfer of sensory memory to short-term memory requires attention and the transfer of short-term memory to long-term memory requires repetition and organization. If all these interrelated processes fail due to the presence of a pathology such as AD, memory will not fulfill its function (Ostrosky-Soís & Lozano, 2003, Palenzuela, 2018), which will create alterations not only in the functions themselves, but also in other cognitive processes that require their correct functioning to be executed, such as language and whose alterations, they also cause major functional limitations (Pérez, 2012; Palenzuela, 2018).

On the other hand, the memory that Tulving calls semantics (already explained above, a kind of "mental dictionary") has also been the subject of interest in the study of cognitive profiles of AD, and this has led to more controversy over the years. As we know, initially Tulving proposed episodic and semantic memory systems as functionally independent systems, but as a result of countless neuropsychological studies today it is clear that these systems interact considerably, especially in the stages of information encoding and retrieval (Becker & Overman, 2002).

The results of the different studies on deficits in semantic memory in AD patients are, in general, quite contradictory: Studies with nomination by visual confrontation or word generation, for example, have shown that this aspect is seriously affected, while in other types of dementia (such as Huntington's disease) it is relatively preserved, so it seems that the loss of semantic knowledge is a fundamental characteristic of pathological processes (such as AD) that affect the functional integrity of the temporal lobes (Becker & Overman, 2002). However, the main problem is that it is not clear whether these findings reflect a true disorder of semantic knowledge (actual loss of information), a disorder in the ability to access/use the knowledge base, or both (Becker & Overman, 2002); thus some models propose that the fundamental characteristic of semantic impairment in AD is the loss of information storage, others that there is a deficiency in information retrieval and others that there is an alteration in the basic structure of semantic memory (Becker & Overman, 2002). Are some of the questions raised, for example, in the case of pathological results in

nomination for visual comparison, due to a loss of knowledge about the semantic characteristics that define the lexical references corresponding to a given object? Is it because there is an alteration in the perceptual analysis of the object? Or is it because there is a problem accessing the specific name of the object? (Becker & Overman, 2002).

Several authors have suggested that AD patients have a deficit in knowledge of the meaning of things. This is based on the fact that these patients generally retain knowledge of the semantic category to which the object belongs, but lose information about its specific attributes (they tend to name objects using the category name) (Becker & Overman, 2002). This pattern of cognitive impairment suggests that categorical information is conserved, but a loss of specific attributes is beginning to be perceived that makes it possible to differentiate semantically related objects (Becker & Overman, 2002).

However, on the other hand, there is evidence to suggest that AD patients may retain knowledge of semantic attributes; the authors Nebes and Brady (1988) applied a test in which they measured the time it took elderly people (healthy and AD) to decide whether a particular word related to a particular object or not. and on the basis that there is a loss of semantic consciousness in AD, these patients should respond more slowly; But that wasn't the case. However, it should be noted that these results have not been found by other authors, but at least we could say that this conflicting aspect of the study suggests that AD patients are to some extent aware of the relationship between concepts and their attributes (Becker & Overman, 2002).

On the other hand, already using more innovative techniques, Chan et al. (cited by Becker & Overman, 2002) showed that the structure of semantic connections is altered in AD. Therefore, it is difficult to reconcile the results of studies that show contradictory conclusions about the knowledge of the concepts in AD, which is how Nebes (cited by Becker & Overman, 2002) has proposed a possible cause for this discrepancy, attributing it to the degree of cognitive demands imposed by the different neuropsychological tests, which can lead to draw erroneous or not entirely accurate conclusions depending on the difference of evidence that can evaluate a same look. Thus, what in some studies may appear as an affectation of the structure of semantic memory in AD could mean, on the other hand, a more generalized failure in the processing of cognitive information (Becker & Overman, 2002).

Finally, regarding the neuroimaging studies, it seems that they all suggest the existence of anomalies in the functional networks involved in the processes of semantic memory retrieval in AD. Saykin et al (cited by Becker & Overman, 2002) found differences in brain zone

activation between AD patients and healthy controls when performing two semantic decision-making tasks related to a phonological decision-making task (figure 17).

Figure 17

Group semantic and phonological performance task (MA/C)

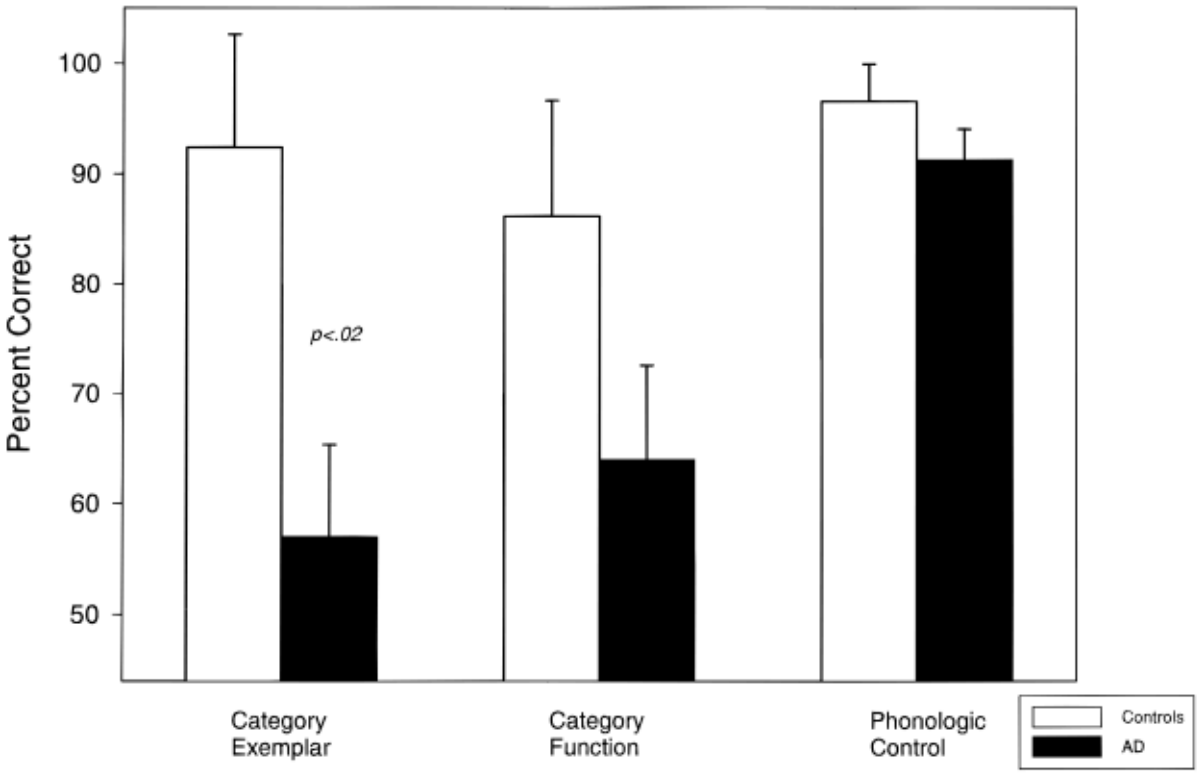


Fig. 1. Semantic and phonologic task performance by group.

Note. Adapted from *Semantic and phonologic task performance by group*, by Saykin et al. (1998).

Finally, as a final conclusion within all this controversy of studies, the process of relationship between information loss and retrieval can ultimately be explained on the basis of relative atrophy of the frontal and temporal lobes (Becker & Overman, 2002).

2.5. FACIAL RECOGNITION AND PERCEPTION

When we talk about facial perception we refer to the processes of interpretation, conception and understanding of faces as visual stimuli. So let's talk about processes outside the conscious plane.

On the other hand, when we talk about facial recognition, we refer to the process of connecting the information collected from this perceptual process with the information stored internally on that specific face, so that a process of memoristic recovery intervenes in order to connect if we know that face or not.

2.5.1. BASES OF PERCEPTION AND FACIAL PROCESSING: HOW DOES IT WORK?

Haxby et al. (2000), cited by Werheid & Clare (2007), integrating the various neuroimaging findings of the time, proposed a distributed neural system for the perception of faces, which involved, on the one hand, the interaction of highly specialized fusiform brain areas located with the primary visual cortex and, on the other hand, projections to widely distributed brain areas that would serve facial processing depending on the task, such as the amygdala to process facial emotion, or the anterior temporal cortex to recover the name that corresponds to the face -if known-. In fact, Cuevas (2021) explains that evidence indicates that there is an area of the brain especially dedicated to facial perception; the medial region of the fusiform gyrus of the right hemisphere (region later baptized as FFA) showed greater activation in the presentation of faces than in front of any other stimulus. Cuevas (2021) also explains that both the exact location of the area varies interindividually and, in certain cases, activation occurs bilaterally or in the left hemisphere.

Tsao and Livingstone (2008) proposed a unifying hypothesis, deduced from computational, neurological, fMRI and individual experiments on how this face processing system works, which should explain this dissociative line between deficits in face and object recognition according to certain pathologies. Thus, they propose that what makes face processing special is that it is limited by a mandatory screening process that precedes identification in this processing.

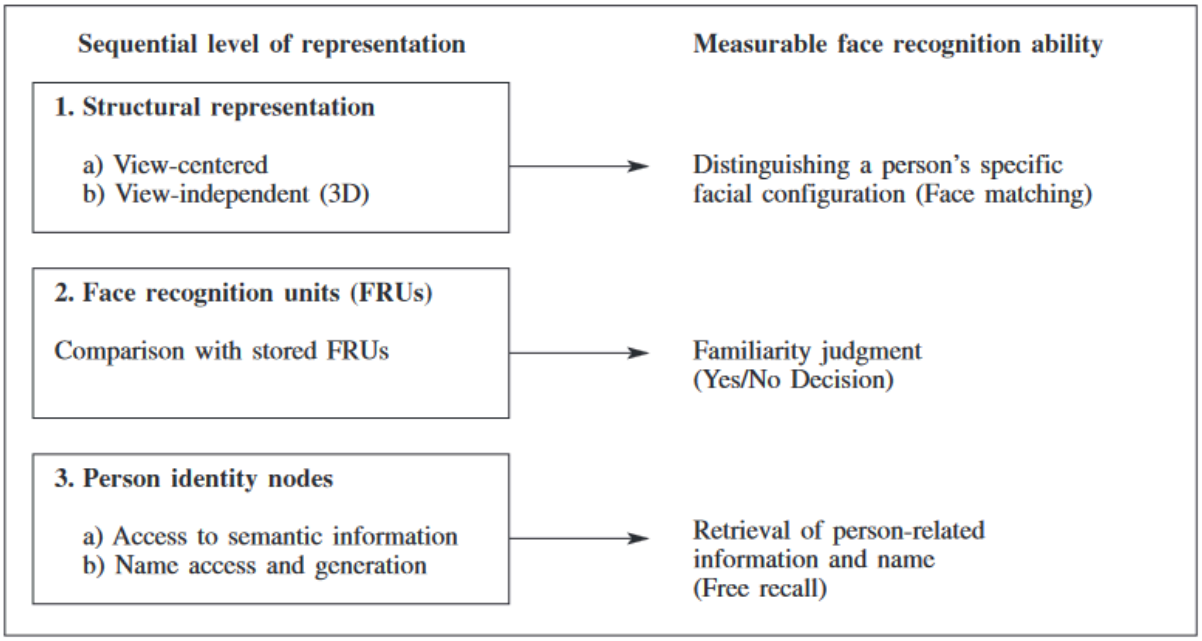
One of the most important factors when talking about facial perception is understanding it as a holistic process; this is shown by the evidence, as pointed out by Tsao and Livingstone (2008), Alfaro et al. (2016) and Werheid and Clare (2007), for example. Understanding this process as holistic means understanding that faces are mentally represented as complete entities rather than a combination of independent parts; Tsao and Livingstone (2008) point to two effects that support this idea: the part-whole effect and the composite effect. The first refers to people being better at distinguishing two parts of a face in the context of a full face than in isolation, and the second indicates that people are slower to identify half of a face aligned with the other half unconscious than if the two halves are desalinated (Tsao and Livingstone, 2008).

To understand how facial perception works in humans, it is important to first take a brief look at the first significant historical proposals in this regard, such as Bruce and Young's model from 1986. Integrating the findings of cognitive psychology and clinical observations in a unified framework, Bruce and Young (1986) developed the classical model of facial recognition (Werheid & Clare, 2007) (figures 18 and 19). According to this model, as

explained by Werheid and Clare (2007), recognizing a person by their face implies the transformation of a particular perceived face into a point-invariant structuring code: this three-dimensional representation of the face is then compared with previously stored structural representations (the so-called "facial recognition units"), thus allowing a familiarity judgement. Finally, if the face is considered familiar, semantic information is accessed followed by the recovery and generation of the person's name.

Figure 18

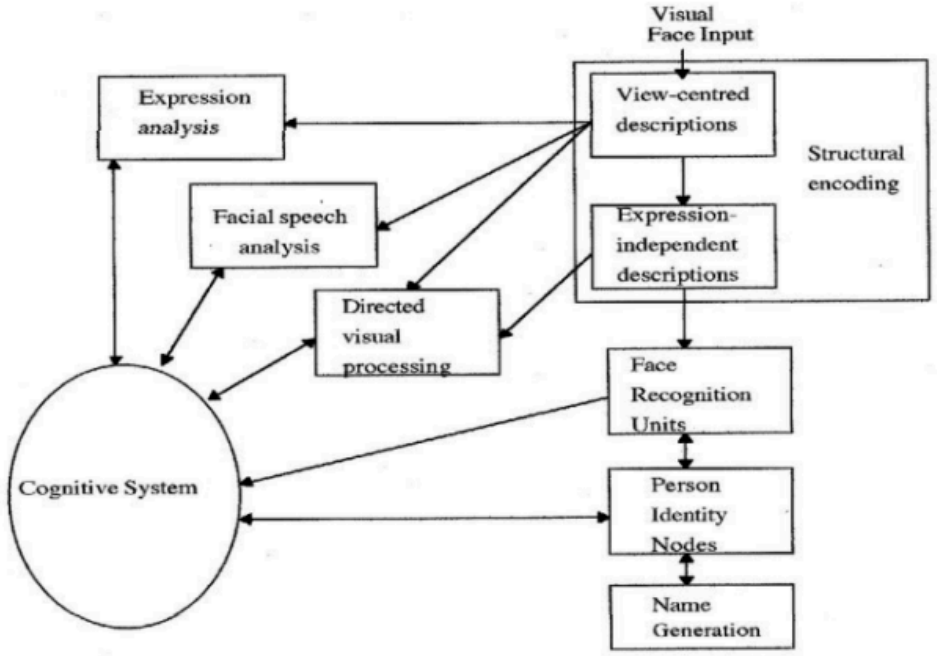
Schematic illustration of Bruce and Young's sequential facial recognition model (1986)



Note. Adapted from "Are faces special in Alzheimer's disease? cognitive conceptualisation, neural correlates, and diagnostic relevance of impaired memory for faces and names", by Werheid and Clare (2007), *Cortex, 43* (7). AD initially affects level 3, progressively to level 2, and level 1 is saved until the severe stage of dementia (Werheid & Clare, 2007).

Figure 19

Bruce and Young's face processing model (1986)



Note. Adapted from "Facial perception and recognition: theoretical bases of recognition wheels" by Alfaro et al. (2016), *Gac. int. cienc. forensics, 18*.

As noted in chapter 43 of the *Handbook of Neuroscience for Behavioral Science* , there is evidence that face perception involves specialized cognitive and neural mechanisms, different from those involved in the perception of other visual stimuli (Kanwisher & Yovel, 2009). Specific areas in the temporal lobe have been found to be involved in face processing (Tsao & Livingstone, 2008). The main brain regions involved in the process of facial perception are the fusiform cortex, the fusiform area of the faces (FFA) -it has been shown to respond selectively to facial stimuli-, the occipital region of the face (OFA) -essential for the processing of parts of the face and in contrast to FFA that responds to the identity perceived by the face- and the region of the superior temporal surcus (FSS). These regions have been identified through both neuropsychological and functional neuroimaging (fMRI) studies, and are all involved in facial identity perception, discrimination of social and dynamic information in faces, and in the perception of emotional expressions (Kanwisher & Yovel, 2009).

This hypothesis that supports the specificity of faces in perception comes from both neuropsychological, behavioral, electrophysiology and neuroimaging. From the first source respectively, Kanwisher and Yovel (2009) mention that neuropsychological studies have demonstrated the existence of double dissociations between face recognition and object

recognition (non-facial stimuli), suggesting that these two phenomena do not share the same processing mechanisms. Regarding the second source respectively, this evidence has shown through exposure to stimuli of facial inversions and partial or whole faces a series of specific effects of processing that are not observed in the case of other non-facial stimuli. Regarding the third source respectively, with these electrophysiological measurements these specific regions of FFA and fSTS that are activated by facial stimuli have been identified, while also the evidence showing brain lesions in certain selective areas (such as prosopagnosia) supports this hypothesis.

Finally, other types of research that seem to provide evidence on the specific areas of the brain for the perception of facial stimuli are those related to children; according to Cuevas (2021) it is known that babies a few days old are born to show a greater preference for observing faces than other stimuli (Slater, 1993), and although the visual system is still immature, research has found these preferences. In the case of Johnson et al. (1991), cited by Cuevas (2021), they showed a group of newborns three types of stimuli: a drawing of a face, a drawing of a face but with mixed facial features, and the outline of a face without facial features; The babies showed a greater preference for the first stimulus respectively. Similarly, Heering & Rossion (2015), cited by Cuevas (2021), found evidence of different electrical activity (right occipitotemporal region) in infants aged 4-6 months for facial stimuli.

2.5.2. FACTORS N170, VPP AND N250

Although the identification of familiar faces in everyday life is preserved in ageing, faces are still complex and highly specific visual stimuli for us, and this processing is altered in aging, mild cognitive impairment (MCI) and mild Alzheimer's disease (Saavedra et al., 2015). There is evidence that during ageing, the initial configurational processing of faces preceding facial recognition is affected, and that semantic processing underlying the recognition of familiar faces is also affected in mild MCI and AD (Saavedra et al., 2015).

Evoked potentials constitute neurophysiological responses that allow neurocognitive processes to be analysed with high temporal resolution (Saavedra et al., 2015); thus, the scientific literature on evoked potentials specifically related to face processing has focused phonally on the N170 component, a negative component observed at around 170 ms in electrodes located in occipital and temporal zones. and is defined with a greater negative amplitude in front of facial stimuli than in other stimuli (Saavedra et al., 2015).

In relation to face processing, it is considered that this component reflects a neurocognitive mechanism involved in face detection compared to other types of stimuli and, particularly, is

responsible for the coding processes of the face structure for subsequent recognition (Saavedra et al., 2015).

This component also coincides temporally with another evoked potential located in the anterior areas of inverse polarity, the VPP; this is also related to face processing, presenting a greater positive amplitude in front of faces compared to other objects (Saavedra et al., 2015).

According to Saavedra et al. (2015) the existing scientific literature on the effects of both aging on the one hand and cognitive decline on the other, on face-specific evoked potentials is scarce and the results are not totally convergent; for example, in the effects of cognitive impairment, there are studies that have found no effects on evoked potentials related to facial processing in patients with mild AD, while there are others that have. Thus, in the study by Saavedra et al. of 2015, the differences in cortical activity related to these two components were studied transversally and longitudinally between young adults, older adults and patients with cognitive impairment (a group with AD), thus finding significant differences between all groups and, with respect to the present study, especially compared to the older adult group and the older adult group with mild AD. These changes appear from the early stages of facial processing (Saavedra et al., 2015).

Finally, the N250 component is another evoked potential that appears as a negative wave around 250 ms detected in temporo-occipital zones which, according to Sommer et al. (2021) is sensitive to the construction of a stable representation of a face in memory, and the increase in amplitude of this component reflects the acquisition of memory representations of a new facial image, so it is specifically sensitive to facial familiarity. In this way, it is believed that he is involved in the processing of facial identity, the recognition of familiar faces and reflects the memory of them.

2.5.3. FACIAL RECOGNITION IN AD: MNESIC LINE AND GNOSIC LINE

The difficulties in facial recognition prevalent in AD (difficulties, for example, in recognising and knowing who family members themselves are when seeing them) have usually been attributed to underlying memory impairment (episodic and semantics) (Lavallée et al., 2016). This is because this lack of recognition is one of the most striking symptoms, while memory deficit is also one of the most central aspects associated with the disease. Thus, these aspects have been extensively studied in relation to episodic anterograde memory of faces, as in the retrograde semantic memory of people (studies with celebrities), and the deficits in both cases are present and confirmed in AD (Lavallée et al., 2016), but there are lines of research that emphasize that they are not the only explanation.

Thus, it is not only a question of taking into account the *mainstream* mnesic line of this deficit, but the possible Gnosian line; a more perceptual deficit specific to this complex and special typology of stimuli such as human faces, as explained above. This study aims to provide evidence in this second less-explored line, for which there is already some empirical evidence (although little) that supports it.

2.5.4. EXPERIMENTS AND LITERATURE OF FACIAL RECOGNITION IN AD

A 2016 study (Lavallée et al., 2016) in order to investigate exactly whether AD patients have these specific difficulties in recognizing faces, simultaneously compared the ability of patients with healthy controls to pair individual faces and non-facial stimuli (in this case, cars), in both cases with the stimuli presented both right-oriented and inverted. The results showed that patients had a reduction in the effect of facial inversion, suggesting difficulties in the perception of faces (Lavallée et al., 2016). With these findings of lower overall performance by AD patients, it was suggested that in addition to memory difficulties, these people also have difficulties in higher-level visual processes, specifically in the perception of individual faces (Lavallée et al., 2016), providing new options and lines about the nature of this deficit.

On the other hand, in the study conducted by Becker et al. (1995) in which they also proposed understanding the impaired analysis of faces by patients with probable AD, a test was carried out composed of three different tasks: the first on shape discrimination (analysis of visual information), a second on discrimination against unfamiliar faces and a third on naming famous familiar faces (president's test). In this second task, that involved essentially in aspects of facial visuoperceptive skills, they found that the performance of AD patients likely was significantly impaired compared to controls, concluding the study that the face discrimination defect in AD is not due to an overall deterioration in visuospatial or attention capabilities, thus pointing to a possible specific deficit.

In another study, for example, in the case of Huang et al. (2020), they wanted to examine how AD progressively alters the visual processing network evoked by faces, thus providing literature on this gnosic line of facial recognition. They conducted a study with 19 AD patients (8 severe AD and 11 medium/moderate MA) and 26 functional magnetic resonance imaging (fMRI) controls while looking at facial photographs. In this study, it was found that the pathology of this disease not only alters local neuronal activation but also the activity of the associated visual neural network (Hung et al., 2020); An altered visual network pattern

was identified that was reduced and interrupted completely linked to the degree of severity and progression of the disease.

The temporal sequence of the pathology of this disease moves from the areas of visual association of 1st order to the participation of lower-order visual processing regions as the disease progresses spreading to the primary visual cortex, so that the neuronal loss associated with the accumulation of amyloid plaques and neurofibrillary tangles in these areas can alter, as mentioned, not only local neuronal activation but also visual neural network activity (Hung et al., 2020). Through these types of studies and with others on the specialty of facial stimuli as visual stimuli, these different researches begin to be connected.

In another study, in the case of Mazzi et al. (2020) they published research on the importance of electrophysiological markers in order to make a differential diagnosis between amnesia or agnosia in facial recognition deficits in AD patients (the same objective as the present study but from a neuropsychological perspective closer to psychology, without using markers). The authors in this study examined these deficits in an AD patient through three experiments (as opposed to a control group). The results effectively showed difficulties in recognizing patient faces, along with lower performance in accuracy and slower reaction times (Mazzi et al., 2020). At the same time, a general attenuation of the N170 component was also observed so that the finding suggests that the patient's results in the tests are due to difficulties in the structural coding of faces. However, significant modulation of the N400 component (related to the post-perceptual representation of familiar faces) was found, indicating implicit processing of face identity despite the lack of the reliable N170 component (Mazzi et al., 2020), so these findings support the hypothesis that deficits in facial recognition in the patient are gnosic in nature, This suggests the existence of indirect access to mnesic fingerprints and cannot be used consciously by the patient (Mazzi et al., 2020).

3. OBJECTIVES:

3.1. GENERAL OBJECTIVES

-Evaluate if there is a specific deficit in facial perception in people with Alzheimer's disease

3.2. SPECIFIC OBJECTIVES

- Schedule Benton's facial recognition test (revised) (BFRT-r) computerized with the program for experiments in psychology *PsychoPy*

- Analyze and compare facial visual perception abilities in a control group (C) and a group with Alzheimer's disease (AD)

- Analyze and compare the generic visual perception abilities in a control group (C) and one with Alzheimer's disease (AD)

3.3. HYPOTHESES

Hypothesis 1: The results obtained in facial perception tests will be significantly lower in the AD group than in the control group

Hypothesis 2: The results obtained in generic visual perception tests will not differ significantly between groups

4. METHOD:

4.1. DESIGN AND STUDY VARIABLES

- ➢ Variables:
  - Main dependent: facial perceptual deficit (results in BFRT-r)
  - Other dependent variables: results in visuoperceptual tests
  - Independent: diagnosis of Alzheimer's disease

The design of this study is a quasi-experimental design of group between subjects and transversal:

- ➢ Quasi-experimental: you do not have full control over the independent variable (condition of having or not having AD) since participants cannot be randomly assigned to these conditions but existing groups have had to be used.
- ➢ Group design between subjects: two cohorts of participants have been compared (one group with AD and one group without), this design allows to examine the differences between these groups in the different variables measured (facial perception and visuoperceptive generic).
- ➢ Transversal: the data were collected at a specific time and the differences between the groups at that time were compared

4.2. PARTICIPANTS

The participants in this study are ten people (8 women and 2 men), aged between 61 and 94 years. Five of them are diagnosed with Alzheimer's while the rest were in the control group.

The participants have been recruited from different environments: the *Centre Cívic Monitlivi* (Girona) and the *Centre de dia Onyar* (Girona) with whom an educational collaboration

document was signed together with the university (see annexes, section 9.5). In addition to these two centers, we contacted with Hospital Dr. Josep Trueta, Hospital Santa Caterina, Hospital de la Santa Creu i Sant Pau, Hospital de Palamós, the day center and residence ORPEA Girona, the Sanitas residence, the Pasqual Maragall foundation, among others, but no response was obtained or they did not want to cooperate.

Participants diagnosed with Alzheimer's have been diagnosed from their respective health centers by the referring geriatric neurologist in accordance with IWG-2 criteria and complying with those of the NINCDS-ADRDA, all were in a staging corresponding to a GDS 3-5, and have been administered, in addition (as in the control group) a cognitive screening test (the *Mini-mental State Examination*) in order to know the different profiles that make up the sample. In the case of the control participants, their cognitive impairments or undiagnosed profiles were ruled out both by the residence (administered cognitive impairment tests periodically every 6 months) and by the study with the MMSE, serving as a filter.

Regarding criteria for the inclusion of the sample, those in the clinical group had to have the diagnosis of AD, at a staging between GDS 3 and 5, while those in the control group only had to have a corresponding age range and without cognitive impairment. With regard to the exclusion criteria, however, all participants with vision problems (not corrected) (large % of the sample to which participation in the study was considered could not be accepted; in the civic center 10 people were rejected because of this deficit and in the day center 8), hallucinatory pathologies, subtypes of Alzheimer's dementia with specific visuoperceptive affectation, with comorbidities from other neurodegenerative diseases and medicated with medications indicated in the leaflet side effects on vision were not included in the study. Also, as part of the exclusion criteria, visual deficits are included, medication that can alter eyesight and have cognitive impairment while in the control group of the study.

Table 3 below describes the study sample.

Table 3

Demographic data of patients with Alzheimer's (AD) and controls (C) included in the study. Age, gender, years of education (Educ.), GDS phase, MMSE score, BFRT-r score, score on the VOSP battery number location test, RBANS battery line orientation test score (OL), exam time, place of origin (Natural), main profession, manual laterality, main language (CAT: Catalan; SPAN: Spanish) and place of evaluation and recruitment.

Code	Group (AD/C)	Age	Sex	Educ.	GDS	MMSE	BFRT-r	VOSP	OL	Time
PP01	C	77	F	3	1	29	41	8	15	30’
PP02	C	80	F	3	1	27	36	10	14	32’
PP03	C	84	F	6	1	30	37	8	17	30’
PP04	AD	61	F	6	3	24	31	5	17	34’
PP05	AD	84	F	1	5	21	31	10	9	31’
PP06	AD	94	F	2	3	21	24	3	17	30’
PP07	AD	81	M	3	4	20	28	3	17	36’
PP08	AD	82	F	1	5	22	31	10	20	32’
PP09	C	74	F	2	1	30	37	8	20	30’
PP10	C	77	M	5	1	29	42	10	20	31’

Natural	Profession	Laterality	Language	Studio place
Spain	Cleaning staff	Ambidextrous	SPAN	Centre Cívic Montilivi
Spain	Housewife	Right	CAT	Centre Cívic Montilivi

Spain	Master	Right	SPAN	Centre Cívic Montilivi
Spain	Doctor	Right	SPAN	Centre Cívic Montilivi
Spain	Cleaning staff	Right	SPAN	Centre de dia Onyar
Spain	Housewife	Right	SPAN	Centre de dia Onyar
Spain	Vendor	Right	CAT	Centre de dia Onyar
Spain	Farmer	Ambidextrous	SPAN	Centre de dia Onyar
Spain	Cleaning staff	Right	SPAN	Centre Cívic Montilivi
Spain	Vendor	Right	SPAN	Centre Cívic Montilivi

Note. The numbers of the years of education correspond to an ordinal numbering of different categorical measures corresponding to 1 with "out of school", 2 with "acquisition of literacy", 3 with "primary studies", 4 with "elementary baccalaureate, EGB, ESO or BUP", 5 with "middle studies" and 6 with "higher education".

4.3. INSTRUMENTS

➢ *Benton Facial Recognition Test - revised* (BFRT-r):

The original Benton Facial Recognition Test (BFRT) is a paper-and-pen task consisting of assessing facial perception skills in neurological, clinical and psychiatric conditions (Murray et al., 2021); it is a simple test to perform consisting of observing a series of sheets and looking for objective faces among 6 possible face responses that appear just below it, through visual comparison. The images show grayscale face images of different people with various expressions, angles and illuminations. The short version contains 27 items and the long version 54; In both cases, the test is divided into two parts: a first in which the

participant must recognize an objective face among 6 photographs presented below, and a second in which instead of finding a single corresponding face they must find 3.

More recently in 2018, this test was computerized and the BFRT-c appeared, maintaining the same operation and instructions of the test, but now computerized. Finally, Murray et al. (2021) presented a revised version of this latest computerized version: the basic paradigm is maintained but using new, higher quality stimuli that reflect theoretical advances in the field, better internal reliability and content validity, and when comparing the results obtained with BFRT-c in patients with prosopagnosia, BFRT-r showed better sensitivity in diagnostic detection (Murray et al., 2021).

This instrument, therefore, will be used to evaluate the facial perception skills of the participants.

➢ RBANS battery line orientation test

RBANS (*Repeatable Battery for the Assessment of Neuropsychological Status*) developed by Muntal et al. in 1998 and adapted to Spanish in 2011, is a battery commonly used in clinical practice in the evaluation of different cognitive functions. It is a multidimensional neuropsychological evaluation method that has 12 different tasks (one evaluates memory, another language, another constructive praxis, etc.). In place number 4, there is the test called line orientation, consisting of observing a drawing with a range of 13 lines with a number associated with each one from 1 to 13 numbering them. The test consists of a notebook with 10 plates where this drawing appears successively at the top, and below it only two lines of the drawing above appear drawn (without the corresponding number). Participants must point with their finger or say the numbers aloud of which lines correspond to the drawing above.

In this way, this test evaluates visuoperception and visuospatial perception, exploring the abilities to estimate the spatial relationships between the lines presented on each sheet. This test, together with the one exposed below, will be responsible for contrasting the data obtained from the participants in relation to facial visuoperceptive skills in order to study where the possible deficit lies.

➢ VOSP battery number location test

The *Visual Object Space Perception battery* is a battery of tests developed by Warrington and James in 91 (and since then has not been modified) to explore the perception of objects and space according to the Warrington model, in which 3 subtypes of deficit in object perception

are recognized: visual sensory discrimination disorder, aperceptive agnosia and associative agnosia (Calvo et al., 2013). Within this battery, two parts are divided (A and B) with 4 subtests in each of them. In part A (object perception) there is the subtest of incomplete letters, silhouettes, object decision and progressive silhouettes; In section B (spatial perception) we find the subtest of counting points, position discrimination, location of numbers and analysis of cubes.

In this study, the number location subtest is used: this consists of 10 sheets on paper with two centered boxes (one on top of the other, spaced a few centimeters apart), the upper one full of randomly distributed numbers, and the lower one with a black dot located randomly inside the box. The participant is asked, if she mentally moved the box with the black dot above the box full of numbers, which number would cover the black point. On each sheet, the numbers and location of the black point vary.

This test, together with the previous line orientation test, are the two (generic) visuoperception tests used in the study to compare the results obtained in the facial perception test by the sample.

➢ *Mini-Mental State Examination* (MMSE):

The MMSE is a short (approximately 5-10 minutes) and quantitative test, developed by Folstein in 1975; It is the most well-known and widely used cognitive tracing test today. This test is used to evaluate cognitive function in adults and allows to detect cognitive impairment or dementia. It explores a total of eleven cognitive areas: temporal orientation, fixation, attention and calculation, memory, nomination, repetition, comprehension, reading, writing and drawing.

The score ranges from 0 to 30: 0 to 24 marks probable cognitive impairment, and 25 to 30 without impairment.

This instrument will be used in the study as part of the neuropsychological evaluation forming the cognitive *screening* part in order to have an orientation of the profile of the participants. Obviously, it is expected that patients with Alzheimer's mark a score lower than 25 and controls higher than 24, according to the inclusion criteria of the study and with the logic of the participants' diagnoses.

4.4. PROCEDURE

➢ Bibliographic search:

First of all, a bibliographic search was carried out in order to know all the updated information that exists on Alzheimer's disease, facial perception, and the studies that have been done in relation to facial recognition in both AD patients or other pathologies.

The research was carried out in the following databases: *APA PsycInfo, PubMed, BioMed* and *Scopus*. The research was mostly done in English and sometimes in Spanish. The main keywords of the searches were "Facial perception", "Alzheimer's disease", "facial perception in Alzheimer's disease", "faces stimuli in perception", "face recognition experiments alzheimer's disease", among others.

➢ Paradigm:

The paradigm finally chosen for the study was the *Benton Face Recognition Test revised* (BFRT-r), a test already explained above (see section 4.3). In this new version of the BFRT, as indicated by the authors (Murray et al., 2022) the task contained 22 tests / screens (always presented in the same order and with an interval between stimuli of about 800 ms) with 7 images per screen of faces of Caucasian men (between 18 and 34 years M = 21.9 years and SD = 3.2); only images of men were used due to gender bias demonstrated by the recognition of female but non-male faces (Herlitz & Lovén, 2013; Lovén et al., 2011; Murray et al., 2022). These images were taken within a laboratory and/or were pre-existing images provided by the participant that had been taken in the space of a single year (Murray et al., 2022), in order to avoid any major effects of aging. However, images of participants with heavy facial hair obscuring the image were discarded. Some inter-image variations were given by light condition, skin spots or hairstyle, but the photographs were not manipulated and all consisted of a sufficiently high quality (no less than 96 DPI). The images, however, were presented in a grayscale in order to avoid the ceiling effects in the typical population that could be achieved with color images (Murray et al., 2022).

For each *trial* , a single target person was used that was not reused for any other or as a distractor, nor was any distracting image repeated within the same matrix.

The target faces to ensure good match in search were not cropped to exclude any part of the head, hair, or ears, but the other 6 images were cropped around the hairline (Murray et al., 2022).

According to Murray et al. (2022), in the first 12 trials, all faces appear from frontal viewpoints; In the last 10, the faces also appear frontal but in this case with a little more naturalness (very little rotation, about 10-30 degrees); only 7 images appear with a larger rotation of about 45 degrees (N = 7).

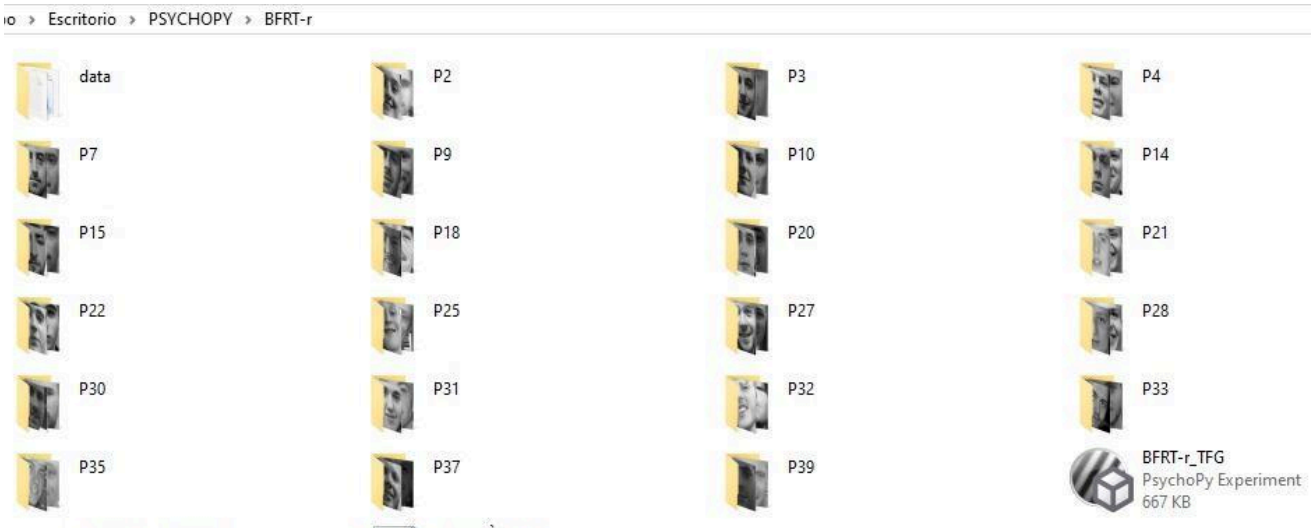
PARADIGM CONSTRUCTION:

In order to have a deeper immersion in a real and own research study, this test was personally computerized by programming it with the *PsychoPy* program, an open source software written in Python used in research in experimental psychology and neuroscience.

First of all, a folder was created with all documents related to the experiment. This contained: all the folders with the test stimuli (grouped as the authors had done), a folder called "date" where the program generated all the outputs of each time a participant performed the test with all the information of their participation, and the experiment itself "BFRT-r TFG". See figure 20.

Figure 20

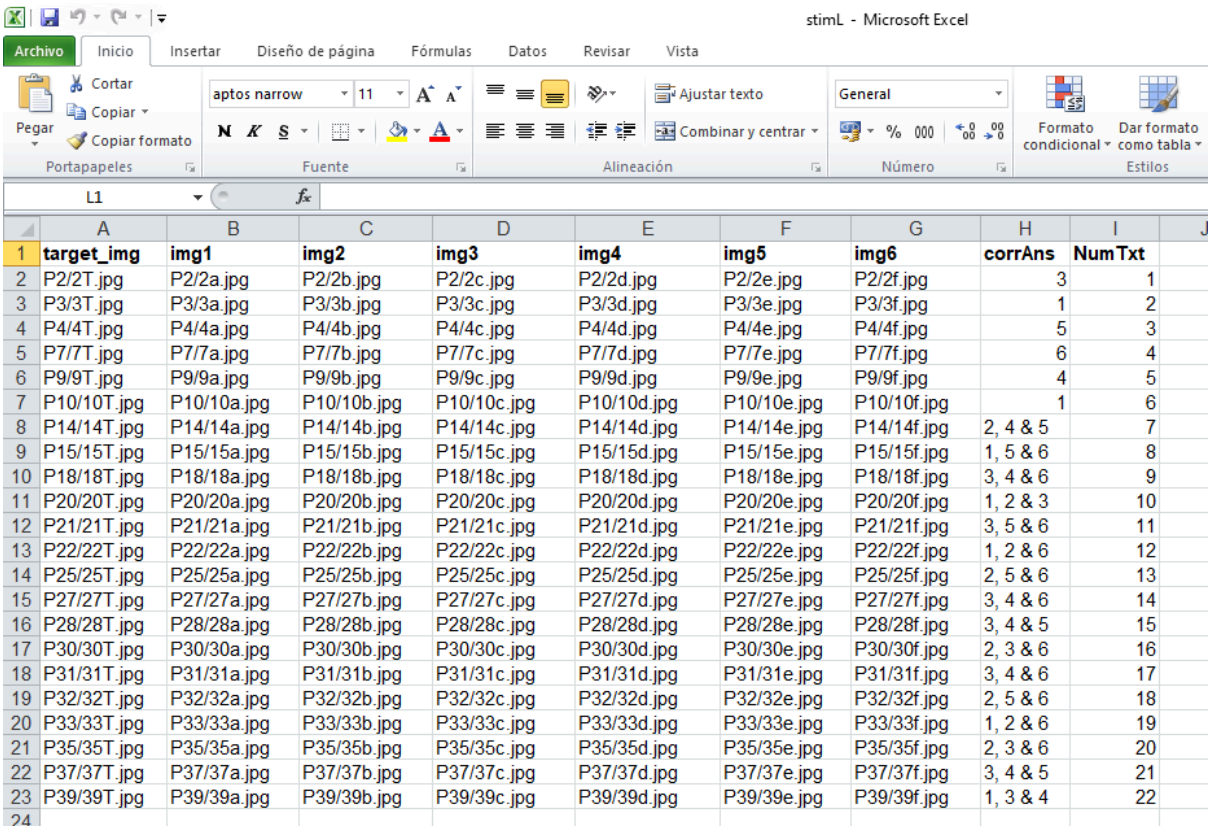
Organization of experiment folders and documents



Then, an excel was created (see figure 21) with all the stimuli that the experiment would contain, with which later, when the test was scheduled, all the images and data on which image is the correct answer and to which *trial* they correspond (from 1 to 22) would be extracted.

Figure 21

Excel document with the necessary information to schedule the test with PsychoPy



stimL - Microsoft Excel

	A	B	C	D	E	F	G	H	I
1	target_img	img1	img2	img3	img4	img5	img6	corrAns	NumTxt
2	P2/2T.jpg	P2/2a.jpg	P2/2b.jpg	P2/2c.jpg	P2/2d.jpg	P2/2e.jpg	P2/2f.jpg	3	1
3	P3/3T.jpg	P3/3a.jpg	P3/3b.jpg	P3/3c.jpg	P3/3d.jpg	P3/3e.jpg	P3/3f.jpg	1	2
4	P4/4T.jpg	P4/4a.jpg	P4/4b.jpg	P4/4c.jpg	P4/4d.jpg	P4/4e.jpg	P4/4f.jpg	5	3
5	P7/7T.jpg	P7/7a.jpg	P7/7b.jpg	P7/7c.jpg	P7/7d.jpg	P7/7e.jpg	P7/7f.jpg	6	4
6	P9/9T.jpg	P9/9a.jpg	P9/9b.jpg	P9/9c.jpg	P9/9d.jpg	P9/9e.jpg	P9/9f.jpg	4	5
7	P10/10T.jpg	P10/10a.jpg	P10/10b.jpg	P10/10c.jpg	P10/10d.jpg	P10/10e.jpg	P10/10f.jpg	1	6
8	P14/14T.jpg	P14/14a.jpg	P14/14b.jpg	P14/14c.jpg	P14/14d.jpg	P14/14e.jpg	P14/14f.jpg	2, 4 & 5	7
9	P15/15T.jpg	P15/15a.jpg	P15/15b.jpg	P15/15c.jpg	P15/15d.jpg	P15/15e.jpg	P15/15f.jpg	1, 5 & 6	8
10	P18/18T.jpg	P18/18a.jpg	P18/18b.jpg	P18/18c.jpg	P18/18d.jpg	P18/18e.jpg	P18/18f.jpg	3, 4 & 6	9
11	P20/20T.jpg	P20/20a.jpg	P20/20b.jpg	P20/20c.jpg	P20/20d.jpg	P20/20e.jpg	P20/20f.jpg	1, 2 & 3	10
12	P21/21T.jpg	P21/21a.jpg	P21/21b.jpg	P21/21c.jpg	P21/21d.jpg	P21/21e.jpg	P21/21f.jpg	3, 5 & 6	11
13	P22/22T.jpg	P22/22a.jpg	P22/22b.jpg	P22/22c.jpg	P22/22d.jpg	P22/22e.jpg	P22/22f.jpg	1, 2 & 6	12
14	P25/25T.jpg	P25/25a.jpg	P25/25b.jpg	P25/25c.jpg	P25/25d.jpg	P25/25e.jpg	P25/25f.jpg	2, 5 & 6	13
15	P27/27T.jpg	P27/27a.jpg	P27/27b.jpg	P27/27c.jpg	P27/27d.jpg	P27/27e.jpg	P27/27f.jpg	3, 4 & 6	14
16	P28/28T.jpg	P28/28a.jpg	P28/28b.jpg	P28/28c.jpg	P28/28d.jpg	P28/28e.jpg	P28/28f.jpg	3, 4 & 5	15
17	P30/30T.jpg	P30/30a.jpg	P30/30b.jpg	P30/30c.jpg	P30/30d.jpg	P30/30e.jpg	P30/30f.jpg	2, 3 & 6	16
18	P31/31T.jpg	P31/31a.jpg	P31/31b.jpg	P31/31c.jpg	P31/31d.jpg	P31/31e.jpg	P31/31f.jpg	3, 4 & 6	17
19	P32/32T.jpg	P32/32a.jpg	P32/32b.jpg	P32/32c.jpg	P32/32d.jpg	P32/32e.jpg	P32/32f.jpg	2, 5 & 6	18
20	P33/33T.jpg	P33/33a.jpg	P33/33b.jpg	P33/33c.jpg	P33/33d.jpg	P33/33e.jpg	P33/33f.jpg	1, 2 & 6	19
21	P35/35T.jpg	P35/35a.jpg	P35/35b.jpg	P35/35c.jpg	P35/35d.jpg	P35/35e.jpg	P35/35f.jpg	2, 3 & 6	20
22	P37/37T.jpg	P37/37a.jpg	P37/37b.jpg	P37/37c.jpg	P37/37d.jpg	P37/37e.jpg	P37/37f.jpg	3, 4 & 5	21
23	P39/39T.jpg	P39/39a.jpg	P39/39b.jpg	P39/39c.jpg	P39/39d.jpg	P39/39e.jpg	P39/39f.jpg	1, 3 & 4	22
24									

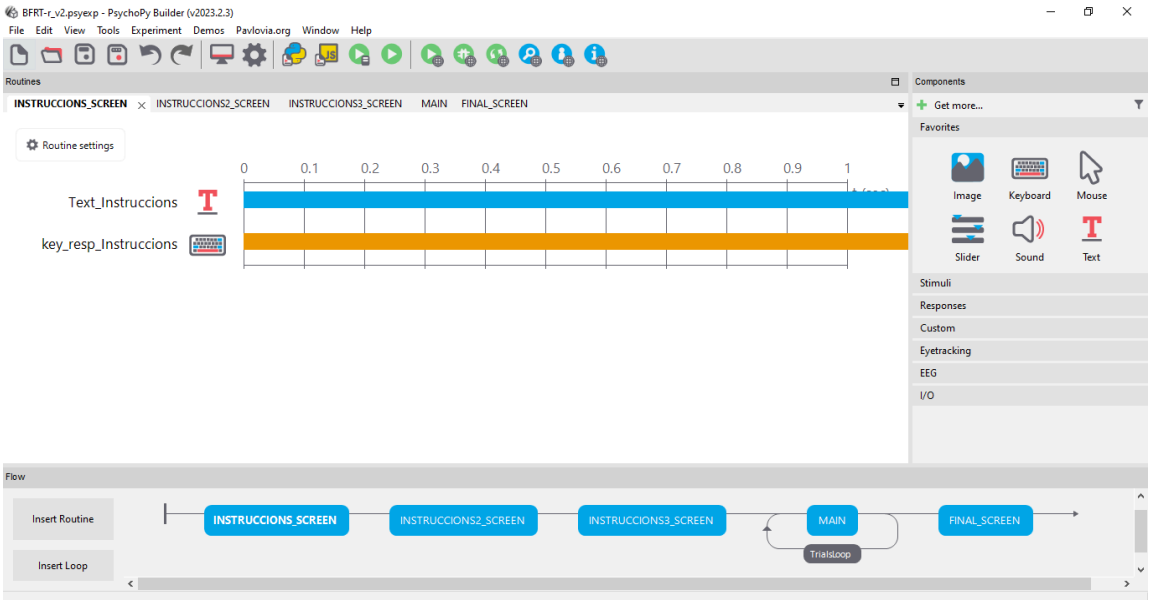
Note. Column A: corresponds to the target image of each *trial* (1-22), first the name of the folder where it is located, and then separated by a "/" is put the name of the photograph; Column B-G: correspond to the 6 images of each *trial* (1-22) in the order that the authors explained; Column H: corresponds to the correct answer or answers of each *trial* (1-22); Column I: corresponds to the *trial number*.

Then the experiment with the *PsychoPy Builder began to be built*:

- 5 different trials were created: 3 of them for instruction screens, another as the main one (which, together with an associated loop, would compose the experiment), and the last as the final screen. Figure 22 shows the main screen of the program where all of them can be seen.

Figure 22

Main screen of the program

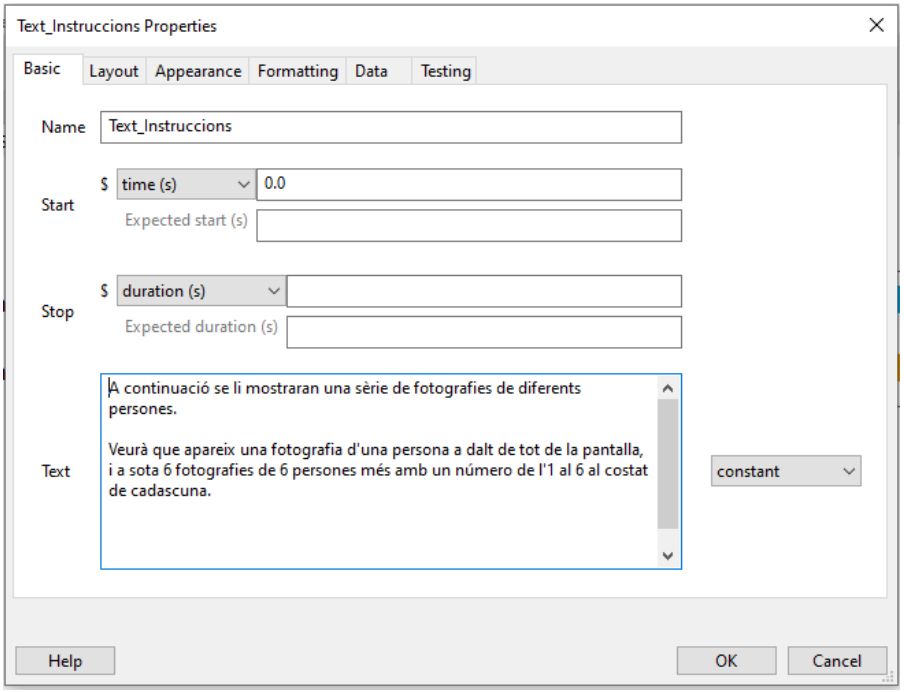


- Instruction screens:

INSTRUCTION SCREEN 1 (figure 23 and 24):

Figure 23

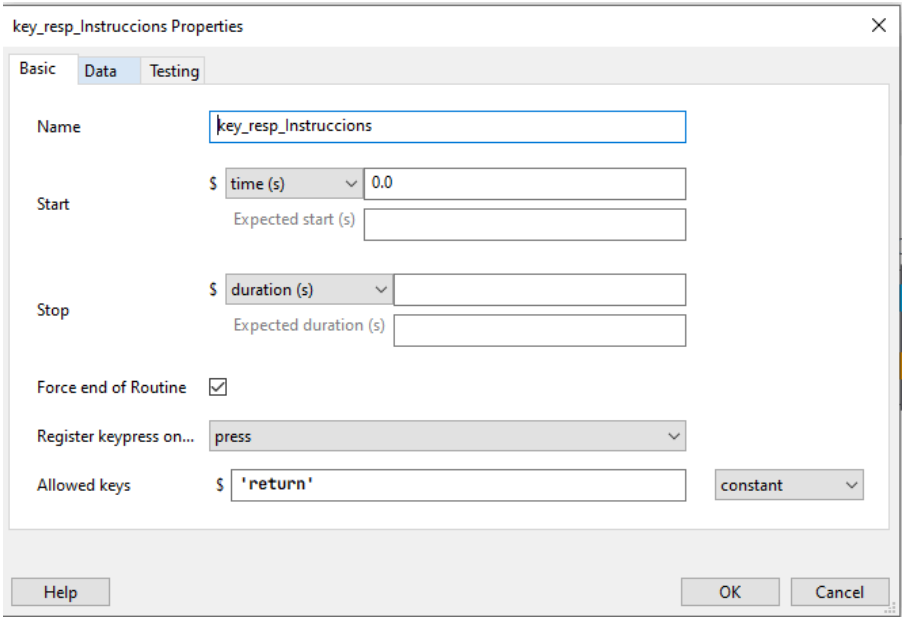
Instruction "Text_Instruccions"



Note. It is the instruction that contains the instruction text on the first screen, which appears from the second 0.0 and without an end instruction. In addition, the text is in Catalan because the experiment was carried out in Girona.

Figure 24

Instruction "key_resp_instruccions"



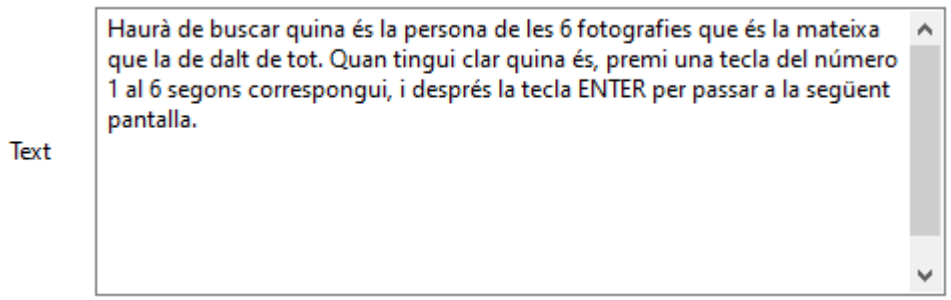
Note. It is the keyboard instruction that starts at the second 0.0 in which, when the person finishes reading the text, pressing the "Enter" ('return') key, the trial ends ('*force end of routine*').

INSTRUCTION SCREEN 2:

The operation and instructions used of PsychoPy are the same, but with the second part of the instructional text for the participant (see figure 25).

Figure 25

Second part of the test instructions

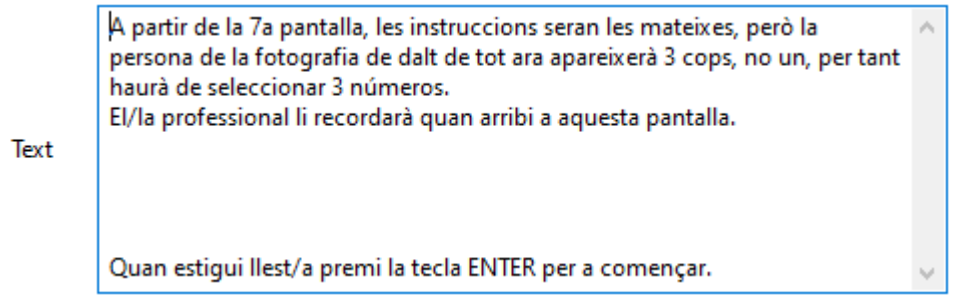


INSTRUCTION SCREEN 3:

The operation and instructions used of PsychoPy are the same, but with the third part of the instructional text for the participant (see figure 26).

Figure 26

Part Three of the Test Instructions



- Final screen:

The final screen works exactly the same as the first three instructions, but with the difference in the text that appears, thanking the participant for their participation.

- Main screen ("*Main*") (see figure 27 and 28):

Figure 27

'MAIN' main trial instructions

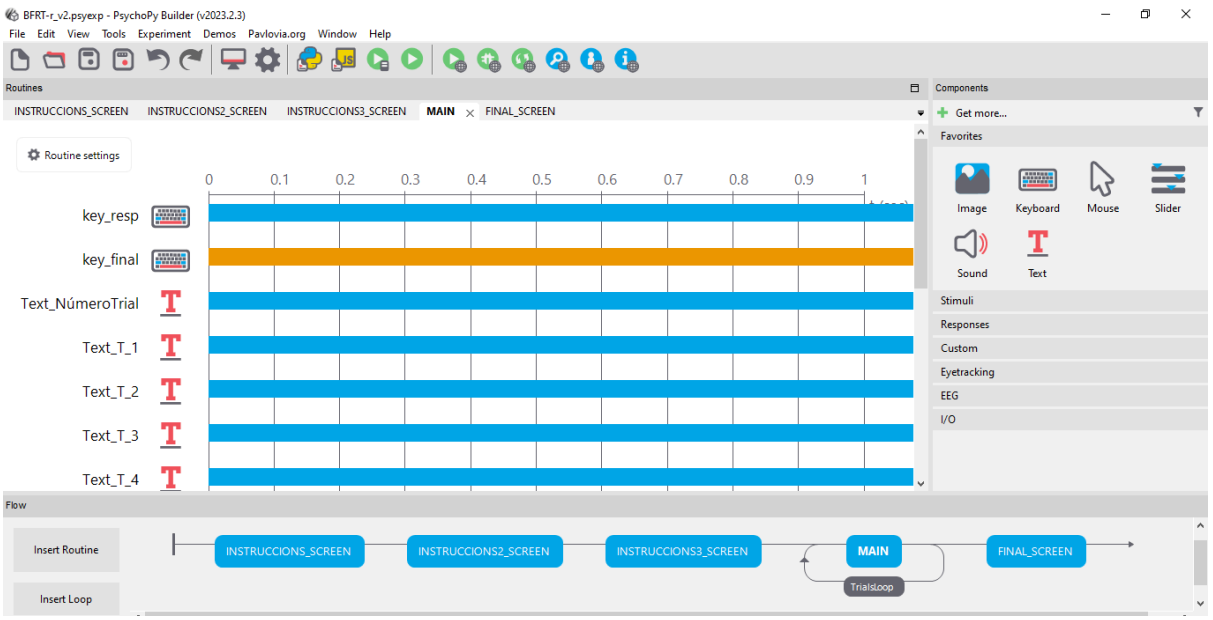
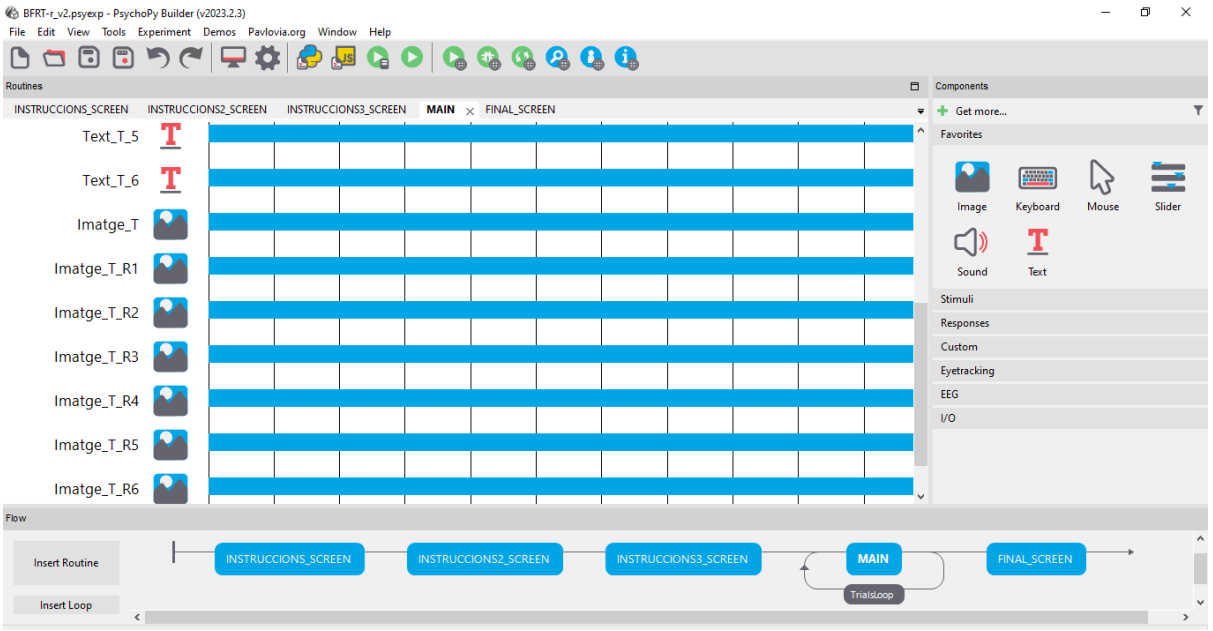


Figure 28

'MAIN' main trial instructions (2)



First of all there are two keyboard instructions: "key_resp" and "key_final". The first corresponds to the instruction that will allow the participant to select on the keyboard the answer that they consider correct on each screen (numbers from 1 to 6 that will correspond to each answer image) and that when pressing the keys the end of the routine is not forced, since from trial 7 there are three correct answers, not just one (see figure 29). In addition, on the "date" tab, the excel column created earlier on the correct answer will be indicated in the correct answer section, so that it will be saved in the subsequent output that generates which keys the person has selected and whether these were correct or not (see figure 30). Thus, with the second keyboard instruction, when the person has already pressed their answer, it will allow you to finish the routine and move to the next screen by pressing the "Enter" key.

Figure 29

Answer options keyboard instruction: keys 1-6

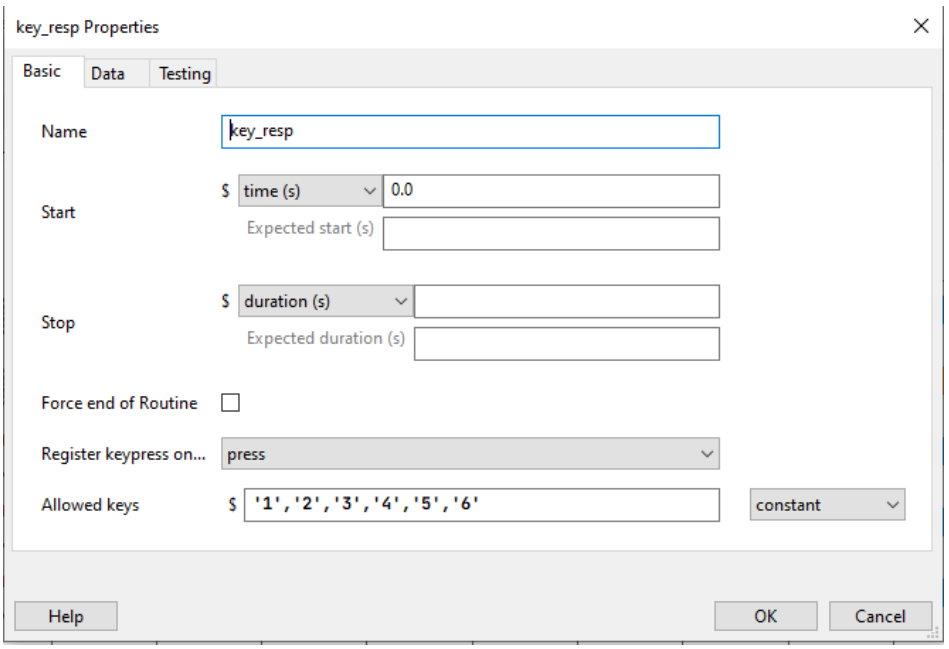
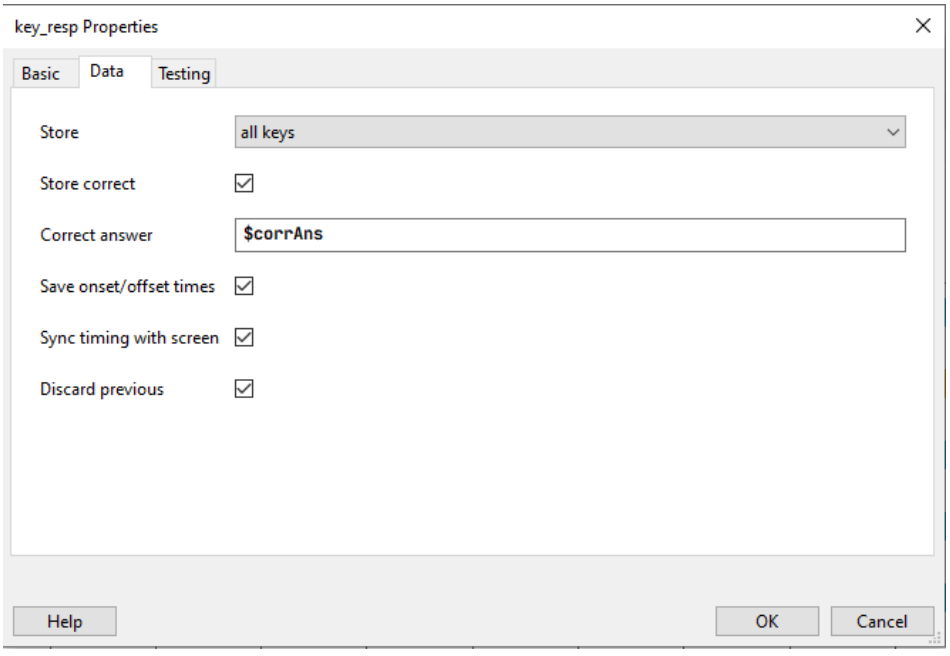


Figure 30

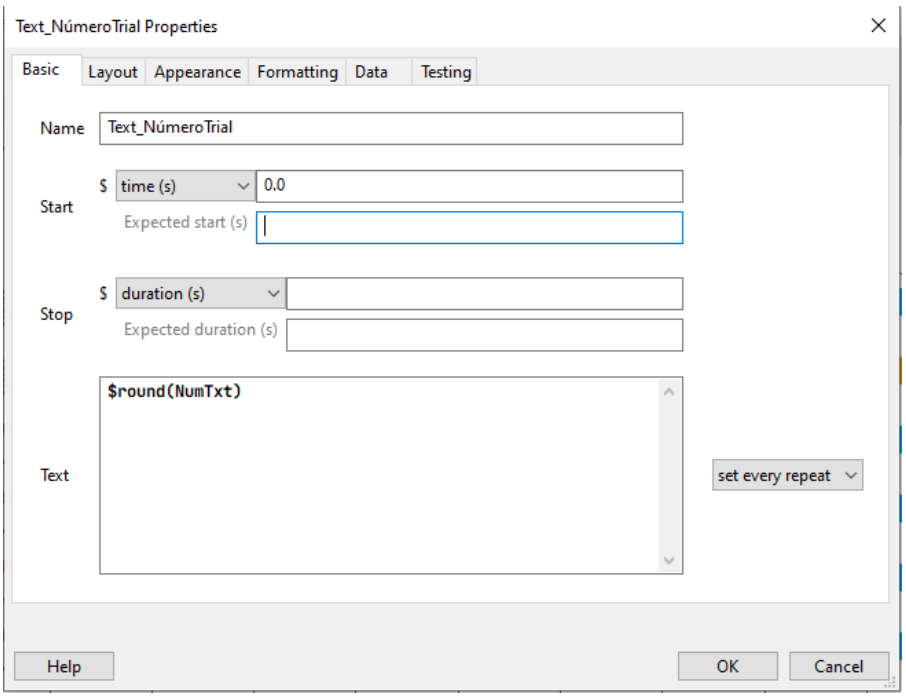
Answer Options Keyboard Instruction: Recording the Correct Answer



Then, the instruction "Text_NúmeroTrial" refers to the screen number (total 22) in which the person will be at any given time, which will appear at the top left. In this case, the reference is also put in the excel column, with an instruction to rotate on each screen (each time a number, sequentially from 1 to 22). See figure 31.

Figure 31

Display reference number instruction in the upper left margin



Below come all the text instructions, which refer to the numbers that will appear on screen from 1 to 6 accompanying each photo answer option. Thus, there are 6 such instructions, one for each number. Figure 32 shows the example of the number 1, with the only difference that, in each number, there is a position (x, y) on the specific screen, so that they accompany each image next to it (see figure 33).

The screen resolution determined for the experiment is 1366 x 768 pixels, according to the author's computer screen (so when you want to administer the experiment from another computer, so that the entire distribution of the square screen is left, the resolution must be modified to this number of pixels). Thus, calculations were made to square all the elements that would appear on each screen, and, in this case, in terms of the numbers of the images they were distributed as follows in position "(x,y)" and in "normal" units: the number 1 at (-0.33, -0.15), the 2 at (0.17, -0.15), 3 at (0.67, -0.15), 4 at (-0.33, -0.87), 5 at (0.17, -0.87) and 6 at (0.67, -0.87).

Figure 32

Stimulus numbering text instruction

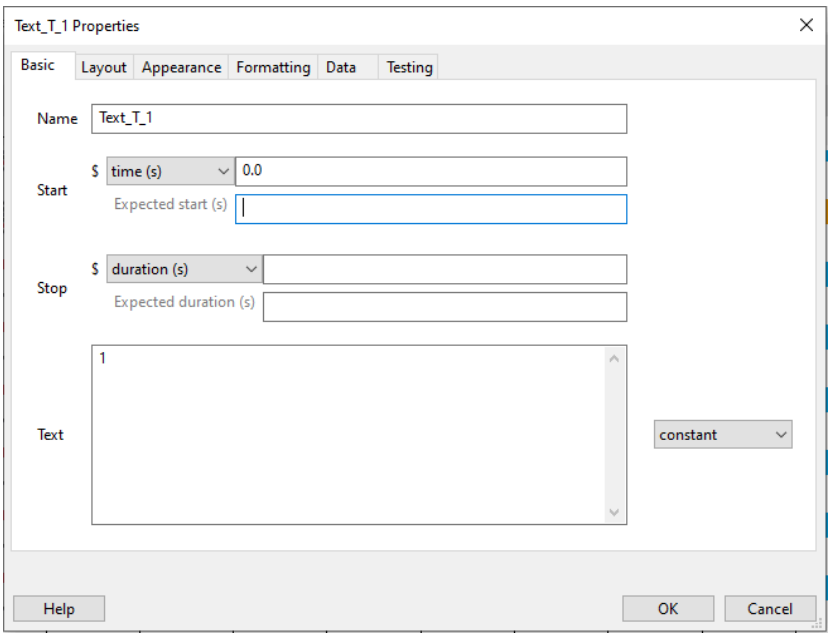
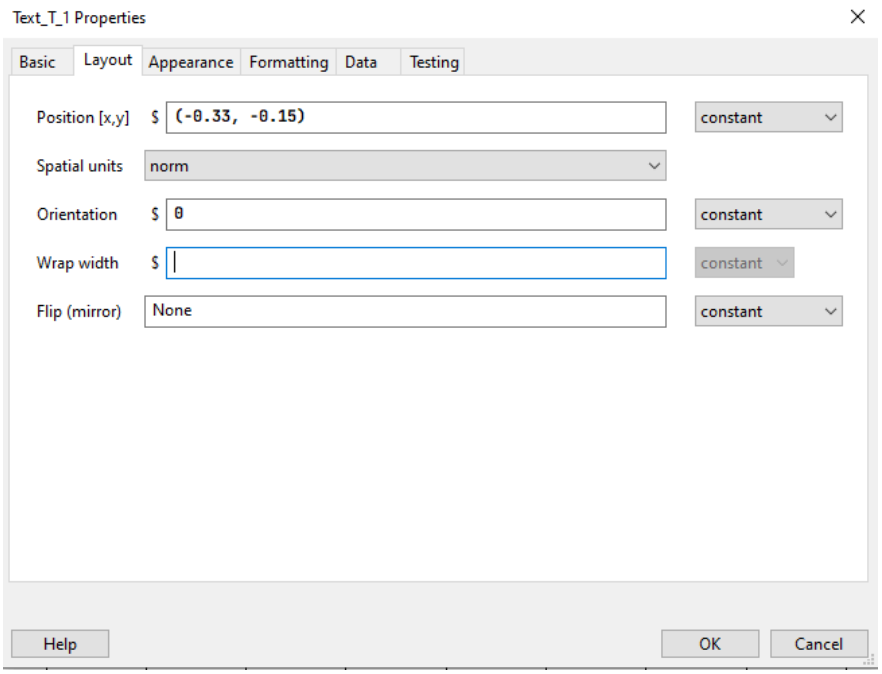


Figure 33

Text instruction for stimulus numbering: position and spatial units



Then come all the stimulus instructions (the pictures of faces). First of all, there is the "target image", the target image of each screen, and then the other 6 which are the answer options. These (accompanied by a number from 1 to 6 except the target one) are also positioned at a

specific orientation of the screen according to the calculations made so that they are all framed and at the same distance.

As for the size of the images, faithful to what the authors indicated, the *target* measures 156 x 232 pixels, and the other 6 answer options measure 153 x 200 pixels. As for the position, also after making the calculations, they are as follows in position "(x,y)" and in units of pixels: target at (0, 288), 1 at (-341.5, 40), 2 at (0, 40), 3 at (341.5, 40), 4 at (-341.5, -232), 5 at (0, -232) and 6 at (341.5, -232).

In addition, in order to know which images should appear on the screen, once again excel connects to the program and asks to take the reference images that appear in the column in question (depending on whether it is the *target* image, 1, 2...).

An example of how it is programmed is shown in Figures 34 and 35.

Figure 34

Instruction for connecting image stimuli to appear with the excel file where they are referenced

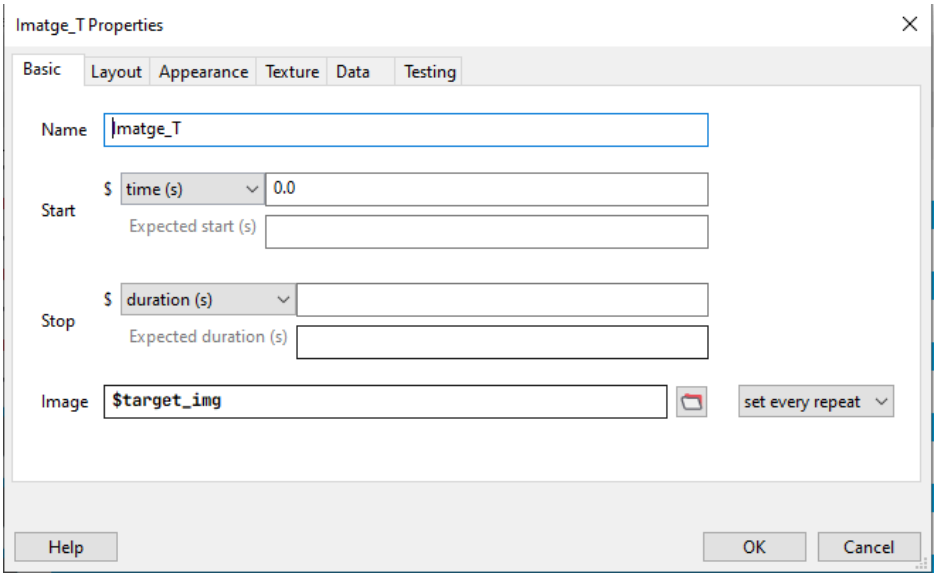
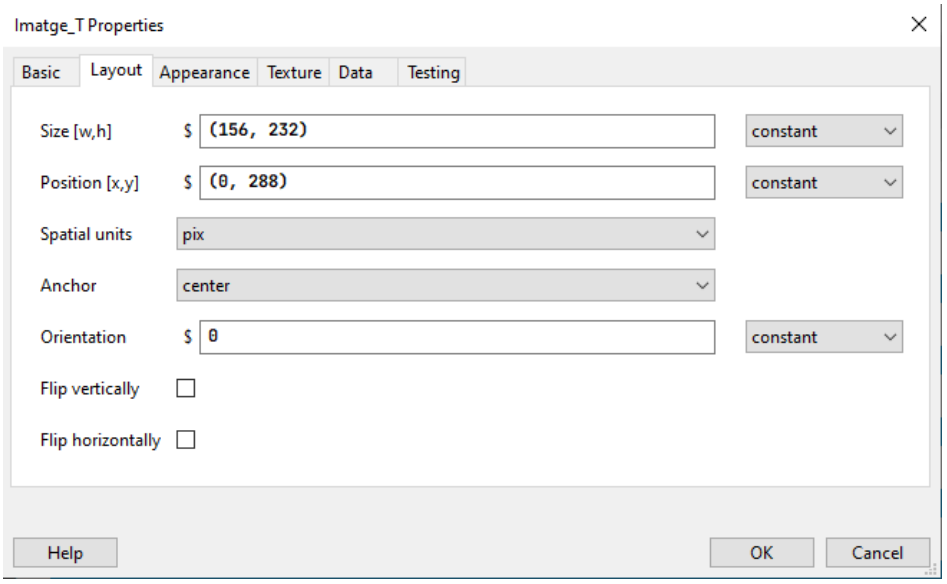


Figure 35

Instruction to connect image stimuli to appear with the excel file where they are referenced: size and position of the image



In this case, none of the instructions also have an end time, but all of them end with the keyboard instruction mentioned at the beginning when selecting the "Enter" key.

- Loop (figure 36):

Finally, a loop instruction has also been created in which its task is, that after reproducing the main routine of the previous section, it is repeated in a loop up to 22 times (one for each screen). Thus, the first time the routine is executed, all the images (and associated information, such as the correct answers from that screen) on screen number 1 will appear. The second time, those on screen number 2, and so on until reaching screen 22. Therefore, the type of loop selected is sequential, and previously when the main routine in which this loop will act has been programmed, in each image (the 7 in total) the option "*set every repeat*" has been selected next to the excel reference, so that each time the loop starts, it is moved to the next image in the specific column of excel. All this can be seen in Figures 37 and 38.

Figure 36

Main trial loop

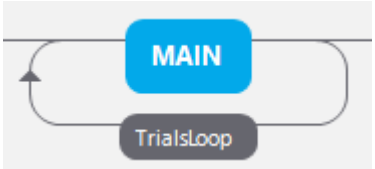
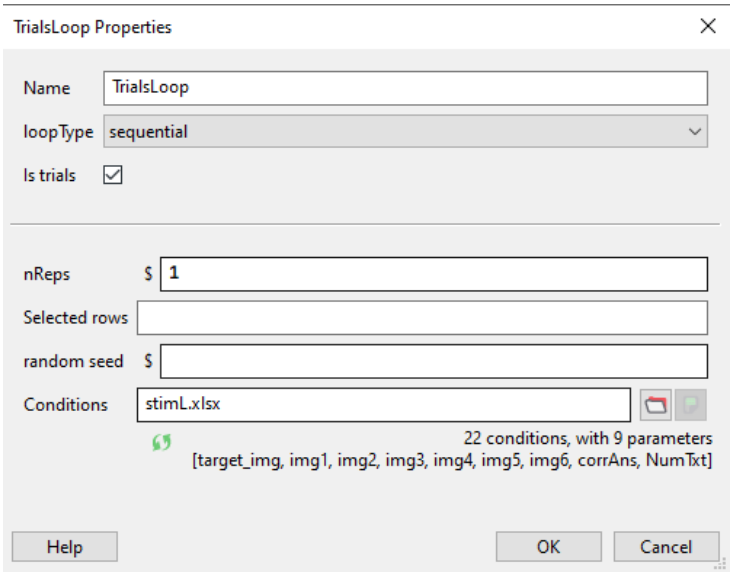


Figure 37

Programming the loop connected with the excellence of stimuli



Note. In the image you can see how we talk about 22 conditions with 9 parameters: these 22 conditions are the 22 groups of stimuli (the folders containing the 7 photographs of each screen; 1 lens and 6 answers) entered in rows in the Excel document, and the 9 parameters entered in columns (information on which photo will correspond to the target image, which is the one that will correspond to the image located at number 1 (...) until 6, and finally two columns about what is the correct answer on each screen and on the number (1-22) that will appear at the top left of the screen to indicate in which screen number they are).

Figure 38

Reference instruction in the excel column with the stimuli where the target images are located

Thus, after all this, figures 39, 40, 41 and 42 show how the text and instruction screens look, and an example of one of the 22 screens of the test.

Figure 39

Instruction screen 1

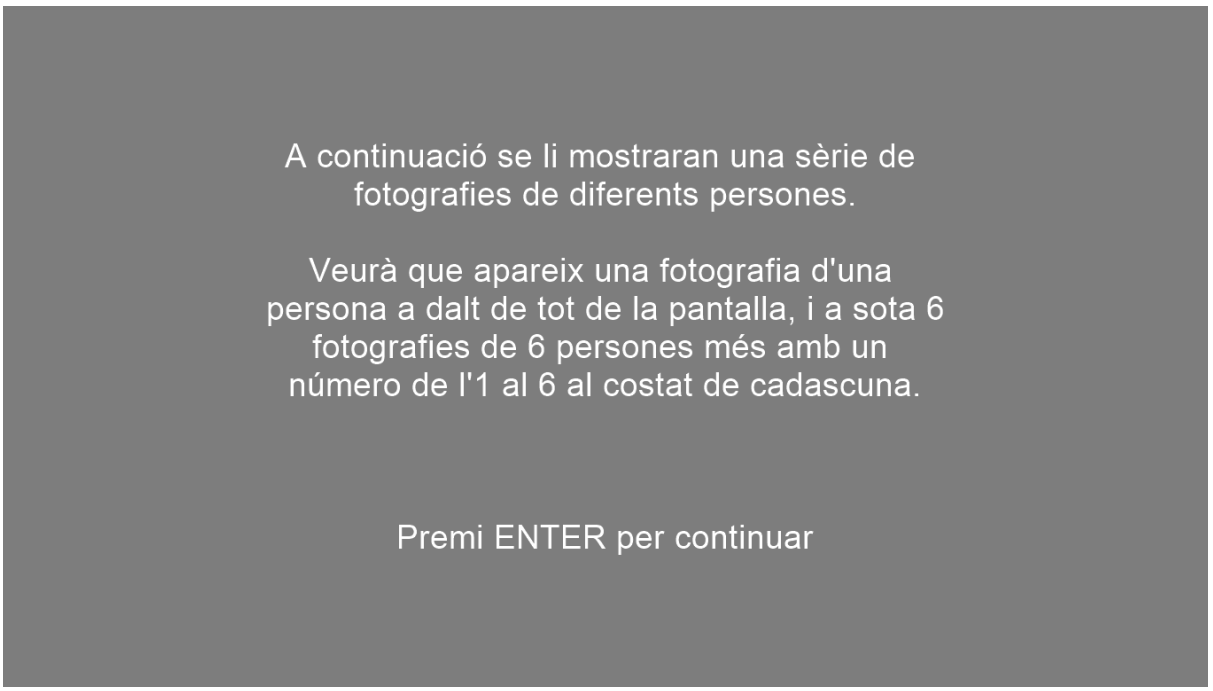


Figure 40

Instruction screen 2

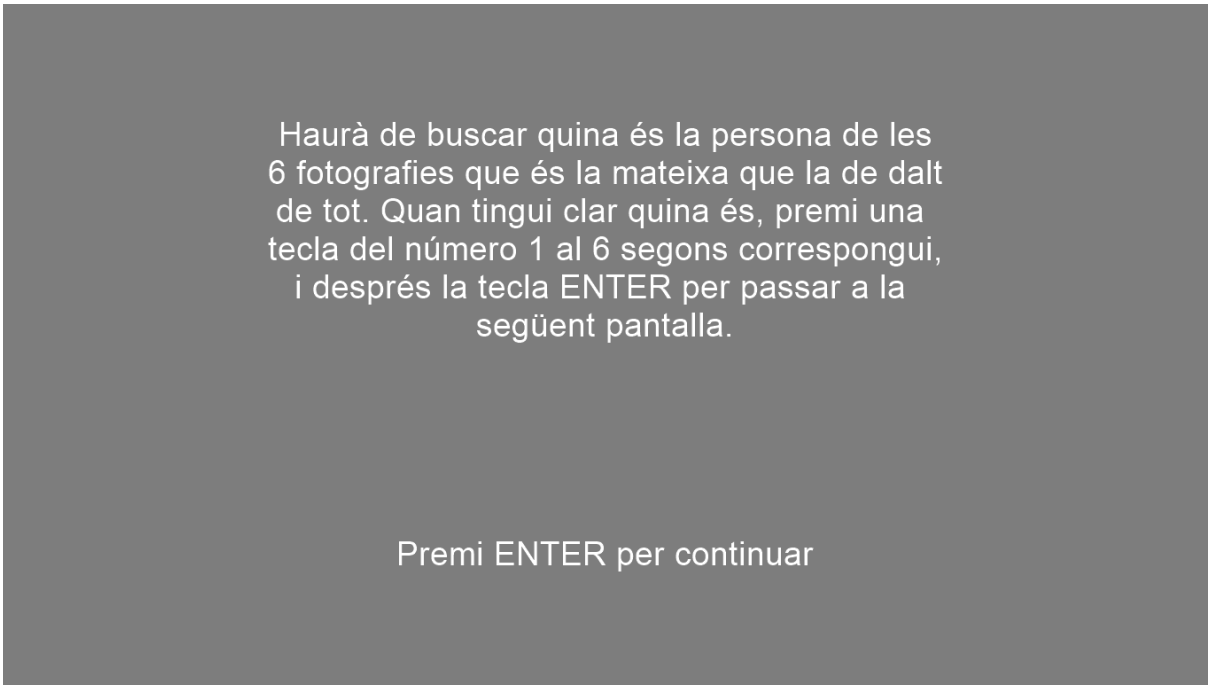


Figure 41

Instruction screen 3

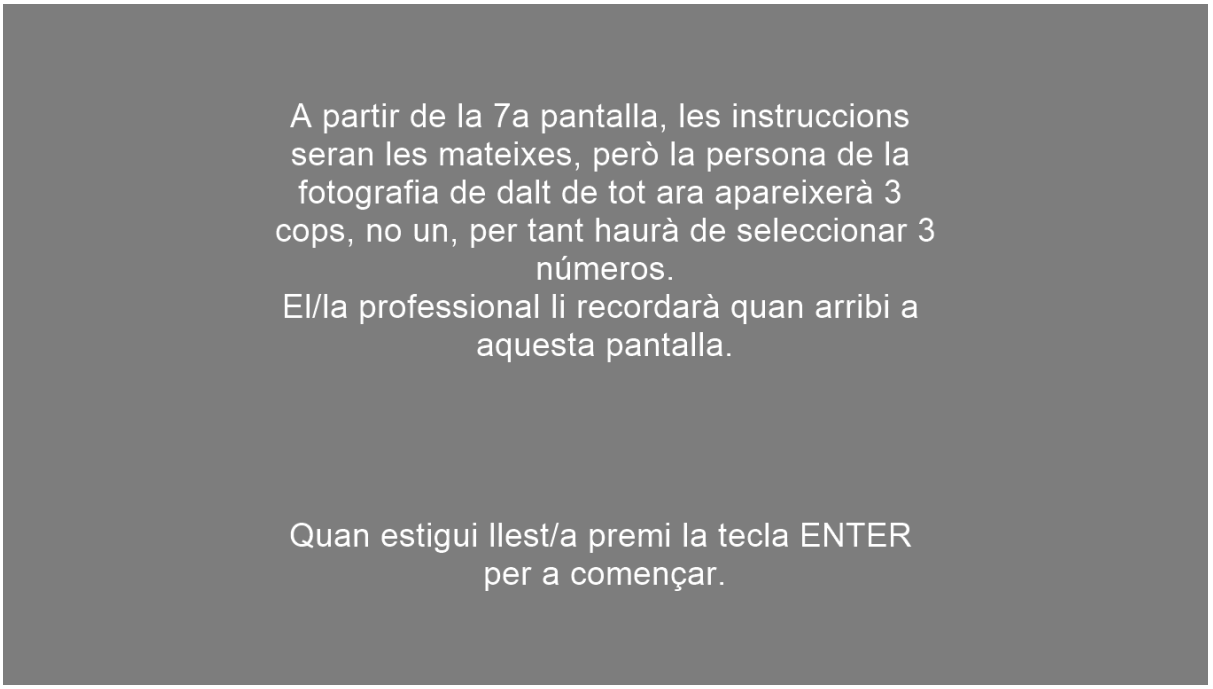


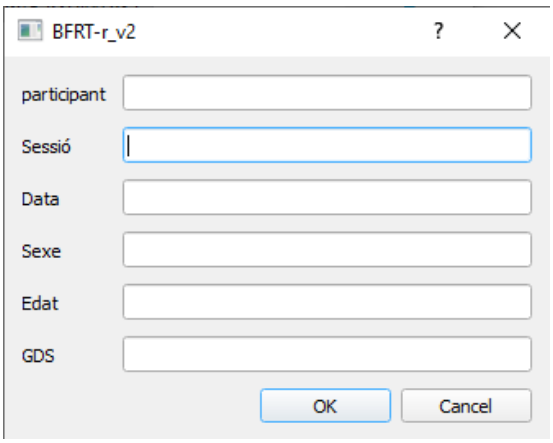
Figure 42

First screen of the test

In penultimate place, when the participants go to start the task, a small screen has also been programmed before the start of the test in which some data is collected in order to identify, after the data *output*, which participant it is -among others-. See figure 43.

Figure 43

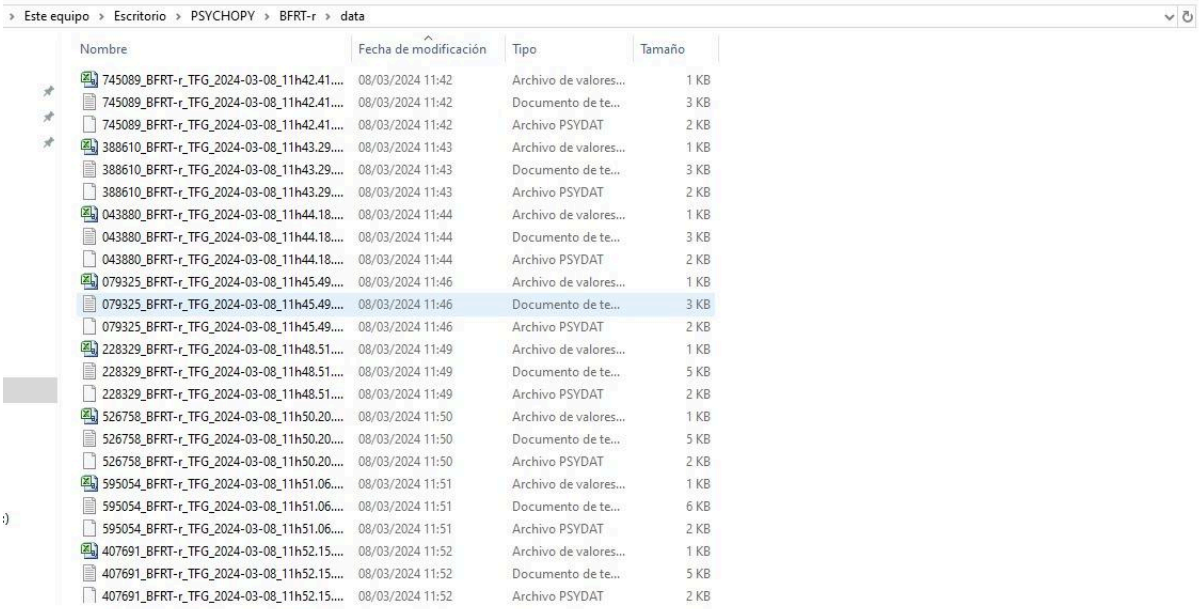
Participant data window



Finally, when the participants have already completed the task, the program generates on each occasion an output of the data in excel (see figure 44).

Figure 44

Output files with the results obtained that the program generates automatically



In the excel document generated for each participant, all the data that the program has on the entire execution are collected, but the most important part for the study are the two columns in which it is indicated which were the correct answers, and next to it if they have got it right or not (a 0 when an answer is not right, and a point - up to 3 - for each correct answer). See figure 45.

Figure 45

Excel columns that present participants' responses and points obtained

BC	BD
key_resp.ke	key_resp.corr
['5']	0
['1']	1
['5']	1
['4']	0
['2']	0
['5']	0
['4', '5', '6']	2
['1', '4', '5']	2
['1', '3', '6']	2
['2', '5', '6']	1
['3', '5', '6']	3
['1', '2', '3']	2
['1', '3', '6']	1
['4', '2', '6']	2
['5', '4', '6']	2
['5', '2', '4']	1
['5', '6', '1']	1
['2', '3', '5']	2
['6', '2', '5']	2
['6', '2', '4']	2
['5', '1', '4']	2
['5', '3', '4']	2

➢ Assessment documents

Before carrying out each individual evaluation, a series of documents were created necessary for them:

- Informed consent: two versions were created, one for the participants themselves, and another just in case there were cases in which the consent had to be signed by a legal representative. Both versions can be consulted in the appendices, sections 9.6 and 9.7.
- Information sheet: an information sheet of the study was written to deliver to the participants who agreed to participate. This sheet is attached to the annexes, section 9.8.
- Datasheet: a table of 4 columns was made (diagnosis, name and surname, code and contact information) where the data of the participants were collected with the corresponding anonymous code. This table is also attached to the annexes, section 9.9.

- Clinical sheet: a clinical sheet was made as summary tables on the cognitive profile of each participant and the results obtained in the tests. This sheet is attached to the annexes, section 9.10.
- Answer sheet: a card was prepared for each test (except for the MMSE, which costs from its own sheet) to collect the answers and calculate the score. This file is attached to the annexes, section 9.11.
- Correction sheet: a sheet was prepared with all the correct answers of each test as a template for subsequent correction. This sheet is attached to the annexes, section 9.12.

➢ Procedure

The participants, both in the Montilivi civic center and in the Onyar day center, were evaluated individually in a well-lit and quiet indoor room, and located 0.5m from the computer screen in the computerized tests.

First of all, they were explained what the study consists of, the informed consents were signed in order to comply with ethical and anonymity guarantees and the information sheets were delivered. However, some initial discarding questions were asked in relation to medical history according to the inclusion and exclusion criteria from the study. Next, the order of the tests followed was: MMSE, BFRT-r, and finally line guidance and number location.

The data obtained from the evaluation have been collected, in the case of the BFRT-r in a computerized way in excel format as explained above, and, in the case of the other tests, by hand: the MMSE in the answer sheet of the notebook itself, and in the case of the other two in the sheets created by me explained in the previous section ("answer sheet"), which can be consulted in the appendices (section 9.11). Once all the data have been obtained, they have been emptied into the data matrix created in the SPSS program (see annexes, section 9.13 figures 1-4) and the relevant analyses have been carried out. The data have been entered in direct scores, since being a small sample -which cannot be considered to follow normal law- and by the type of study, it has been considered relevant.

4.5. ETHICAL GUARANTEES

In order to ensure the confidentiality and anonymity of the participants and ensure the ethical guarantees of the study, the current regulations of the UdG regarding final degree projects have been complied with, the approval of the work tutor has been contemplated in relation to correct professional practice, the corresponding informed consents have been administered

and the responsibilities of the researcher have been fulfilled as well as the administration of the informative document on the study and The correct collection and management of data.

Regarding regulatory and ethical obligations, the study was carried out in accordance with the principles established in the Helsinki Declaration of 1964 and subsequent modifications.

4.6. DATA ANALYSIS

The data analysis of the study was carried out with the *IBM SPSS Statistics* program. A data matrix was created (see annexes, section 9.13) with all the information collected from each participant: diagnosis (or not) of AD, sex, age, grade of GDS, score obtained in the MMSE test, score obtained in the BFRT-r test, score obtained in the VOSP battery number location test, score obtained in the line orientation test (OL) of the RBANS battery, Time of administration of the tests, place of birth, main profession, manual laterality, mother tongue, years they have been living in Catalonia, years of education and place where the tests have been administered (place of recruitment of the sample).

In the data analysis, different tests have been carried out:
First of all, in order to see the relationship between having Alzheimer's and having worse scores in the Benton test, 3 nonparametric tests U of Mann-Whitney have been done (with grouping variable the diagnosis of Alzheimer's in all of them): one between the variable scores in the Benton test with the diagnostic variable (or not) of Alzheimer's, another with the variable of the scores in the RBANS battery line orientation test with the diagnostic variable (or not) of Alzheimer's and finally another with the variable of the scores in the VOSP battery number location test with the diagnostic variable (or not) of Alzheimer's. U of Mann-Whitney’s tests have been performed because the sample is small (n = 10) and quite heterogeneous, and by using a nonparametric test it allows to remove all the singularities that it may have and make a more sensitive analysis.

Secondly, several Spearman correlations (nonparametric test) have been made to see if there is a correlation coefficient on the joint degree of variation of the variables. 3 of these have been done: one with the variable of the scores in the Benton test with the variable of the score in the MMSE test, another with the variable of the scores in the RBANS battery line orientation test with the score in the MMSE test and finally another with the variable of the scores in the VOSP battery number location test with the score in the MMSE test. These correlations have been made with the Spearman coefficient and not Pearson's because the

sample is small (N = 10) and quite heterogeneous, and by using a nonparametric test it allows to remove all the singularities that it may have and make a more sensitive analysis.
The correlations have all been made with the MMSE variable because both variables must necessarily be quantitative, so that with the diagnostic variable (categorical dichotomous) it was not feasible to do them. However, since the participants of this study with a low MMSE this score was due to cognitive impairment due to Alzheimer's, it has been considered a good option to use this measure to perform the 3 correlations.

Thirdly, three simple linear regressions have been performed in order to find a mathematical function that expresses without irregularities the relationship between two variables: first, between the score in the BFRT-r test (VD) and the score in the MMSE test (VI), second between the score in the RBANS battery line orientation test (VD) and the score in the MMSE test (VI), and finally between the score in the VOSP battery number location test (VD) and the score in the MMSE test (VI). By creating these mathematical models, it will allow us to observe whether independent variables significantly explain the variability of the dependent variable (and to what extent).

In penultimate place, in order to confirm and collect the simple regressions carried out previously, a multiple regression has been performed (adding other new variables) for two reasons: on the one hand, to check that there are no other variables not contemplated that predict the scores of the BFRT-r test in the model; On the other hand, with the stepwise method with a backward process through this multiple regression analysis, a final selection of the best predictive model is made in which the variables that are not predictive are eliminated one by one until finally giving the final model good with the only significant predictor variable(s). The multiple regression was performed with the score-dependent variable in the BFRT-r test and with the independent variables of: age, score in the MMSE test, score in the RBANS battery line orientation test and score in the VOSP battery number location test.

Finally, in order to check if the variable of years of education that the participants had also had some influence on the results obtained in the BFRT-r test, due to the fact that it was entered in the matrix as a categorical variable (of more than two modalities), a nonparametric Kruskal-Wallis test has been carried out in order to see the relationship between these two variables.

Table 4 provides a compilation of all the data analyses performed:

Table 4

Collection of data analyses carried out

ANALYSIS	VARIABLES	HYPOTHESES
NPar U of Mann-Whitney	BFRT-r and AD	Ho: the two groups are the same H1: the two groups are different
	OL and AD	Ho: the two groups are the same H1: the two groups are different
	VOSP and AD	Ho: the two groups are the same H1: the two groups are different
NPar Spearman correlation coefficient	BFRT-r and MMSE	Ho: variables are independent H1: The variables are related
	OL and MMSE	Ho: variables are independent H1: The variables are related
	VOSP and MMSE	Ho: variables are independent H1: The variables are related
Simple linear regression	BFRT-r and MMSE	Ho: the variables are independent ($R^2 = 0$) the variability explained by the regression model is null H1: variables are related ($R^2 \neq 0$) The variability explained by the regression model is significantly different from 0
	OL and MMSE	Ho: the variables are independent ($R^2 = 0$) the variability explained by the regression model is null H1: variables are related ($R^2 \neq 0$) The variability explained by the regression model is significantly different from 0

	VOSP and MMSE		Ho: the variables are independent ($R^2 = 0$) the variability explained by the regression model is null H1: variables are related ($R^2 \neq 0$) The variability explained by the regression model is significantly different from 0
Multiple regression	BFRT-r with	Age OL VOSP MMSE	Ho: $R^2 = 0$ (the variability of V.D. explained by the regression model is not significant. The model is not well adjusted / is not a good predictive model) H1: $R^2 \neq 0$ (the variability of V.D. explained by the regression model is significant. The model is well adjusted / is a good predictive model)
NPar Kruskal-Wallis	BFRT-r and Years of Education		Ho: all 6 groups* are the same H1: the 6 groups* are different

Note. BFRT-r: quantitative variable scoring on the BFRT-r test; MA: categorical variable Alzheimer's disease group (patient/control); OL: quantitative score variable in the RBANS battery line orientation test; VOSP: quantitative score variable in the VOSP battery number location test; MMSE: quantitative score variable in the MMSE test; Ho: null hypothesis; H1: alternative hypothesis; V.D.: dependent variable.

*The categorical variable of years of education is divided into 6 numerical values of grouping, forming 6 different groups of participants.

5. RESULTS:

5.1. DESCRIPTIVE RESULTS OF THE SAMPLE

Table 5 shows the descriptive results obtained in relation to the study sample.

Table 5

Demographic information of patients and controls on median scores and interquartile ranges of age data, years of education, administration time and neuropsychological data (GDS, MMSE cognitive impairment score, BFRT-r facial perception score, visuoperception score, VOSP battery number location test, visuoperception score, RBANS battery line orientation test).

	AD			Controls		
Variables	Rank (min / max)	Median	Quartiles (25 / 50 / 75)	Rank (min / max)	Median	Quartiles (25 / 50 / 75)
Age	33 (61 / 94)	82	71 / 82 / 89	10 (74 / 84)	77	75,5 / 77 / 82
Years Ed.	5 (1 / 6)	2	1 / 2 / 4,5	4 (2 / 6)	3	2,5 / 3 / 5,5
GDS	2 (3 / 5)	4	3 / 4 / 5	0 (1 / 1)	1	1 / 1 / 1
MMSE	4 (20 / 24)	21	20,5 / 21 / 23	3 (27 / 30)	29	28 / 29 / 30
BFRT-r	7 (24 / 31)	31	26 / 31 / 31	6 (36 / 42)	37	36,5 / 37 / 41,5
VOSP	7 (3 / 10)	5	3 / 5 / 10	2 (8 / 10)	8	8 / 8 / 10
OL	11 (9 / 20)	17	13 / 17 / 18,5	6 (14 / 20)	17	14,5 / 17 / 20
Time Adm.	6 (30 / 36)	32	30,5 / 32 / 35	2 (30 / 32)	30	30 / 30 / 31,5

5.2. RESULTS OF DATA ANALYSIS TESTS

The results of the data analysis discussed above in Table 4 are presented below:

Table 5 shows the results obtained in the 3 U of Mann-Whitney carried out. Between the dichotomous categorical variable of the diagnosis of AD and the quantitative score of the BFRT-r test, the average ranges of the group with Alzheimer's 3 and on average of control group 8 are observed, a Z value of -2.652 and a p value of 0.008, smaller than 0.05 so that the null hypothesis with alpha risk is rejected: There is a relationship between the group variable

(control/AD) and the variable BFRT-r (scores in the BFRT-r test): there are significant differences between groups, with the control group (AD=2) having higher scores (therefore, recognising more faces) than the group with Alzheimer's (AD=1).

Secondly, between the same group variable and the quantitative variable of the scores obtained in the RBANS battery line orientation test (OL), the average ranges of the Alzheimer's group 5.3 and in the control group 5.7, a Z value of -0.22 and a p value of 0.827, greater than 0.05, are observed so there is no reason to reject the null hypothesis and therefore does not exist relationship between the group variable and the OL variable. There are no significant differences in test scores depending on group.

Thirdly, between the same group variable and the quantitative variable of the scores obtained in the VOSP battery number location test, the average ranges of the Alzheimer's group 4.60 and in the control group 6.40, a Z of -0.986 and a p value of 0.324, greater than 0.05, are observed so there is no reason to reject the null hypothesis. There is no relationship between the group variable and the VOSP variable; There are no significant differences in test scores depending on the group.

Table 5

NPar Tests Mann-Whitney Tests

Variables	Z	p value (Asymp. sig.)	Average ranks	
			AD	C
BFRT-r and AD	-2,652	0,008	3	8
OL and AD	-0,22	0,827	5,3	5,7
VOSP and AD	-0,986	0,324	4,6	6,4

Table 6 presents the results obtained in the 3 Spearman correlations made. Among the variables of scores in the MMSE and BFRT-r tests, a Spearman correlation coefficient of 0.857 (high correlation) and a significance of $p = 0.002 < 0.05$ are observed, so that the null hypothesis with alpha risk is rejected: there is a relationship between both variables. The correlation coefficient is high and positive, so there is a direct relationship between both variables: when one increases, so does the other. Therefore, the higher the score in the MMSE

(person without cognitive impairment, and therefore, in this sample, without Alzheimer's) the higher the score in the BFRT-r test (more faces are recognized).

Secondly, among the variables of scores in the MMSE and OL tests, a Spearman correlation coefficient of 0.288 (very low correlation) and a significance of $p = 0.42 > 0.05$ are observed, so there is no reason to reject the null hypothesis; There is no relationship between the two variables.

Thirdly, and finally, among the variables of scores in the MMSE and VOSP tests, a Spearman correlation coefficient of 0.26 (very low correlation) and a significance of $p = 0.469 > 0.05$ are observed, so there is no reason to reject the null hypothesis; There is no relationship between the two variables.

Table 6

NPar Tests Spearman Correlation Coefficients

Variables	Correlation coefficient	p value (Asymp. sig.)
BFRT-r and MMSE	0,857	0,002
OL and MMSE	0,288	0,420
VOSP and MMSE	0,260	0,469

Table 7 presents the results obtained in the 3 simple linear regressions performed. First of all, between the variable BFRT-r (VD) and MMSE (VI) the following regression line is obtained: $Y = 2.429 + 1.240X$. An $R^2 = 0.786$ is also obtained, so that the model explains 78.6% of the variability of the dependent variable (the score in the BFRT-r test). The p value is $0.001 < 0.005$, so the null hypothesis with alpha risk is rejected and it is confirmed that the variables are related (with a positive, direct relationship) and this is a good predictive model. The independent variable is a good predictor of the scores of the dependent variable: the higher the scores in the MMSE (person without cognitive impairment, and therefore, in this sample, without Alzheimer's) the higher the score in the BFRT-r test (more faces are recognized).

Secondly, between the variable OL (VD) and MMSE (VI) the following regression line is obtained: $Y = 10.412 + 0.245X$. An $R^2 = 0.089$ is also obtained, so that the model explains 8.9% of the variability of the dependent variable (the score in the line orientation test). The p value is $0.403 > 0.05$ so there is no reason to reject the null hypothesis; the independent

variable (MMSE score) does not predict scores on the line orientation test. The two variables are independent.

In third and last place, between the variable VOSP (VD) and MMSE (VI) the following regression line is obtained: Y = -0.401 + 0.312X. An $R^2 = 0.205$ is also obtained, so that the model explains 20.5% of the variability of the dependent variable (the score in the VOSP number localization test). The p value is 0.189 > 0.05 so there is no reason to reject the null hypothesis; the independent variable (score on the MMSE) does not predict scores in the VOSP battery number location test. The two variables are independent.

Table 7

Simple linear regressions

Variables	Coefficient B (Constant / VI)	R^2	p value (Asymp. sig.)
BFRT-r and MMSE	2,429 / 1,240	0,786	0,001
OL and MMSE	10,412 / 0,245	0,089	0,403
VOSP and MMSE	-0,401 / 0,312	0,205	0,189

Table 8 presents the results obtained in the multiple regression between the dependent variable BFRT-r score and the independent variables age, score in OL, score in VOSP and score in MMSE.

In this multiple stepwise regression, it is observed how variables that are not significant and therefore not predictive have been removed from the model. The variables that have been eliminated are age, score in the VOSP test and score in the line orientation test. In this method of the test, the best predictive model has been selected preserving only the MMSE variable (the score in the MMSE test of the participants).

The results of the regression between the dependent variable BFRT-r and the independent variable MMSE are as follows:

Adjustment of the regression line: Y = 2.429 + 1.240 X. $R^2 = 0.786$; the model explains 78.6% of the variability of the dependent variable (the score in the BFRT-r test). A significance of $p = 0.001 < 0.05$ is obtained so that the null hypothesis with alpha risk is rejected, therefore the variables are related and it is a good predictive model: there is a positive (direct) relationship. The independent variable is a good predictor of the scores of the

dependent variable: the higher the scores in the MMSE (person without cognitive impairment, and therefore, in this sample without Alzheimer's) the higher the score in the BFRT-r test (more faces are recognized).

Table 8

Multiple linear regression of the effects of age, of the scores obtained in the Mini-Mental State Exammination test, in the VOSP battery number location test and in the RBANS line orientation test on the scores obtained in the Benton facial recognition test (BFRT-r).

Dependent variable	Predictor variables	Coefficient B (Constant / VI)	R^2	p value (Asymp. sig.)
BFRT-r	MMSE	2,429 / 1,240	0,786	0,001

	Excluded variables	β	Partial correlation	p value (Asymp. sig.)
	Age	-0,101	-0,207	0,593
	VOSP	-0,318	0,612	0,08
	OL	-0,119	-0,245	0,524

Finally, table 9 presents the results obtained in the nonparametric Kruskal-Wallis test between the quantitative variable BFRT-r and the categorical multiple of the years of education. You get a Kruskal Wallis H of 3.159 and a p value of 0.532 > 0.05 so there is no reason to reject the null hypothesis; The variables are not related.

Table 9

Kruskal-Wallis NPar Test

Variables	H	p value (Asymp. sig.)	Average ranks				
			NS	LA	PS	MS	HE
BFRT-r and Years of education	3,159	0,532	4	4,25	5,67	10	5,75

Note. NS: Not schooled; LA: Literacy acquisition; PS: Primary studies; MS: Middle studies; HE: Higher education.

6. DISCUSSION:

The main objective of this study was to analyse and detect whether there is a specific deficit in facial visual perception in patients with Alzheimer's disease. In order to test whether this deficit existed, two specific objectives were established (ignoring the first from the *PsychoPy* program): to analyze and compare facial visual perception capacities (with BFRT-r) in a control group and a group with Alzheimer's; and, in contrast to this, analyze and compare generic visual perception capabilities (with the RBANS line orientation test and the VOSP number localization test) in a control group and one with Alzheimer's.

Thus, in relation to these two specific objectives mentioned, the results show that there is a relationship between the scores obtained in the facial perception test (BFRT-r) and the group (AD or controls), while there is no relationship between the scores obtained in the generic visuoperceptive tests (OL and VOSP) and the group (AD or controls). In the U of Mann-Whitney tests, it is shown that there is a relationship between the group variable and the results of BFRT-r (group with Alzheimer's, worst scores), while in the case of the tests for the OL and the VOSP, it is shown that there is no relationship between the variables and the fact of belonging to one group or the other, so that at the moment this indicates that there is a specific deficit in facial visual perception in the group with Alzheimer's disease.

However, in order to corroborate this and be able to confirm the general objective, it is necessary to take into account the rest of the analyses carried out. Thus, with the Spearman correlation coefficients obtained, it is also observed in the same line that between the scores in the MMSE test and the BFRT-r there is a significant positive (direct) high correlation; in this way, it means that the higher a participant has a participant in the MMSE (therefore, who

will not have cognitive impairment, or in case of having one would be on a scale of mild to more severe degrees of Alzheimer's) the more faces will be recognized in the BFRT-r. In other words, people with fewer points on MMSE (cognitive impairment exposed by the test, but that is given by Alzheimer's due to the corroborated diagnosis of the participants) will have more difficulties in the task of facial perception. In contrast, in these same Spearman correlation coefficients in the case of facts with OL and VOSP in relation also to BFRT-r, the results show that there is no relationship between the variables. Thus, this deficit of the AD group at the moment points to be specific in a facial type, not in a generic visuoperceptive deficit.

On the other hand, with simple linear regressions it has also been shown how there is indeed a relationship with having Alzheimer's and the BFRT-r score: the model built with the MMSE variable (VI) predicts significantly ($p = 0.001$) 78.6% the variability of the dependent variable (BFRT-r) and also in a positive way (direct), so that the higher the score in the MMSE (non-deterioration, control group; or if you do have impairment, milder phase of Alzheimer's), higher BFRT-r score (more faces are recognized). In contrast, in simple linear regressions made with OL and VOSP, these are not significant and it is shown how both variables are independent with the score obtained in the MMSE.

Apart from simple linear regressions, a multiple regression has been made to include other possible predictor variables in the variability of the BFRT-r model, and also by re-including these three previous ones of the simple regressions to corroborate this. In this case, it is evident that neither age, nor the score in the OL nor in the VOSP are predictive variables, so that in the stepwise method they have been eliminated, and a good and predictive final model of the dependent variable BFRT-r has been obtained where only the variable of the score in the MMSE with the positive (direct) relationship appears.

Finally, also in order to see if there was a relationship and influence of the variable of years of education, since it was categorized as qualitative of multiple modality, in the Kruskal-Wallis test it is evident that there is no relationship between it and the score obtained in the BFRT-r. Therefore, years of education do not influence the evaluated ability to perceive faces.

Thus, with all these analyses, the specific objectives are specifically met, and finally also the general objective of the study, where all the results seem to indicate that there is this specific deficit in facial visual perception in people with AD.

Regarding the initial hypotheses in relation to the objectives, both have also been met, obtaining results in facial perception tests significantly lower in the AD group than in the control group, and results in generic visual perception tests that do not differ significantly between groups.

These results are consistent with those obtained by Lavellée et al. (2016) when studying this same perceptual ability between a sample with AD and controls, in which AD patients performed lower. In the same line, these results also agree with those obtained in the study by Becker et al. (1995), finding significant differences between the results of the facial discrimination test between both groups, although in this study the clinical group had a diagnosis of probable AD.

The other studies carried out in the field of the gnosic line of this deficit, as discussed in section 2.5.4 are electrophysiological and/or imaging studies; In this sense, it cannot be corroborated that the results obtained in this study are comparable to the rest because no activations of brain areas or evoked potentials have been observed, but the results found with this study reinforce the findings of all these regarding the specific areas of the brain (and / or networks, processes) related to facial perception, especially those that have been carried out studying specific pathologies (such as AD): for example, the study by Hung et al. (2020) or that of Mazzi et al. (2020).

6.1. THEORETICAL-PRACTICAL AND CLINICAL IMPLICATIONS OF THE RESULTS

At a theoretical level, the results obtained contribute to increasing the thickness of the literature in order to shed light on how facial perception mechanisms work (both in the non-clinical population and in the clinical population), and on the gnosic (understudied) and mnesic lines that have studied these deficits in AD to date. Although it is true that completely firm conclusions cannot be drawn solely from this study because a multidisciplinary approach is needed to attribute causality to a specific deficit, providing literature does help to incorporate new explanatory perspectives on these mechanisms and how exactly the clinical and neuroscience of AD works. In the same way, it helps to provide information on the functioning of the relationship between cognitive and perceptual processes by showing how these areas can interact and influence each other, in a non-clinical or neurodegeneration context.

Regarding the practical implications, these results can help illustrate interventions aimed at improving the quality of life of these people and their follow-up, better understanding which mechanisms are responsible for the clinic of the disease. These interventions may also

include tasks aimed at improving these skills with exercises of visual or cognitive therapies for these skills, or the elaboration of visual materials that help patients recognize the faces of their relatives / caregivers / social circle. So much so, that it is also worth highlighting the involvement in the screening and diagnosis part of the disease; facial skills are rarely evaluated in clinical practice, although these skills are critical in the lives of people with AD (Lavallée, et al., 2016), so the development of new clinical tools to evaluate these aspects may be especially relevant and useful for practitioners (Lavallée, et al., 2016).

Finally, in the clinical field, the fact of knowing better the clinic of the disease and in a more specific and specific way, will improve the differential diagnosis with other neurodegenerative disorders with similar clinics, while adapting its treatment and intervention.

6.2. LIMITATIONS

This study has been limited by several aspects. At a methodological level, the **sample** is very small (N = 10); it was not possible to access a larger sample -and, therefore, more representative- due to the limitation of **time** and the inclusion criteria so specific to this study; the patients of the AD group needed to have this diagnosis, without being in early stages or in a staging too advanced (because they would not understand the tasks) and could not have biological deficits Visual. In this sense, a large % of the sample to which participation in the study was considered could not be accepted; in the civic center 10 people were rejected because of this deficit and in the day center 8). In addition to this, no hospital in Girona or Barcelona agreed to collaborate. In the case of all nursing homes in Girona, except for one, either.

At the instrument level, it is considered a limitation not to have more **time** for the study in order to have been able to administer a **more exhaustive neuropsychological evaluation** in order to have a more accurate cognitive profile of the participants. The fact of administering only the MMSE as a cognitive screening test in order to have a vision of the profile of each participant is considered limiting.

On the other hand, not having been able to administer the tests in a laboratory context is also considered a limitation.

It is also worth mentioning that the fact of not having combined these tasks with neuroimaging and/or electrophysiological tests (for example, as in the studies of Huang et al., 2020, or Rossion et al., 2012, or Bentin et al., 1996) is considered a limitation in order to address it in a more complete way and to be able to draw conclusions from an integrated

perspective. They have not been carried out due to limitations of time and access to the instruments.

On the other hand, another limitation to consider is the fact of not having been able to carry out the study with a longitudinal perspective: it would have been interesting and more solid to carry out this same study with repeated measurements of the participants over the years, before having AD (in the case of the clinical group) and afterwards, similar to what Saavedra et al. (2015) did. Despite the uncertainty of which participants will develop AD, being the most frequent type of dementia worldwide and advances in medicine in terms of detection of risk factors and precursor genes of AD, this line could be considered.

7. CONCLUSIONS:

This research, to the best of our knowledge, was the third study in the literature to specifically assess and study facial visuoperceptive deficit in the clinical population of AD (at least without studying it through imaging tests and evoked potentials); these skills (with the BFRT-r test) have been evaluated in relation to generic visuoperceptive skills (with the RBANS battery line orientation tests and VOSP number localization tests) in two groups, one with an AD diagnosis and a control, in order to shed light on whether the deficit in facial recognition of patients is due only to a mnesic problem or can also be attributed to gnosic deficits.

The main findings of this study found significant differences between the two groups in the test assessing specific deficits in facial perception, whereas in the other two tests not. This study, due to the limitations of the sample, time, and the non-possibility of multidisciplinary evaluation, is not sufficient to attribute a (single) causality, but it does provide evidence of significant different results in these capacities between both groups. Thus, although the mnesic reason why patients have problems recognizing familiar faces, this study provides significant literature that reinforces the gnosic line on the addition of possible facial perceptual deficit.

7.1. FUTURE LINES

For future research, as mentioned in the limitations, it would be interesting to repeat the study with a larger, more homogeneous sample, and be able to do it longitudinally in order to avoid individual biases.

It would also be interesting to combine this paradigm with other types of assessments simultaneously such as neuroimaging and/or electrophysiological tests.

On the other hand, study modalities can also be proposed aimed at finding out more specifically, in this gnostic line, at what specific point or process the deficit is, or from what stage / staging, or if it is not only specific to AD but to other dementias or cognitive impairments in general. Mohr et al. (2018), for example, in relation to holistic facial processing performed on face recognition, found evidence through the N250 component on recognition based on facial features. Thus, taking into account studies and approaches in this regard, the present study can be replicated under other conditions in order to find where the specific deficits lie. Eye-tracker studies can also be carried out to expand the information and see which characteristics are the most used by facial discrimination processes, and study the most common errors in the selected faces in paradigm tasks.

Also on the other hand, one could try to replicate the study by focusing on changing aspects of the paradigm; the face stimuli that were made were all Caucasian males (Murray et al., 2021) so that the results found with these same stimuli could be studied by applying the test to populations of other ethnicities (e.g., Asian), or vice versa: apply the study here as it has been done but using stimuli from people of another ethnicity; in this way, another informative way of how the human facial perceptual system works could also be explored.

Finally, the most important thing to consider, is that this gnosic line is very little studied; there are numerous studies of facial perception, but not in AD. Thus, the essential thing is nothing more than to provide more studies in the field and help to better characterize and identify these deficits.

8. REFERENCES:

Abellón, S. P. (2018). *ALZHEIMER, MEMORIA Y LENGUAJE DISEÑO DE UN PROGRAMA DE INTERVENCIÓN: ESTUDIO DE CASO*. Universidad de Valladolid.

Alfaro, E., Vázquez, N., Pérez, I. & Real, S. (2016). PERCEPTION AND FACIAL RECOGNITION: THEORETICAL BASES OF LINEUPS. *Gaceta Internacional de Ciencias Forenses* (18) 5-11.

American Psychological Association [APA]. (2013). *Diagnostic and Statistical Manual of mental disorders* (5th ed.). https://doi.org/10.1176/appi.books.9780890425596

Ayuso, T., Ederra M., Manubens J., Nuin M., Villar, D. & Zubioca, J. (2007). *Abordaje de la demencia. Guía de actuación en la coordinación Atención Primaria - Neurología*. Servicio Navarro de Salud-Osasunbidea.

Becker, J. T., Lopez, O. L., & Boller, F. (1995). Understanding Impaired Analysis of Faces by Patients with Probable Alzheimer's Disease. *Cortex*, *31*(1), 129–137. https://doi.org/10.1016/S0010-9452(13)80111-6

Becker, J. T., & Overman, A. A. (2000). El déficit de la memoria semántica en la enfermedad de Alzheimer. *REV NEUROL*.

Bentin, S., Allison, T., Puce, A., Perez, E., & McCarthy, G. (1996). Electrophysiological Studies of Face Perception in Humans. *Journal of Cognitive Neuroscience*, *8*(6), 551–565. https://doi.org/10.1162/jocn.1996.8.6.551

Bentin, S., & Deouell, L. Y. (2000). STRUCTURAL ENCODING AND IDENTIFICATION IN FACE PROCESSING: ERP EVIDENCE FOR SEPARATE MECHANISMS. *Cognitive Neuropsychology*, *17*(1–3), 35–55. https://doi.org/10.1080/026432900380472

Benton, A. L., Sivan, A. B., Hamsher, K. deS, Varney, N. R., & Spreen, O. (1983). Facial recognition : stimulus and multiple choice pictures : contributions to neuropsychological assessment. Oxford University Press, Inc.

Blackburn, H. L., & Benton, A. L. (1957). Revised administration and scoring of the Digit Span Test. *Journal of Consulting Psychology*, *21*(2), 139–143. https://doi.org/10.1037/h0047235

Calvo, L., Casals-Coll, M., Sánchez-Benavides, G., Quintana, M., Manero, R. M., Rognoni, T., Palomo, R., Aranciva, F., Tamayo, F., & Peña-Casanova, J. (2013). Estudios normativos españoles en población adulta joven (proyecto NEURONORMA jóvenes): Normas para las pruebas Visual Object and Space

Perception Battery y Judgment of Line Orientation. *Neurología*, *28*(3), 153–159. https://doi.org/10.1016/j.nrl.2012.03.007

Calvo, M. G., Avero, P., Fernández-Martín, A., & Recio, G. (2016). Recognition thresholds for static and dynamic emotional faces. *Emotion*, *16*(8), 1186–1200. https://doi.org/10.1037/emo0000192

Caño, A. (2020). *NEUROCIENCIA COGNITIVA: LÒBUL OCCIPITAL I COGNICIÓ*.

Carlesimo, G., Caltagirone, C., Gainotti, G. (1995). The Mental Deterioration Battery: Normative Data, Diagnostic Reliability and Qualitative Analyses of Cognitive Impairment. *European Neurology,* (36), 378-384.

Crawford, J. R., & Howell, D. C. (1998). Comparing an Individual's Test Score Against Norms Derived from Small Samples. *The Clinical Neuropsychologist*, *12*(4), 482–486. https://doi.org/10.1076/clin.12.4.482.7241

Cuetos, F., Rodríguez-Ferreiro, J., & Martínez, C. (n.d.). *Alteraciones de memoria en los inicios de la enfermedad de Alzheimer*.

Cuevas, L. (2021). RECONOCIMIENTO FACIAL HUMANO Y RETRATO HABLADO 4.0. *Revista Mexicana de Ciencias Penales, 13* (92-118).

Daini, R., Comparetti, C. M., & Ricciardelli, P. (2014). Behavioral dissociation between emotional and non-emotional facial expressions in congenital prosopagnosia. *Frontiers in Human Neuroscience*, *8*. https://doi.org/10.3389/fnhum.2014.00974

Dering, B., Martin, C. D., & Thierry, G. (2009). Is the N170 peak of visual event-related brain potentials car-selective? *NeuroReport*, *20*(10), 902–906. https://doi.org/10.1097/WNR.0b013e328327201d

Donix, M., Jurjanz, L., Meyer, S., Amanatidis, E. C., Baeumler, D., Huebner, T., Poettrich, K., Smolka, M. N., & Holthoff, V. A. (2013). Functional Imaging During

Recognition of Personally Familiar Faces and Places in Alzheimer's Disease. *Archives of Clinical Neuropsychology*, *28*(1), 72–80. https://doi.org/10.1093/arclin/acs093

Dubois, B., Feldman, H. H., Jacova, C., Cummings, J. L., DeKosky, S. T., Barberger-Gateau, P., Delacourte, A., Frisoni, G., Fox, N. C., Galasko, D., Gauthier, S., Hampel, H., Jicha, G. A., Meguro, K., O'Brien, J., Pasquier, F., Robert, P., Rossor, M., Salloway, S., … Scheltens, P. (2010). Revising the definition of Alzheimer's disease: A new lexicon. *The Lancet Neurology*, *9*(11), 1118–1127. https://doi.org/10.1016/S1474-4422(10)70223-4

Duyckaerts, C., Delatour, B., & Potier, M.-C. (2009). Classification and basic pathology of Alzheimer disease. *Acta Neuropathologica*, *118*(1), 5–36. https://doi.org/10.1007/s00401-009-0532-1

Gich, J. (n.d.). *Introducció al funcionament normal del SNC*.

Gich, J. (n.d.). *TRASTORNS DEL RECONEIXEMENT: LES AGNÒSIES*.

Gich, J. (n.d.). *TRASTORNS DE LA MEMÒRIA: LES AMNÈSIES*.

Göbel, N., Möller, J. C., Hollenstein, N., Binder, A., Oechsner, M., Ide, J., Urwyler, P., Cazzoli, D., & Müri, R. M. (2021). Face Perception and Pareidolia Production in Patients With Parkinson's Disease. *Frontiers in Neurology*, *12*, 669691. https://doi.org/10.3389/fneur.2021.669691

Gollan, T. H., Weissberger, G. H., Runnqvist, E., Montoya, R. I., & Cera, C. M. (2012). Self-ratings of spoken language dominance: A Multilingual Naming Test (MINT) and preliminary norms for young and aging Spanish–English bilinguals. *Bilingualism: Language and Cognition*, *15*(3), 594–615. https://doi.org/10.1017/S1366728911000332

Gregory, E. (2018). Processing faces in Alzheimer's disease patients: How a familiar face becomes unfamiliar. Canadian Journal of Undergraduate Research.

Guía oficial de práctica clínica en demencia. (2018). Luzán 5 Health Consulting S.A. ; Ediciones SEN.

Huang, J., Beach, P., Bozoki, A., & Zhu, D. C. (2020). Alzheimer's Disease Progressively Alters the Face-Evoked Visual-Processing Network. *Journal of Alzheimer's Disease*, *77*(3), 1025–1042. https://doi.org/10.3233/JAD-200173

Imirizaldu, J. J. Z. (n.d.). *167 Anamnesis y exploración del paciente con sintomatología del sistema nervioso*.

Itier, R. J. (2004). N170 or N1? Spatiotemporal Differences between Object and Face Processing Using ERPs. *Cerebral Cortex*, *14*(2), 132–142. https://doi.org/10.1093/cercor/bhg111

Ivanova, I., Salmon, D. P., & Gollan, T. H. (2013). The Multilingual Naming Test in Alzheimer's Disease: Clues to the Origin of Naming Impairments. *Journal of the International Neuropsychological Society*, *19*(3), 272–283. https://doi.org/10.1017/S1355617712001282

Jack, C. R., Bennett, D. A., Blennow, K., Carrillo, M. C., Dunn, B., Haeberlein, S. B., Holtzman, D. M., Jagust, W., Jessen, F., Karlawish, J., Liu, E., Molinuevo, J. L., Montine, T., Phelps, C., Rankin, K. P., Rowe, C. C., Scheltens, P., Siemers, E., Snyder, H. M., . . . Silverberg, N. (2018). NIA‐AA Research Framework: Toward a biological definition of Alzheimer's disease. *Alzheimer's & Dementia*, *14*(4), 535-562. https://doi.org/10.1016/j.jalz.2018.02.018

Jemel, B., Pisani, M., Rousselle, L., Crommelinck, M., & Bruyer, R. (2005). Exploring the functional architecture of person recognition system with event-related

potentials in a within- and cross-domain self-priming of faces. *Neuropsychologia*, *43*(14), 2024–2040. https://doi.org/10.1016/j.neuropsychologia.2005.03.016

Kanwisher, N. (2009). Face Perception. Inside G. Bernstson & J. Cacioppo (ed.), *Handbook of Neuroscience for the Behavioural Science* (841-858). John Wiley &Sons, Inc.

Komolovaitė, D. (n.d.). *EEG Signal Analysis of the Visual Cortex in Individuals with Alzheimer's Disease*.

Kurth, S., Moyse, E., Bahri, M. A., Salmon, E., & Bastin, C. (2015). Recognition of personally familiar faces and functional connectivity in Alzheimer's disease. *Cortex*, *67*, 59–73. https://doi.org/10.1016/j.cortex.2015.03.013

Lavallée, M. M., Gandini, D., Rouleau, I., Vallet, G. T., Joannette, M., Kergoat, M.-J., Busigny, T., Rossion, B., & Joubert, S. (2016). A Qualitative Impairment in Face Perception in Alzheimer's Disease: Evidence from a Reduced Face Inversion Effect. *Journal of Alzheimer's Disease*, *51*(4), 1225–1236. https://doi.org/10.3233/JAD-151027

Llibre-Rodríguez, J. de J., Herrera, R. G., & Hernández, M. A. G. (n.d.). Enfermedad de Alzheimer: Actualización en su prevención, diagnóstico y tratamiento. *2022*, *21*(3), 7.

López-Pousa, S., Garre-Olmo, J., Turon-Estrada, A., Hernández, F., Expósito, I., Lozano-Gallego, M., Hernández-Ferrándiz, M., Gelada-Batlle, E., Pericot-Nierga, I., & Vilalta-Franch, J. (2004). Análisis de los costes de la enfermedad de Alzheimer en función del deterioro cognitivo y funcional. *Medicina Clínica*, *122*(20), 767–772. https://doi.org/10.1016/S0025-7753(04)74381-X

Manzanero, A. L. (2023, desember 25th). Percepción Facial. Psicología de la Percepción - UCM: páginas sobre la psicología de la percepción: teorías sobre la percepción visual y auditiva, ilusiones perceptivas y otros fenómenos perceptivos. https://psicologiapercepcion.blogspot.com/p/percepcion-facial.html

Mazzi, C., Massironi, G., Sanchez-Lopez, J., De Togni, L., & Savazzi, S. (2020). Face Recognition Deficits in a Patient With Alzheimer's Disease: Amnesia or Agnosia? The Importance of Electrophysiological Markers for Differential Diagnosis. *Frontiers in Aging Neuroscience*, *12*, 580609. https://doi.org/10.3389/fnagi.2020.580609

McKhann G et al. Clinical diagnosis of Alzheimer's disease: Report of the NINCDS-ADRDA Work Group under the auspices of Department of Health and Human Services Task Force on Alzheimer's Disease. Neurology 1984; 34: 939-944.

Mishra, M. V., Fry, R. M., Saad, E., Arizpe, J. M., Ohashi, Y.-G. B., & DeGutis, J. M. (2021). Comparing the sensitivity of face matching assessments to detect face perception impairments. *Neuropsychologia*, *163*, 108067. https://doi.org/10.1016/j.neuropsychologia.2021.108067

Mohr, S., Wang, A., & Engell, A. D. (2018). Early identity recognition of familiar faces is not dependent on holistic processing. *Social Cognitive and Affective Neuroscience*, *13*(10), 1019–1027. https://doi.org/10.1093/scan/nsy079

Muntal, S., Doval, E., Badenes, D., Casas-Hernanz, L., Cerulla, N., Calzado, N., & Aguilar, M. (2020). Nuevos datos normativos de la versión española de la Repeatable Battery for the Assessment of Neuropsychological Status (RBANS) forma A. *Neurología*, *35*(5), 303–310. https://doi.org/10.1016/j.nrl.2017.09.001

Murray, E., Bennetts, R., Tree, J., & Bate, S. (2021). An update of the Benton Facial Recognition Test. *Behavior Research Methods*, *54*(5), 2318–2333. https://doi.org/10.3758/s13428-021-01727-x

National Alzheimer's Coordinating Center. (2024, january 5th). Forms and documentation Uniform Data Set (UDS). https://naccdata.org/data-collection/forms-documentation/uds-3

Neurowikia. (2024, may 23rd). Bases neurobiológicas de los síndromes de negligencia espacial. NeuroWikia: El portal de contenidos en neurologia. http://www.neurowikia.es/content/bases-neurobiol%C3%B3gicas-de-los-s%C3%ADndromes-de-negligencia-espacial

OR, Otorrinolaringología (6ª ed). (2013). Academia de Estudios MIR.

Palau, F. G., Berrios, W., Basalo, M. M. G., Quintana, M. O., Fernández, M., Basalo, M. J. G., Fernández, M. C., Cámpora, N., Matusevich, L. D., & Golimstok, Á. (2018). *Validez de la prueba de Evaluación Cognitiva de Montreal (MoCA) como una herramienta de screening para detectar Deterioro Cognitivo Leve en población de Buenos Aires, Argentina*.

Portellano, J. A. (2011). *Introducción a la neuropsicología*. McGraw-Hill España.

Porto, M. F., Benitez-Agudelo, J. C., Aguirre-Acevedo, D. C., Barceló-Martinez, E., & Allegri, R. F. (2022). Diagnostic accuracy of the UDS 3.0 neuropsychological battery in a cohort with Alzheimer's disease in Colombia. *Applied Neuropsychology: Adult*, *29*(6), 1543–1551. https://doi.org/10.1080/23279095.2021.1897007

Prieto, E. (2011). Early (N170/M170) face-sensitivity despite right lateral occipital brain damage in acquired prosopagnosia. *Frontiers in Human Neuroscience*, *5*. https://doi.org/10.3389/fnhum.2011.00138

Quintana, M. (2009). Test Barcelona Abreviado: Datos normativos. Aproximación desde la Teoría de Respuesta a los Ítems y redes neuronales artificiales en el deterioro cognitivo leve y enfermedad de Alzheimer.

R.I. (15th of desember 2022). La causa por la que las mujeres tienen más alzhéimer que los hombres. ABC: Canal salud. https://www.abc.es/salud/enfermedades/causa-mujeres-alzheimer-hombres-20221214133907-nt.html?ref=https://www.abc.es/salud/enfermedades/causa-mujeres-alzheimer-hombres-20221214133907-nt.html

Reina, M. R.-A. (n.d.). EL PAPEL DEL PSICÓLOGO EN LA ENFERMEDAD DE ALZHEIMER. *2000*.

Reitan, R. M. (1971). Trail Making Test Results for Normal and Brain-Damaged Children. *Perceptual and Motor Skills*, *33*(2), 575–581. https://doi.org/10.2466/pms.1971.33.2.575

Rossion, B., & Caharel, S. (2011). ERP evidence for the speed of face categorization in the human brain: Disentangling the contribution of low-level visual cues from face perception. *Vision Research*, *51*(12), 1297–1311. https://doi.org/10.1016/j.visres.2011.04.003

Rossion, B., Gauthier, I., Tarr, M. J., Despland, P., Bruyer, R., Linotte, S., & Crommelinck, M. (n.d.). *The N170 occipito-temporal component is delayed and enhanced to inverted faces but not to inverted objects: An electrophysiological account of face-speci®c processes in the human brain*. *3*.

Rossion, B., Hanseeuw, B., & Dricot, L. (2012). Defining face perception areas in the human brain: A large-scale factorial fMRI face localizer analysis. *Brain and Cognition*, *79*(2), 138–157. https://doi.org/10.1016/j.bandc.2012.01.001

Rossion, B., & Jacques, C. (2011a). *The N170: Understanding the Time Course of Face Perception in the Human Brain*. Oxford University Press. https://doi.org/10.1093/oxfordhb/9780195374148.013.0064

Rossion, B., & Jacques, C. (2011b). *The N170: Understanding the Time Course of Face Perception in the Human Brain*. Oxford University Press. https://doi.org/10.1093/oxfordhb/9780195374148.013.0064

Rossion, B., & Michel, C. (2017). *Normative data for accuracy and response times at the computerized Benton Facial Recognition Test (BFRT-c)*. https://doi.org/10.31234/osf.io/twmz2

Saavedra, C., Olivares, E. I., & Iglesias, J. (n.d.). *Estudio transversal y longitudinal sobre el envejecimiento y la enfermedad de Alzheimer en estadio leve mediante los potenciales cerebrales N170-VPP durante el procesamiento de caras conocidas y desconocidas*.

Sadr, J., & Sinha, P. (2004). Object recognition and Random Image Structure Evolution. *Cognitive Science*, *28*(2), 259–287. https://doi.org/10.1207/s15516709cog2802_7

Sagiv, N., & Bentin, S. (2001). Structural Encoding of Human and Schematic Faces: Holistic and Part-Based Processes. *Journal of Cognitive Neuroscience*, *13*(7), 937–951. https://doi.org/10.1162/089892901753165854

Sarmiento, L. A. S. (2023). *Enfermedad de Alzheimer: Etiología y principales factores de riesgo.*

Saykin, A. J., Flashman, L. A., Frutiger, S. A., Johnson, S. C., Mamourian, A. C., Moritz, C. H., O'Jile, J. R., Riordan, H. J., Santulli, R. B., Smith, C. A., & Weaver, J. B. (1999). Neuroanatomic substrates of semantic memory impairment in Alzheimer's disease: Patterns of functional MRI activation. *Journal of the*

International Neuropsychological Society, *5*(5), 377–392. https://doi.org/10.1017/S135561779955501X

Scheltens, P., Blennow, K., Breteler, M. M. B., De Strooper, B., Frisoni, G. B., Salloway, S., & Van Der Flier, W. M. (2016). Alzheimer's disease. *The Lancet*, *388*(10043), 505–517. https://doi.org/10.1016/S0140-6736(15)01124-1

Scheltens, P., De Strooper, B., Kivipelto, M., Holstege, H., Chételat, G., Teunissen, C. E., Cummings, J., & Van Der Flier, W. M. (2021). Alzheimer's disease. *The Lancet*, *397*(10284), 1577–1590. https://doi.org/10.1016/S0140-6736(20)32205-4

Sisakhti, M., Hosseini, H., Batouli, S. A. H., & Farrahi, H. (2024). The Benson Complex Figure Test: Normative Data for the Healthy Iranian Population. *Frontiers in Biomedical Technologies*. https://doi.org/10.18502/fbt.v11i2.15339

Sociedad Española de Neurología. (2024, january 5th). Criterios NINCDS-ADRDA para el diagnóstico de la enfermedad de Alzheimer. Sociedad Española de Neurologia: Grupo de estudio de conducta y demencias: notícias destacadas. https://demencias.sen.es/index.php/noticias-destacadas/125-documentos-de-interes/criterios-diagnosticos/215-criterios-nincds-adrda-para-el-diagnostico-de-la-enfermedad-de-alzheimer

Sommer, W., Stapor, K., Kończak, G., Kotowski, K., Fabian, P., Ochab, J., Bereś, A., & Ślusarczyk, G. (2021). The N250 event-related potential as an index of face familiarity: A replication study. *Royal Society Open Science*, *8*(6), 202356. https://doi.org/10.1098/rsos.202356

Tak, S. H., & Hong, S. H. (2014). Face-name memory in Alzheimer's disease. *Geriatric Nursing*, *35*(4), 290–294. https://doi.org/10.1016/j.gerinurse.2014.03.004

Takizawa, C., Thompson, P. L., Van Walsem, A., Faure, C., & Maier, W. C. (2014). Epidemiological and Economic Burden of Alzheimer's Disease: A Systematic Literature Review of Data across Europe and the United States of America. *Journal of Alzheimer's Disease*, *43*(4), 1271–1284. https://doi.org/10.3233/JAD-141134

Tanaka, H. (2018). Face-sensitive P1 and N170 components are related to the perception of two-dimensional and three-dimensional objects. *NeuroReport*, *29*(7), 583–587. https://doi.org/10.1097/WNR.0000000000001003

Tanaka, H. (2020). Mental rotation of alphabet characters affects the face-sensitive N170 component. *NeuroReport*, *31*(12), 897–901. https://doi.org/10.1097/WNR.0000000000001476

Taubert, J., Aagten-Murphy, D., & Parr, L. A. (2012). A Comparative Study of Face Processing Using Scrambled Faces. *Perception*, *41*(4), 460–473. https://doi.org/10.1068/p7151

Toloza-Ramírez, D., Méndez-Orellana, C., & Martella, D. (2021). Diagnóstico neuropsicológico diferencial en enfermedad de Alzheimer y demencia frontotemporal: Una revisión sistemática cualitativa. *Neurology Perspectives*, *1*(1), 82–97. https://doi.org/10.1016/j.neurop.2021.03.004

Torres Mendonça De Melo Fádel, B., Santos De Carvalho, R. L., Belfort Almeida Dos Santos, T. T., & Dourado, M. C. N. (2019). Facial expression recognition in Alzheimer's disease: A systematic review. *Journal of Clinical and Experimental Neuropsychology*, *41*(2), 192–203. https://doi.org/10.1080/13803395.2018.1501001

Tsao, D. Y., & Livingstone, M. S. (2008). Mechanisms of Face Perception. *Annual Review of Neuroscience*, *31*(1), 411–437. https://doi.org/10.1146/annurev.neuro.30.051606.094238

Werheid, K., & Clare, L. (2007). Are Faces Special in Alzheimer's Disease? Cognitive Conceptualisation, Neural Correlates, and Diagnostic Relevance of Impaired Memory for Faces and Names. *Cortex*, *43*(7), 898–906. https://doi.org/10.1016/S0010-9452(08)70689-0

Wimo, A., Reed, C. C., Dodel, R., Belger, M., Jones, R. W., Happich, M., Argimon, J. M., Bruno, G., Novick, D., Vellas, B., & Haro, J. M. (2013). The GERAS Study: A Prospective Observational Study of Costs and Resource Use in Community Dwellers with Alzheimer's Disease in Three European Countries – Study Design and Baseline Findings. *Journal of Alzheimer's Disease*, *36*(2), 385–399. https://doi.org/10.3233/JAD-122392

Yin, R. K. (n.d.). *LOOKING AT UPSIDE-DOWN FACES*.

Zarranz, J. J. (Ed.). (2018). *Neurología* ([Sixth edition]). Elsevier.

9. ANNEXES

9.1. TABLE 1

Table 1

Diagnostic criteria for mild cognitive impairment (MCI)

May Clinic criteria for amnesic MCI (Petersen et al., 1999)
1. Subjective memory disorder, preferably corroborated by an informant 2. Essentially normal global cognition 3. Activities of practically normal daily living 4. Impaired target memory for age and educational level 5. Absence of dementia
Diagnostic criteria according to IPA-WHO criteria (Levy, 1994)
-No age restriction -Decrease in cognitive ability affirmed by the patient and/or informant -Gradual decrease and minimum duration of 6 months -Any of the following areas may be affected: a) Memory and learning b) Attention and concentration c) Thought d) Language e) Visuospatial function -Decreased mental state assessment scores or neuropsychological tests, a standard deviation below the control group value -There are no brain, systemic or psychiatric processes that can explain the picture
Criteria of the Spanish Society of Neurology for the diagnosis of mild cognitive impairment (Robles et al., 2002)
1. Alteration of one or more of the following cognitive areas: a. Attention/concentration b. Language c. Gnosias

d. Memory
e. Praxias
f. Visuospatial functions
g. Executive functions

2. This alteration must be:
   a. Acquired: indicates a deterioration with respect to the individual's previous abilities
   b. Referred by the patient or by a reliable informant
   c. Objectified in neuropsychological exploration
   d. Lasting months and confirmed in the patient with a normal level of consciousness
3. Cognitive impairment only minimally interferes with instrumental or advanced daily activities
4. Cognitive alteration is not associated with disorders of the level of consciousness

Note. Adapted from *Mild cognitive impairment*, by Quintana (2009).

9.2. TABLE 2

Table 2

2011 ISA-AA diagnostic criteria

NIA-AA CRITERIA (2011)	
PROBABLE ALZHEIMER'S DEMENTIA	MILD COGNITIVE IMPAIRMENT DUE TO ALZHEIMER'S TYPE DEMENTIA
1. WITH CLINICAL CRITERIA 1.1. Only clinical criteria: dementia criteria, to which the following are added: - Insidious start in months or years. - Cognitive worsening in terms of observation. - The most relevant initial cognitive deficits in history and on the exam affect one of the following	**1. CLINICAL CRITERIA FOR MCL** 1.1. Concern about the presence of a cognitive change with respect to the previous level, obtained from the patient, an informant or a clinician who knows the patient. 1.2. Alteration in one or more cognitive spheres, through evidence of low performance in one or more cognitive

categories:

- Amnesic presentation
- Non-amnesic presentation: language impairment, visuospatial or executive.

1.2. With other criteria that increase the level of certainty

1.2.1. With documented cognitive impairment, especially with previous neuropsychological study.

1.2.2. Evidence of a genetic mutation (APP, PSEN1, PSEN2).

2. WITH EVIDENCE OF THE PATHOPHYSIOLOGICAL PROCESS

In people who meet the clinical criteria for probable AD, the use of biomarkers when one of deposition of β-amyloid protein (CSF or PET) and another of neuronal degeneration (tau to LCR, PET-FDG or structural MRI) are positive, may increase the certainty that the basis of the dementia syndrome is the pathophysiological process of AD.

spheres, with respect to what is expected for their age and educational level.

1.3. Preservation of independence in functional capacities.

1.4. Absence of dementia.

2. MCI DUE TO HIGH PROBABILITY AD

The patient meets the clinical criteria for MCI, but also has positive biomarkers for Aβ and neurodegeneration.

3. MCI DUE TO INTERMEDIATE PROBABILITY AD.

The patient meets clinical criteria of MCI and also has a biomarker of positive Aβ deposit, but those of neurodegeneration not examined or a biomarker of positive neurodegeneration and those of Aβ deposit not examined.

4. MCI BECAUSE OF UNLIKELY AD.

Patients who meet clinical criteria for MCI but with both types of negative biomarkers.

PRECLINICAL ALZHEIMER'S DEMENTIA	PROVEN ALZHEIMER'S DEMENTIA
Stage 1. Asymptomatic cerebral amyloidosis. Patients with evidence of brain Aβ protein accumulation but without evidence of	When patients meet the clinical and cognitive criteria for AD, and the neuropathological study demonstrates the presence of AD pathology.

neurodegeneration data or cognitive or behavioural symptomatology.

Stadium 2. Positive amyloidosis and evidence of synaptic dysfunction and/or incipient neurodegeneration.

They are individuals with evidence of positivity for amyloid and presence of one or more neurodegeneration markers.

Stadium 3. Positive amyloidosis with evidence of neurodegeneration and subtle cognitive impairment

POSSIBLE ALZHEIMER'S DEMENTIA	UNLIKELY ALZHEIMER'S DEMENTIA
1. WITH CLINICAL CRITERIA 1.1. Due to atypical evolution. Due to the presence of rapid onset of cognitive alterations, insufficient historical details or insufficient objective documentation of progressive deterioration 1.2. By mixed etiological presentation. In people who meet clinical AD criteria but present evidence of a) concomitant cerebrovascular disease, b) findings compatible with dementia with Lewy bodies or c) evidence of other neurological or non-neurological diseases as well as medications that may affect cognition. **2. WITH EVIDENCE OF THE PATHOPHYSIOLOGICAL PROCESS** In people who meet the clinical criteria for a non-AD dementia but have evidence of a	1. It does not meet the clinical criteria for AD. 2. It meets one of the following criteria: a) Despite meeting clinical criteria of possible or probable AD, there is sufficient evidence for an alternative diagnosis such as HIV dementia, Huntington's disease dementia, or others that can rarely be mixed with AD. b) Despite meeting the clinical criteria for possible AD, the biomarkers of Aβ and neurodegeneration are negative.

pathophysiological process of AD using biomarkers (with alteration in both categories) or meet the neuropathological criteria for AD.

Note. Adapted from *Guía oficial de práctica clínica en demencia* (p.28-29), by Manzano et al. (2018)

9.3. TABLE 3

Table 3

2014 IWG-2 diagnostic criteria

IWG-2 CRITERIA (2014)	
Typical Alzheimer's disease (A + B at any stage)	Atypical Alzheimer's disease (A + B at any stage)
A. Presence of specific clinical phenotype (first and significant impairment of episodic memory: - which is progressive over more than 6 months - objectified through a specific test for AD) B. In vivo evidence of AD pathology (decrease in Aβ1-42 protein levels, together with an increase in T-tau or P-tau in CSF; increased tracer deposit in amyloid PET or presence of an autosomal dominant AB mutation (in PSEN1, PSEN2 or APP). C. A series of exclusion criteria are added when unusual data appear in	A. Specific clinical phenotype (one of the following). - posterior variants (occipitotemporal or biparietal), - variant speech therapy, - Frontal variant - variant of Down syndrome. B. In all of them there must be at least one of the following evidence of Alzheimer's pathology: decreased levels of Aβ1-42 protein, along with an increase from T-tau or P-tau to CSF; increased tracer deposit in amyloid PET or presence of an autosomal dominant AD mutation (in PSEN1, PSEN2 or APP). C. A series of exclusion criteria are

medical records, examinations or the presence of other diseases serious enough to justify alterations in memory and related symptoms.	added when unusual data appear in medical records, examinations or the presence of other diseases serious enough to justify memory alterations and related symptoms.
Mixed Alzheimer's disease (A+B)	**Preclinical Alzheimer's disease**
A. Clinical evidence and AD biomarkers (both required): - Amnesic hippocampic syndrome or one of the clinical phenotypes of atypical AD. - Decrease from Aβ1-42 along with increased T-tau or P-tau to CSF, or increased retention of the amyloid PET tracer. B. Clinical and biomarker evidence of mixed pathology. I) For cerebrovascular disease (the following two are required): - Documented history of stroke, or focal neurological traits, or both - MRI evidence of one or more of the following: vascular lesions corresponding to the clinic, small gout disease, strategic lacunar infarctions or cerebral haemorrhages. II) For disease with Lewy bodies (the following two are required): - One of the following: extrapyramidal signs, early	**1. ASYMPTOMATIC PATIENTS AT RISK OF AD (must be given A+B):** A. Absence of specific clinical phenotype (the following two must be given) - Absence of hippocampal amnesic syndrome - Absence of any atypical AD clinical phenotype B. In vivo evidence of Alzheimer's pathology (one of the following): - Descent of Aβ1-42 along with increase from T-tau or P-tau to CSF - Increased retention of fibrillar amyloid in PET. **2. PRESYMPTOMATIC AD (A+B must be given):** A. Absence of specific clinical phenotype (the following two must be given) - Absence of hippocampal amnesic syndrome - Absence of any atypical AD clinical phenotype

hallucinations or cognitive fluctuations, plus abnormal dopamine transporter PET scan.	B. Autosomal dominant AD mutation tested in PSEN1, PSEN2 or APP, or other proven genes (including trisomy 21 of Down syndrome).
Exclusion criteria for typical AD	**Exclusion criteria for atypical AD**
A. Medical history. - Sudden start - Early onset of the following symptoms: gait disturbances, seizures, major and dominant behavioral changes. B. Clinical features. Neurological focality, early extrapyramidal signs, early hallucinations, cognitive fluctuations C. Other diseases severe enough to justify the symptoms. - Non-AD dementia, major depression, cerebrovascular disease, metabolic, inflammatory or toxic diseases that require specific research, changes in MRI FLAIR or T2 sequences in the medial temporal lobe that may be compatible with vascular or infectious lesions.	A. Medical history. - Sudden start - Alteration of early and dominant episodic memory. B. Other diseases severe enough to justify symptoms. - Major depression - Cerebrovascular disease - Metabolic, inflammatory or toxic diseases.

Note. Adapted from *Guía oficial de práctica clínica en demencia* (p.30-31), by Manzano et al. (2018)

9.4. TABLE 4

Table 4

Comparison between the different diagnostic criteria for AD

COMPARISON BETWEEN THE DIFFERENT DIAGNOSTIC CRITERIA OF THE AD		
IWG-1 Criteria (2007 + 2010)	ISA-AA criteria (2011)	IWG-2 Criteria (2014)
UNDEFINED: Clinical + pathological	TESTED: Pathophysiological	UNDEFINED: Clinical + biological + pathological
PROBABLE: -Episodic memory impairment + -Expressions of support: -RM -CSF -FART -GENETICS	PROBABLE: - With clinical criteria - With increased level of certainty POSSIBLE: - With clinical criteria PROBABLE OR POSSIBLE with evidence of the pathophysiological process of AD	TYPICAL: Specific clinical phenotype + In vivo *evidence* of AD pathology ATYPICAL: Specific clinical phenotype + In vivo *evidence* of AD pathology
AD exclusion criteria	Improbable	Exclusion criteria - Typical MA - Atypical BA
Prodromal AD	MCI due to MA	Prodromal AD
Mixed MA	Mixed DA: Clinical criteria for AD accompanied by: - Cerebrovascular	Mixed MA: - Clinical evidence and AD biomarkers +

	disease - Signs of Lewy Dementia - Other disease or situation that may affect cognition	- Clinical evidence and biomarkers of mixed pathology

Note. Adapted from *Guía oficial de práctica clínica en demencia* (p.32), by Manzano et al. (2018)

9.5. COLLABORATION DOCUMENT EDUCATIONAL COLLABORATION WITH THE ONYAR DAY CENTRE

Universitat de Girona
Facultat d'Educació
i Psicologia

Jo, Francesc Sidera Caballero, amb DNI 40338976n, professor del Departament de Psicologia de la Universitat de Girona (UdG), com a coordinador de l'assignatura "Treball final de grau (3101G01069)" del grau en psicologia de la UdG, certifico que l'estudiant Elisabet Casado Cuello, amb DNI 41595604n, és una alumna matriculada en aquesta assignatura en el curs 2023-2024. Per altra banda, donat que en el marc d'aquesta assignatura l'Elisabet Casado Cuello està duent a terme un treball d'investigació en percepció facial en pacients amb Malaltia d'Alzheimer, sol·licito la col·laboració del Centre de dia Onyar per tal que l'Elisabet pugui dur a terme una recollida de dades amb pacients del seu centre.

Per qualsevol dubte, poden em poden contactar al correu francesc.sidera@udg.edu.

Cordialment,

Girona, a 19 d'abril de 2024

Francesc Sidera Caballero

9.6. CONSENT INFORMED BY THE PARTICIPANT

Universitat de Girona
Facultat d'Educació i Psicologia

DECLARACIÓ DE CONSENTIMENT INFORMAT

TREBALL FINAL DE GRAU UNIVERSITARI EN PSICOLOGIA

Aquest document vol informar-vos sobre un treball (d'ara en endavant l'anomenarem "Estudi") al que us convidem a participar-hi. Aquest estudi el porta a terme una estudiant en el marc de l'assignatura Treball Final de Grau i ha estat aprovat pel professor responsable de l'assignatura. La nostra intenció és que rebeu la informació correcta i suficient perquè pugueu decidir si accepteu o no participar en aquest Estudi. Us demanem que llegiu aquest document amb atenció i que ens formuleu els dubtes que tingueu.

Títol de l'estudi: PERCEPCIÓ FACIAL EN MALALTIA D'ALZHEIMER: INVESTIGANT LA LÍNIA GNÒSICA DEL RECONEIXEMENT FACIAL.

Objectiu de l'estudi: En aquest Estudi el que volem és explorar si existeix un dèficit específic en la percepció visual facial en la Malaltia d'Alzheimer en comparació a la població no clínica i observar, doncs, si el dèficit en el reconeixement facial que sol aparèixer com a simptomatologia d'aquesta malaltia té relació amb un problema purament mnèsic (memorístic) o també gnòsic (perceptiu) i col·laborar a la literatura de la malaltia. Per a això, volem analitzar una sèrie de dades per tal d'avaluar aquests aspectes: d'una banda, es demanarà als participants informació sobre el seu historial clínic en relació amb el seu diagnòstic i exploració prèvia feta per a determinar-lo. Seguidament, es realitzarà un *Screening* neuropsicològic consistent en la prova MMSE (*Mini-Mental State Examination*) per fer un cribratge cognitiu. Després, es passarà la prova principal de l'estudi, el BFRT-r (*Benton Face Recognition Test - Revised*) per avaluar les habilitats de percepció facial. Per últim, s'administraran dues proves sobre percepció visual (genèriques, no específiques de cares) per tal de poder comparar els resultats amb la de percepció facial i poder atribuir els resultats obtinguts a un dèficit específic facial. Aquestes dues proves són el subtest de localització de números de la bateria VOSP (*Visual Object and Space Perception Battery*) i el Test d'orientació de línies comprès dins del RBANS.

Responsable de l'estudi: Elisabet Casado Cuello

Jo, el Sr./la Sra. _____, major d'edat, amb DNI número _____ i correu electrònic _____, actuant en el meu propi nom i en representació, mitjançant el present document,

MANIFESTO QUE HE ESTAT INFORMAT/DA DE LES QÜESTIONS SEGÜENTS RELACIONADES AMB L'ESTUDI:

- La meva participació en aquest estudi és voluntària i, si en qualsevol moment vull canviar la meva decisió, puc retirar el meu consentiment en qualsevol moment.
- La participació en aquest estudi consisteix a realitzar quatre proves d'avaluació neuropsicològica: el MMSE pel cribratge cognitiu, el BFRT-r per a l'avaluació de les habilitats en percepció facial i, finalment, el subtest de localització de números de la bateria VOSP i el Test d'orientació de línies comprès dins del RBANS per a l'avaluació de les habilitats perceptives visuals genèriques. La persona responsable del tractament de les meves dades personals és l'Elisabet Casado Cuello.
- Les meves dades personals seran recollides i tractades amb finalitats exclusivament docents i de recerca i sense ànim de lucre.
- Les meves dades seran anonimitzades, de manera que no es podrà conèixer la meva identitat a partir de les dades que es recullin.
- Es guardarà secret sobre la informació personal que facilito, i només s'usarà amb finalitat docent i de recerca en el marc d'aquest Estudi, de manera que no se'm pugui identificar als resultats de l'Estudi.
- Seguint el principi de minimització, únicament es recolliran les dades mínimes que siguin necessàries per dur a terme l'estudi, i un cop hagi acabat la finalitat docent o de recerca que es derivi d'aquest estudi, es destruirà tota la informació de caràcter personal que hi hagi facilitat de manera definitiva.

Universitat de Girona
Facultat d'Educació i Psicologia

- He estat informat/da mitjançant un document escrit entregat en paper sobre l'Estudi, sobre la finalitat i sobre les dades que es recolliran, i he consentit participar en aquest Estudi.

- En cas que l'Estudi requereixi recollir dades d'imatge/so/vídeo, aquestes dades es recolliran a través dels mitjans d'enregistrament que utilitzi l'estudiant, i només es faran servir per tal de realitzar la investigació en el marc de l'Estudi. Aquests enregistraments només duraran el temps necessari i indispensable per a l'elaboració del treball, i no rebré cap contraprestació econòmica.

- En cas que l'Estudi requereixi recollir dades d'imatge/so/vídeo, aquestes s'usaran per a l'Estudi respectant la normativa aplicable i en cap cas suposaran una intromissió il·legítima ni una vulneració dels drets al meu honor, intimitat personal i pròpia imatge.

- El tractament de les dades de caràcter personal de tots els participants s'ajustarà al que es disposa al Reglament General de Protecció de Dades (UE) 2016/679 i a la Llei Orgànica 3/2018 de Protecció de Dades Personals i Garantia dels Drets Digitals. D'acord amb el que estableix aquesta legislació, podeu exercir els drets d'accés, modificació, oposició i supressió de les vostres dades de caràcter personal dirigint-vos a la persona Responsable del tractament, identificat a continuació, a través dels canals de contacte establerts.

AUTORITZACIÓ PER PARTICIPAR A L'ESTUDI

Autoritzo a la Sra. Elisabet Casado Cuello, Responsable de l'estudi, amb DNI número 41595604N i correu electrònic personal casadoelisabet@gmail.com , estudiant de l'assignatura Treball Final de Grau (TFG) del Grau en Psicologia de la Universitat de Girona (UdG) perquè tracti les meves dades de caràcter personal facilitades per a la realització de la investigació descrita en el marc de l'Estudi indicat. A la taula següent es resumeix de manera esquemàtica com es tractaran aquestes dades:

Informació bàsica sobre protecció de dades personals	
Responsable del tractament	Elisabet Casado Cuello Correu electrònic: casadoelisabet@gmail.com Tel. 691 20 07 32
Finalitats	- Dur a terme les activitats de recerca detallades en el marc de l'estudi - Només si ens ho autoritzeu, gestionar l'autorització d'ús de la vostra imatge i utilitzar el material fotogràfic i audiovisual que contingui la vostra imatge i veu en el marc de l'Estudi.
Legitimització	- Consentiment de l'/la interessat/da. - En cas que es recullin imatges, el vostre consentiment per l'ús de la vostra imatge/vídeo/so
Destinataris	Les seves dades seran utilitzades únicament per Elisabet Casado Cuello i no es comunicaran a tercers sense el seu consentiment, excepte en els supòsits previstos per la llei.
Drets dels interessats	Podreu exercir el vostre dret d'accés, rectificació, suspensió, oposició, portabilitat i limitació enviant un correu electrònic a casadoelisabet@gmail.com , adjuntant una fotocòpia del DNI o document acreditatiu de la vostra identitat.
Informació addicional	Podeu revisar la informació addicional sobre el tractament de les dades personals a l'apartat següent.

A _____, a ___de _____20

El Sr./La Sra. _____ (nom i signatura)

AUTORITZACIÓ PER A L'ÚS DE L'IMATGE/SO/VÍDEO

Universitat de Girona
Facultat d'Educació i Psicologia

Per tal de dur a terme la recerca en el marc de l'Estudi i l'elaboració del treball, caldrà fer enregistraments audiovisuals de la imatge i veu de les persones que participen a l'Estudi.

Fent una creu al requadre següent, dono permís a Elisabet Casado Cuello per fer ús del material audiovisual que reculli durant la realització de les activitats que formin part de l'Estudi en què seré partícip i on aparegui, en virtut de l'Estudi, la meva imatge i /o la meva veu, durant el temps necessari i indispensable per a l'elaboració de l'Estudi i sense cap contraprestació econòmica.

L'estudiant es compromet a que la utilització d'aquestes imatges respecti la normativa aplicable i que en cap cas no suposi una intromissió il·legítima ni una vulneració dels drets a l'honor, intimitat personal i pròpia imatge dels participants.

☐ Autoritzo l'ús de la meva imatge en els termes indicats.
☐ Autoritzo l'ús de la meva veu en els termes indicats.

A _____, a _de _____20

El Sr./La Sra. _____ (nom i signatura)

9.7. INFORMED CONSENT: VERSION OF LEGAL REPRESENTATIVE

Universitat de Girona
Facultat d'Educació i Psicologia

DECLARACIÓ DE CONSENTIMENT INFORMAT

TREBALL FINAL DE GRAU UNIVERSITARI EN PSICOLOGIA

Aquest document vol informar-vos sobre un treball (d'ara en endavant l'anomenarem "Estudi") al que us convidem a participar-hi. Aquest estudi el porta a terme una estudiant en el marc de l'assignatura Treball Final de Grau i ha estat aprovat pel professor responsable de l'assignatura. La nostra intenció és que rebeu la informació correcta i suficient perquè pugueu decidir si accepteu o no participar en aquest Estudi. Us demanem que llegiu aquest document amb atenció i que ens formuleu els dubtes que tingueu.

Títol de l'estudi: PERCEPCIÓ FACIAL EN MALALTIA D'ALZHEIMER: INVESTIGANT LA LÍNIA GNÒSICA DEL RECONEIXEMENT FACIAL.

Objectiu de l'estudi: En aquest Estudi el que volem és explorar si existeix un dèficit específic en la percepció visual facial en la Malaltia d'Alzheimer en comparació a la població no clínica i observar, doncs, si el dèficit en el reconeixement facial que sol aparèixer com a simptomatologia d'aquesta malaltia té relació amb un problema purament mnèsic (memorístic) o també gnòsic (perceptiu) i col·laborar a la literatura de la malaltia. Per a això, volem analitzar una sèrie de dades per tal d'avaluar aquests aspectes: d'una banda, es demanarà als participants informació sobre el seu historial clínic en relació amb el seu diagnòstic i exploració prèvia feta per a determinar-lo. Seguidament, es realitzarà un *Screening* neuropsicològic consistent en la prova MMSE (*Mini-Mental State Examination*) per fer un cribratge cognitiu. Després, es passarà la prova principal de l'estudi, el BFRT-r (*Benton Face Recognition Test - Revised*) per avaluar les habilitats de percepció facial. Per últim, s'administraran dues proves sobre percepció visual (genèriques, no específiques de cares) per tal de poder comparar els resultats amb la de percepció facial i poder atribuir els resultats obtinguts a un dèficit específic facial. Aquestes dues proves són el subtest de localització de números de la bateria VOSP (*Visual Object and Space Perception Battery*) i el Test d'orientació de línies comprès dins del RBANS.

Responsable de l'estudi: Elisabet Casado Cuello

Jo, el Sr./la Sra. _____, major d'edat, amb DNI número _____ i correu electrònic_____, actuant en nom i representació legal del Sr./de la Sra. _____ , mitjançant el present document,

MANIFESTO QUE HE ESTAT INFORMAT/DA DE LES QÜESTIONS SEGÜENTS RELACIONADES AMB L'ESTUDI:

- La meva participació en aquest estudi és voluntària i, si en qualsevol moment vull canviar la meva decisió, puc retirar el meu consentiment en qualsevol moment.
- La participació en aquest estudi consisteix a realitzar quatre proves d'avaluació neuropsicològica: el MMSE pel cribratge cognitiu, el BFRT-r per a l'avaluació de les habilitats en percepció facial i, finalment, el subtest de localització de números de la bateria VOSP i el Test d'orientació de línies comprès dins del RBANS per a l'avaluació de les habilitats perceptives visuals genèriques. La persona responsable del tractament de les meves dades personals és l'Elisabet Casado Cuello.
- Les meves dades personals seran recollides i tractades amb finalitats exclusivament docents i de recerca i sense ànim de lucre.
- Les meves dades seran anonimitzades, de manera que no es podrà conèixer la meva identitat a partir de les dades que es recullin.
- Es guardarà secret sobre la informació personal que facilito, i només s'usarà amb finalitat docent i de recerca en el marc d'aquest Estudi, de manera que no se'm pugui identificar als resultats de l'Estudi.
- Seguint el principi de minimització, únicament es recolliran les dades mínimes que siguin necessàries per dur a terme l'estudi, i un cop hagi acabat la finalitat docent o de recerca que es derivi d'aquest estudi, es destruirà tota la informació de caràcter personal que hi hagi facilitat de manera definitiva.

Universitat de Girona
Facultat d'Educació i Psicologia

- He estat informat/da mitjançant un document escrit entregat en paper sobre l'Estudi, sobre la finalitat i sobre les dades que s'han fet recolliran, i he consentit participar en aquest Estudi.

- En cas que l'Estudi requereixi recollir dades d'imatge/so/vídeo, aquestes dades es recolliran a través dels mitjans d'enregistrament que utilitzi l'estudiant, i només es faran servir per tal de realitzar la investigació en el marc de l'Estudi. Aquests enregistraments només duraran el temps necessari i indispensable per a l'elaboració del treball, i no rebré cap contraprestació econòmica.

- En cas que l'Estudi requereixi recollir dades d'imatge/so/vídeo, aquestes s'usaran per a l'Estudi respectant la normativa aplicable i en cap cas suposaran una intromissió il·legítima ni una vulneració dels drets al meu honor, intimitat personal i pròpia imatge.

- El tractament de les dades de caràcter personal de tots els participants s'ajustarà al que es disposa al Reglament General de Protecció de Dades (UE) 2016/679 i a la Llei Orgànica 3/2018 de Protecció de Dades Personals i Garantia dels Drets Digitals. D'acord amb el que estableix aquesta legislació, podeu exercir els drets d'accés, modificació, oposició i supressió de les vostres dades de caràcter personal dirigint-vos a la persona Responsable del tractament, identificat a continuació, a través dels canals de contacte establerts.

AUTORITZACIÓ PER PARTICIPAR A L'ESTUDI

Autoritzo a la Sra. Elisabet Casado Cuello, Responsable de l'estudi, amb DNI número 41595604N i correu electrònic personal casadoelisabet@gmail.com , estudiant de l'assignatura Treball Final de Grau (TFG) del Grau en Psicologia de la Universitat de Girona (UdG) perquè tracti les meves dades de caràcter personal facilitades per a la realització de la investigació descrita en el marc de l'Estudi indicat. A la taula següent es resumeix de manera esquemàtica com es tractaran aquestes dades:

Informació bàsica sobre protecció de dades personals	
Responsable del tractament	Elisabet Casado Cuello Correu electrònic: casadoelisabet@gmail.com Tel. 691 20 07 32
Finalitats	- Dur a terme les activitats de recerca detallades en el marc de l'estudi - Només si ens ho autoritzeu, gestionar l'autorització d'ús de la vostra imatge i utilitzar el material fotogràfic i audiovisual que contingui la vostra imatge i veu en el marc de l'Estudi.
Legitimització	- Consentiment de l'/la interessat/da. - En cas que es recullin imatges, el vostre consentiment per l'ús de la vostra imatge/vídeo/so
Destinataris	Les seves dades seran utilitzades únicament per Elisabet Casado Cuello i no es comunicaran a tercers sense el seu consentiment, excepte en els supòsits previstos per la llei.
Drets dels interessats	Podreu exercir el vostre dret d'accés, rectificació, suspensió, oposició, portabilitat i limitació enviant un correu electrònic a casadoelisabet@gmail.com , adjuntant una fotocòpia del DNI o document acreditatiu de la vostra identitat.
Informació addicional	Podeu revisar la informació addicional sobre el tractament de les dades personals a l'apartat següent.

A _____, a ___de _____20

El Sr./La Sra. _____ (nom i signatura)

Universitat de Girona
Facultat d'Educació i Psicologia

AUTORITZACIÓ PER A L'ÚS DE L'IMATGE/SO/VÍDEO

Per tal de dur a terme la recerca en el marc de l'Estudi i l'elaboració del treball, caldrà fer enregistraments audiovisuals de la imatge i veu de les persones que participen a l'Estudi.

Fent una creu al requadre següent, dono permís a Elisabet Casado Cuello per fer ús del material audiovisual que reculli durant la realització de les activitats que formin part de l'Estudi en què seré partícip i on aparegui, en virtut de l'Estudi, la meva imatge i /o la meva veu, durant el temps necessari i indispensable per a l'elaboració de l'Estudi i sense cap contraprestació econòmica.

L'estudiant es compromet a que la utilització d'aquestes imatges respecti la normativa aplicable i que en cap cas no suposi una intromissió il·legítima ni una vulneració dels drets a l'honor, intimitat personal i pròpia imatge dels participants.

☐ Autoritzo l'ús de la meva imatge en els termes indicats.
☐ Autoritzo l'ús de la meva veu en els termes indicats.

A _____, a _de _____20

El Sr./La Sra. _____ (nom i signatura)

9.8. INFORMATION SHEET FOR THE PARTICIPANT

Universitat de Girona
Facultat d'Educació i Psicologia

PERCEPCIÓ FACIAL EN LA MALALTIA D'ALZHEIMER: INVESTIGANT LA LÍNIA GNÒSICA DEL RECONEIXEMENT FACIAL

El present Estudi en què doneu el vostre consentiment per a participar consisteix en un **Treball Final de Grau en Psicologia** realitzat per l'estudiant **Elisabet Casado Cuello** de la Universitat de Girona.

Aquest estudi té per objectiu investigar si els **dèficits** que es donen en el **reconeixement de les cares familiars** en les persones amb **Malaltia d'Alzheimer** tenen a veure amb un **dèficit específic en la percepció visual de tipus facial**. Se sap que el paper de la memòria hi té un gran pes, però el factor perceptiu encara se segueix estudiant. És per això, que per tal d'investigar-ho l'estudiant ha plantejat un treball on, la part pràctica consisteix a aplicar unes **proves neuropsicològiques** a una **població clínica amb Malaltia d'Alzheimer** i a una **població no clínica**, per tal de comparar els resultats i veure si existeixen unes diferències específiques que recolzin la hipòtesi del dèficit perceptiu facial en les persones amb la malaltia esmentada.

Les proves que s'administraran són, en primer lloc, el **MMSE** (*Mini-Mental State Examination*) per fer un cribratge cognitiu. Després, es passarà la prova principal de l'estudi, el **BFRT-r** (*Benton Face Recognition Test - Revised*) per avaluar les habilitats de percepció facial. Finalment, s'administraran dues proves sobre percepció visual (genèriques, no específiques de cares) per tal de poder comparar els resultats amb la de percepció facial i poder atribuir els resultats obtinguts a un dèficit específic amb les cares, no a un dèficit genèric en la percepció. Aquestes dues proves són el **subtest de localització de números de la bateria VOSP** (*Visual Object and Space Perception Battery*) i el **Test d'orientació de línies** comprès al RBANS.

Per a qualsevol dubte o consulta, us podeu comunicar sempre que ho desitgeu amb la responsable de l'estudi a través dels següents mitjans:

Telèfon: 691 20 07 32 - Elisabet Casado Cuello

Correu electrònic: casadoelisabet@gmail.com

Aquest estudi quedarà totalment tancat al mes de juny del present any 2024, de manera que si desitgeu conèixer quins han estat els resultats finals trobats gràcies a la vostra participació, podreu demanar que se us comparteixi una còpia del treball via correu electrònic a l'adreça especificada anteriorment.

Moltes gràcies per la col·laboració!

9.9. TABLE OF THE PARTICIPANTS' DATASHEET

DADES DELS PARTICIPANTS:

	MA/C	Nom i cognoms	Codi	Dades de contacte
1				
2				
3				
4				

9.10. CLINICAL SHEET

DADES PERSONALS					
Codi de participant:		Edat:		Temps:	
Data de naixement:		Lateralitat:			
Natural:		Llengua materna:			
Professió:		Anys a Catalunya:			
Anys d'educació:	1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20		Elemental Batxillerat Diplomat/da Llicenciat/da o doctor/a		
Data d'exploració:		Lloc de reclutament per l'estudi:	☐ Centre de dia Onyar ☐ Centre cívic Montilivi ☐ Altres:		
HISTORIAL CLÍNIC					
Diagnòstic de MA:	☐ Sí ☐ No	Data d'inici dels símptomes de la malaltia:			
GDS:		CDR:			
Altres observacions:					

SCREENING NEUROLÒGIC INICIAL							
Test	Puntuació	Barems normals	Ajust MMSE				
MMSE	/30	25-30 No deteriorament ≤ 24 Probable deteriorating cognitiu			Edat		
IDDD	/99	33			≤50	51-75	>75
GDS Depressió	/30 /15	0-10 no depressió 11-20 depressió lleu 21-30 depressió greu	Educació	≤8	0	+1	+2
				9-17	-1	0	+1
				>17	-2	-1	0

Nota. Finalment l'IDDD i el GDS depressió es van descartar per a l'*screening* inicial. Només es va utilitzar el MMSE.

PROVES		
Prova	Puntuació	Barems
BFRT-r	/54	41-54 normal 39-40 puntuació límit 37-38 discapacitat moderada 0-36 deteriorament clar
Subtest de localització de números de la bateria VOSP	/10	Puntuació escalar ajustada per edat i educació:
Judgement of Line Orientation	/20	Puntuació escalar ajustada per edat i educació:

9.11. ANSWER SHEET

BFRT-r:

TRIAL 1: 1 2 3 4 5 6

TRIAL 2: 1 2 3 4 5 6

TRIAL 3: 1 2 3 4 5 6

TRIAL 4: 1 2 3 4 5 6

TRIAL 5: 1 2 3 4 5 6

TRIAL 6: 1 2 3 4 5 6

TRIAL 7: 1 2 3 4 5 6

TRIAL 8: 1 2 3 4 5 6

TRIAL 9: 1 2 3 4 5 6

TRIAL 10: 1 2 3 4 5 6

TRIAL 11: 1 2 3 4 5 6

TRIAL 12: 1 2 3 4 5 6

TRIAL 13: 1 2 3 4 5 6

TRIAL 14: 1 2 3 4 5 6

TRIAL 15: 1 2 3 4 5 6

TRIAL 16: 1 2 3 4 5 6

TRIAL 17: 1 2 3 4 5 6

TRIAL 18: 1 2 3 4 5 6

TRIAL 19: 1 2 3 4 5 6

TRIAL 20: 1 2 3 4 5 6

TRIAL 21: 1 2 3 4 5 6

TRIAL 22: 1 2 3 4 5 6

Elisabet Casado Cuello – Treball Final de Grau en Psicologia 2024

Test de localització de números de la Bateria VOSP:

PROVA 01: 1 2 3 4 5 6 7 8 9

PROVA 02: 1 2 3 4 5 6 7 8 9

LÀMINA 1: 1 2 3 4 5 6 7 8 9

LÀMINA 2: 1 2 3 4 5 6 7 8 9

LÀMINA 3: 1 2 3 4 5 6 7 8 9

LÀMINA 4: 1 2 3 4 5 6 7 8 9

LÀMINA 5: 1 2 3 4 5 6 7 8 9

LÀMINA 6: 1 2 3 4 5 6 7 8 9

LÀMINA 7: 1 2 3 4 5 6 7 8 9

LÀMINA 8: 1 2 3 4 5 6 7 8 9

LÀMINA 9: 1 2 3 4 5 6 7 8 9

LÀMINA 10: 1 2 3 4 5 6 7 8 9

Universitat de Girona
Facultat d'Educació i Psicologia

Elisabet Casado Cuello – Treball Final de Grau en Psicologia 2024

Test d'orientació de línies del RBANS

4 Orientación de líneas

Tiempo límite: 20 segundos/unidad

Muestre el ejemplo de unidad y diga ***Estas dos líneas de aquí*** (indicar) ***se corresponden con dos de las líneas de arriba. ¿Puede decirme los números, o señalar las líneas con las que se corresponden?*** Corrija cualquier error y asegúrese de que el examinado comprenda la tarea. Continúe con las unidades 1 a 10.

Puntuación: 1 punto por cada línea correctamente identificada.

Unidad	Respuestas	Respuestas correctas	Puntuación (0, 1 ó 2)
Muestra		1, 7	
1.		10, 12	
2.		4, 11	
3.		6, 9	
4.		8, 13	
5.		2, 4	

Unidad	Respuestas	Respuestas correctas	Puntuación (0, 1 ó 2)
6.		1, 6	
7.		3, 10	
8.		5, 8	
9.		1, 3	
10.		11, 13	
		Rango de puntuación total = 0 – 20	

9.12. CORRECTION SHEET

BFRT-r:

TRIAL 1: 3

TRIAL 2: 1

TRIAL 3: 5

TRIAL 4: 6

TRIAL 5: 4

TRIAL 6: 1

TRIAL 7: 2 4 5

TRIAL 8: 1 5 6

TRIAL 9: 3 4 6

TRIAL 10: 1 2 3

TRIAL 11: 3 5 6

TRIAL 12: 1 2 6

TRIAL 13: 2 5 6

TRIAL 14: 3 4 6

TRIAL 15: 3 4 5

TRIAL 16: 2 3 6

TRIAL 17: 3 4 6

TRIAL 18: 2 5 6

TRIAL 19: 1 2 6

TRIAL 20: 2 3 6

TRIAL 21: 3 4 5

TRIAL 22: 1 3 4

Elisabet Casado Cuello – Treball Final de Grau en Psicologia 2024

Test de localització de números de la Bateria VOSP:

PROVA 01: 1

PROVA 02: 5

LÀMINA 1: 7

LÀMINA 2: 4

LÀMINA 3: 3

LÀMINA 4: 7

LÀMINA 5: 8

LÀMINA 6: 2

LÀMINA 7: 6

LÀMINA 8: 4

LÀMINA 9: 8

LÀMINA 10: 5

Test d'orientació de línies del RBANS

Unidad	Respuestas	Respuestas correctas	Puntuación (0, 1 ó 2)
Muestra		1, 7	
1.		10, 12	
2.		4, 11	
3.		6, 9	
4.		8, 13	
5.		2, 4	

Unidad	Respuestas	Respuestas correctas	Puntuación (0, 1 ó 2)
6.		1, 6	
7.		3, 10	
8.		5, 8	
9.		1, 3	
10.		11, 13	
		Rango de puntuación total = 0 – 20	

9.13. DATA MATRIX

Figures 1 and 2

Matrix data view

Participant	Sexe	Edat	MA	GDS	MMSE	BFRTr	VOSP	OL	Temps	Natural
1	1	77	2	1	29	41	8	15	30	Jaén
2	1	80	2	1	27	36	10	14	32	Catalunya
3	1	84	2	1	30	37	8	17	30	Cartagena
4	1	61	1	3	24	31	5	17	30	Catalunya
5	1	84	1	5	21	31	10	9	30	Sevilla
6	1	94	1	3	21	24	3	17	30	Teruel
7	2	81	1	4	20	28	3	17	30	Catalunya
8	1	82	1	5	22	31	10	20	30	Jaén
9	1	74	2	1	30	37	8	20	30	Granada
10	2	77	2	1	29	42	10	20	30	Burgos

Professió	Lateralitat	Llengua	AnysCAT	AnysEd	LlocRec
Personal de neteja	3	2	49	3	Centre cívic Montilivi
Cap	1	1	80	3	Centre cívic Montilivi
Mestra i bibliotecària	1	2	76	6	Centre cívic Montilivi
Metgessa	1	2	61	6	Centre cívic Montilivi
Personal de neteja	1	2	70	1	Centre de dia Onyar
Mestressa de casa	1	2	6	2	Centre de dia Onyar
Venedor	1	1	81	3	Centre de dia Onyar
Agricultora	3	2	40	1	Centre de dia Onyar
Personal de neteja	1	2	60	2	Centre cívic Montilivi
Venedor	1	2	60	5	Centre cívic Montilivi

Figures 3 and 4

Matrix variable view

	Nombre	Tipo	Anchura	Decimales	Etiqueta	Valores	Perdidos
1	Participant	Numérico	8	0	Participant	Ninguna	Ninguna
2	Sexe	Numérico	8	0	Sexe	{1, Dona}...	Ninguna
3	Edat	Numérico	8	0	Edat	Ninguna	Ninguna
4	MA	Numérico	8	0	Diagnòstic de Malaltia d'Alzheimer	{1, MA sí}...	Ninguna
5	GDS	Numérico	8	0	Escala de Deteriorament Global (GDS)	{1, GDS 1: Absència d'al...	Ninguna
6	MMSE	Numérico	8	0	Mini Mental State Examination	Ninguna	Ninguna
7	BFRTr	Numérico	8	0	Benton face recognition test - revised	Ninguna	Ninguna
8	VOSP	Numérico	8	0	Subtest de localització de números de la bateria VOSP	Ninguna	Ninguna
9	OL	Numérico	8	0	Test d'orientació de línies del RBANS	Ninguna	Ninguna
10	Temps	Numérico	8	0	Temps d'examen	Ninguna	Ninguna
11	Natural	Cadena	30	0	Lloc de naixement	Ninguna	Ninguna
12	Professió	Cadena	30	0	Professió	Ninguna	Ninguna
13	Lateralitat	Numérico	8	0	Lateralitat manual	{1, Dreta}...	Ninguna
14	Llengua	Numérico	8	0	Llengua materna	{1, Català}...	Ninguna
15	AnysCAT	Numérico	8	0	Anys a Catalunya	Ninguna	Ninguna
16	AnysEd	Numérico	30	0	Anys d'educació	{1, NE: no escolaritzat}...	Ninguna
17	LlocRec	Cadena	30	0	Lloc de reclutament dels participants	Ninguna	Ninguna

Columnas	Alineación	Medida	Rol
11	Derecha	Nominal	Entrada
7	Derecha	Escala	Entrada
7	Derecha	Escala	Entrada
6	Derecha	Nominal	Entrada
6	Derecha	Escala	Entrada
8	Derecha	Escala	Entrada
8	Derecha	Escala	Entrada
8	Derecha	Escala	Entrada
6	Derecha	Escala	Entrada
8	Derecha	Escala	Entrada
9	Izquierda	Nominal	Entrada
16	Izquierda	Nominal	Entrada
10	Derecha	Nominal	Entrada
9	Derecha	Nominal	Entrada
9	Derecha	Escala	Entrada
10	Derecha	Escala	Entrada
17	Izquierda	Nominal	Entrada

9.14. ASSESSMENT INSTRUMENTS

9.14.1. MMSE

INSTITUTO NACIONAL DE GERIATRÍA

Mini-examen del estado mental

Objetivo:
Detectar deterioro cognitivo leve o demencia.

Descripción:
Esta versión del mini-examen del estado mental que adaptó y validó Sandra Reyes de Beaman y colaboradores en población mexicana, se realizaron algunos cambios a saber: en orientación de tiempo se reemplazó la pregunta acerca de la estación del año por la hora, considerándose correctas respuestas entre 0.5 horas antes o después de la hora exacta real; en orientación en espacio, la pregunta ¿cuál es el nombre de este hospital? solo se le hace a personas que se encuentran hospitalizadas, en otros contextos se cambia por ¿en dónde estamos ahora?, este dominio también considera la colonia o vecindario y la ciudad; las palabras a registrar y recordar se cambiaron por papel, bicicleta y cuchara; en atención y cálculo se mantuvo la resta de 7 en 7 empezando por 100, dejando como alternativa la resta de 3 en 3 empezando por 20; en lenguaje la frase a repetir es ni no, ni si, ni pero. Otro ajuste que se realizó a esta versión fue que en personas con 3 o menos años de escolaridad formal, se les asigna 8 puntos, y no se aplican los reactivos de la resta de 7 en 7, leer la orden escrita "Cierre los ojos", escribir una frase o enunciado, no copiar el dibujo de los pentágonos; lo cual permite utilizar el mismo valor de corte para identificar posible deterioro cognitivo con 24 o menos puntos del mini- examen del estado mental.

Es una prueba cuya puntuación está altamente influenciada por la escolaridad y la edad. Aquellas personas que obtengan una puntuación baja requieren una evaluación clínica y neuropsicológica más exhaustiva para confirmar y determinar el grado de deterioro cognitivo.

Requerimientos:
- Formato del mini-examen del estado mental.
- Bolígrafo.
- Hoja de papel para la orden verbal.
- Espacio privado, ventilado, iluminado, libre de distracciones.

Tiempo de aplicación: 10 minutos.

Instrucciones:

1. Vea directamente a la persona, logre su atención y explíquele: “Le voy a hacer algunas preguntas para evaluar su estado mental”.
2. Pregunte si sabe leer o escribir.
3. Pregunte hasta qué año estudió en caso de “A personas con ≤ 3 años de escolaridad formal, darles 8 puntos de entrada y obviar la resta de 7 en 7 a partir de 100 (5 puntos), la lectura de “cierre los ojos” (1 punto), la esctritura de frase (1 punto) y la copia de los pentágonos (1punto)”.
4. Diga tal cual la instrucción que está establecida en cada dominio del formato.
5. Realice la sumatoria a fin de establecer el resultado final.

Calificación:

- Se dará un punto por cada respuesta correcta.

Sugerencias o pautas de Interpretación:

- **Probable deterioro cognitivo:** Puntaje < 24.
- **Sin deterioro cognitivo:** Puntaje > 24.

Referencias:

• Reyes de Baeman, S., Beaman, P. E., García Peña, C., Villa, M. A., Heres, J., Córdova, A. y Jagger, C. (2004). Validation of a Modifiied Version of the Mini-Mental State Examination (MMSE) in Spanish. Aging Neuropsychol Cong. Aging, Neuropsychology, and Cognition, 11(1), 1-11

INSTITUTO NACIONAL DE GERIATRÍA

Mini-examen del estado mental

Criterio de evaluación: Se dará un punto por cada respuesta correcta

Nombre del/a entrevistado/a: _____

Sabe leer: Sí | No

Sabe escribir: Sí | No

Hasta que año estudió: _____

	0	1	2	3	4	5	6	7	8	9
	0	1	2	3	4	5	6	7	8	9

I. Orientación

(Tiempo)

1. ¿Qué fecha es hoy?

Respuesta						Real					
Día		Mes		Año		Día		Mes		Año	
0	0	0	0	0	0	0	0	0	0	0	0
1	1	1	1	1	1	1	1	1	1	1	1
2	2	2	2	2	2	2	2	2	2	2	2
3	3	3	3	3	3	3	3	3	3	3	3
	4		4	4	4		4		4	4	4
	5		5	5	5		5		5	5	5
	6		6	6	6		6		6	6	6
	7		7	7	7		7		7	7	7
	8		8	8	8		8		8	8	8
	9		9	9	9		9		9	9	9

¿Qué día de la semana es?

Respuesta						
L	M	M	J	V	S	D

¿Qué día de la semana es?

Real						
L	M	M	J	V	S	D

¿Qué hora es aproximadamente?

Respuesta				Real			
Hr.		Min.		Hr.		Min.	
0	0	0	0	0	0	0	0
1	1	1	1	1	1	1	1
	2	2	2		2	2	2
	3	3	3		3	3	3
	4	4	4		4	4	4
	5	5	5		5	5	5
	6	6	6		6	6	6
	7		7		7		7
	8		8		8		8
	9		9		9		9

(máx. 5) 0 | 1 | 2 | 3 | 4 | 5

(Espacio)

	INC	CORR
¿En dónde estamos ahora?	0	1
¿En qué área o departamento estamos ahora?	0	1
¿Qué colonia es esta?	0	1
¿Qué ciudad es esta?	0	1
¿Qué piso es este?	0	1

(máx. 5) 0 | 1 | 2 | 3 | 4 | 5

II. Registro

3. Le voy a decir 3 objetos, cuando yo termine quiero que por favor usted repita:

Papel	Bicicleta	Cuchara

Ahora dígalos usted:

	INC	CORR
Papel	0	1
Bicicleta	0	1
Cuchara	0	1

(máx. 3) 0 | 1 | 2 | 3

III. Atención y Cálculo

4. Le voy a pedir que reste de 7 en 7 a partir del 100.

	INC	CORR	
93	0	1	_____
86	0	1	_____
79	0	1	_____
72	0	1	_____
65	0	1	_____

(máx. 5) 0 | 1 | 2 | 3 | 4 | 5

4a. Le voy a pedir que reste de 3 en 3 a partir del 20.

	INC	CORR	
17	0	1	_____
14	0	1	_____
11	0	1	_____
8	0	1	_____
5	0	1	_____

(máx. 5) 0 | 1 | 2 | 3 | 4 | 5

IV. Lenguaje

Le voy a dar algunas instrucciones. Por favor sígalas en el orden en que se las voy a decir. Sólo se las puedo decir una vez:

	INC	CORR
- TOME ESTE PAPEL CON LA MANO DERECHA	0	1
- DÓBLELO POR LA MITAD	0	1
- Y DEJELO EN EL SUELO	0	1

(máx. 3) 0 | 1 | 2 | 3

(Espacio)

Por favor haga lo que dice aquí:

Cierre los ojos

(máx. 1) 0 | 1

Quiero que por favor escriba una frase que diga un mensaje (atrás de esta hoja)

(máx. 1) 0 | 1

V. Memoria diferida

Dígame los tres objetos que le mencioné al principio:

	INC	CORR
Papel	0	1
Bicicleta	0	1
Cuchara	0	1

(máx. 3) 0 | 1 | 2 | 3

Copie, por favor, este dibujo tal como esta. (mostrar atrás de esta hoja)

(máx. 1) 0 | 1

	INC	CORR
Muestre el RELOJ y diga: ¿Qué es esto?	0	1
Muestre el LÁPIZ y diga: ¿Qué es esto?	0	1

(máx. 2) 0 | 1 | 2

Ahora le voy a decir una frase que tendrá que repetir después de mí. Sólo se la puedo decir una sola vez, así que ponga mucha atención.

NI NO, NI SÍ, NI PERO

(máx. 1) 0 | 1

Puntaje total:

	0	1	2	3						
	0	1	2	3	4	5	6	7	8	9

A personas con ≤ 3 años de escolaridad formal, darles 8 puntos de entrada y obviar la resta de 7 en 7 a partir de 100 (5 puntos), la lectura de "cierre los ojos" (1 punto), la escritura de frase (1 punto) y la copia de los pentágonos (1punto).

Interpretación:

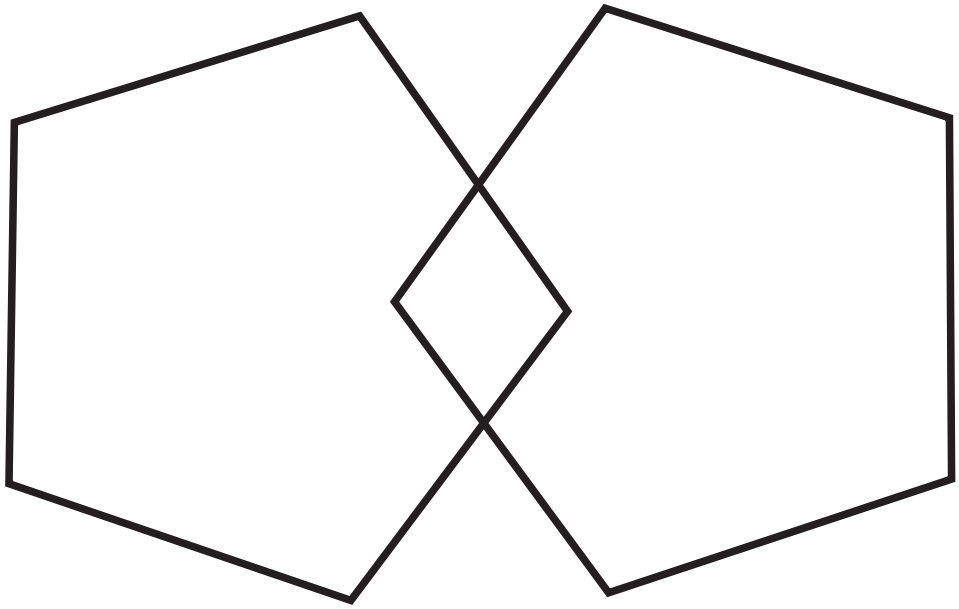
Puntaje ≤ 24 = Probable deterioro cognitivo.
Puntaje > 24 = Sin deterioro cognitivo.

Sensibilidad: 97%
Especificidad: 88%
Área bajo la curva: 0.849

INSTITUTO NACIONAL DE GERIATRÍA

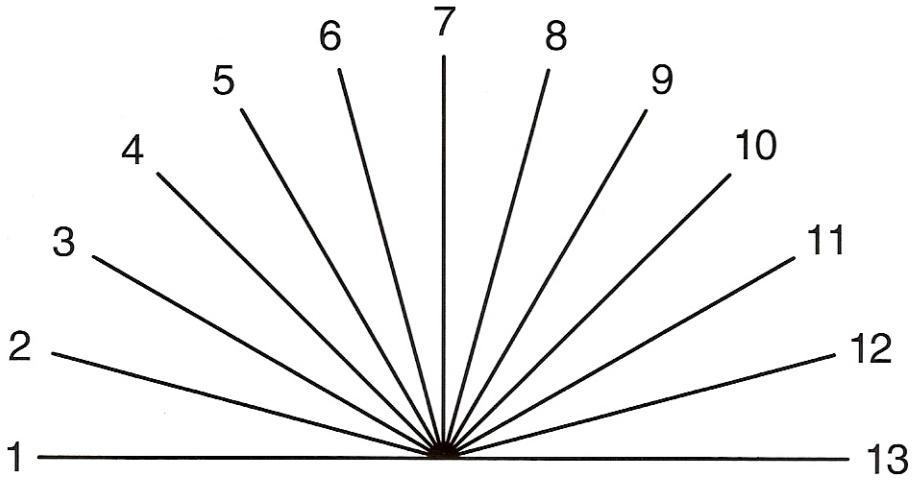
Mini-examen del estado mental

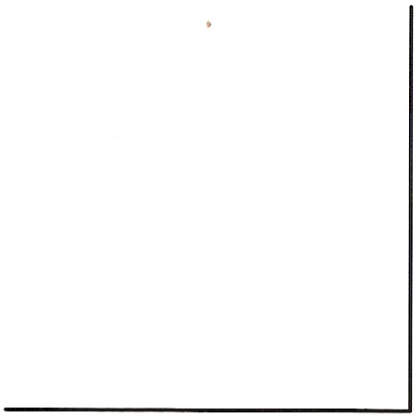
CIERRE SUS OJOS

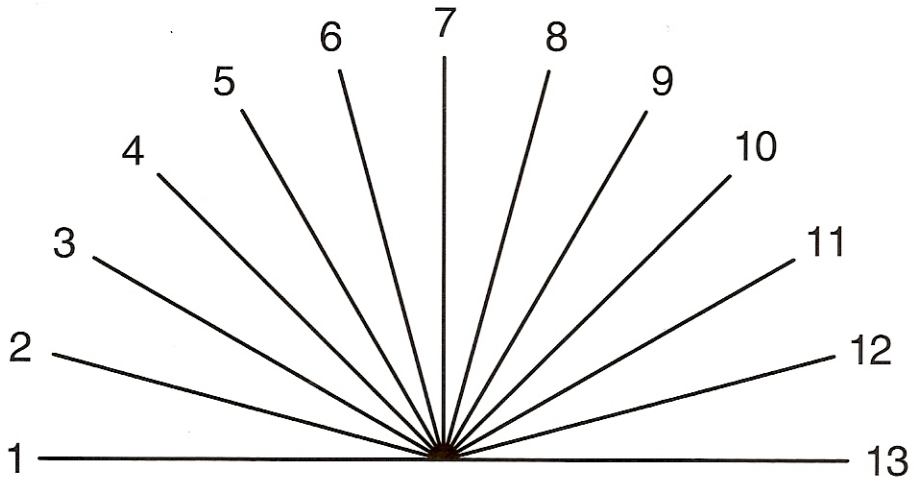


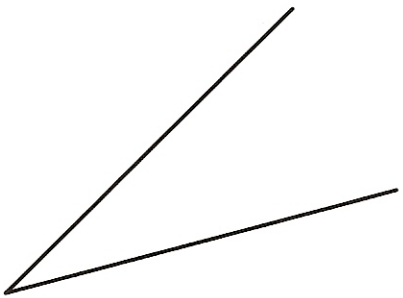
• Reyes de Baeman, S., Beaman, P. E., García Peña, C., Villa, M. A., Heres, J., Córdova, A. y Jagger, C. (2004). Validation of a Modifiied Version of the Mini-Mental State Examination (MMSE) in Spanish. Aging Neuropsychol Cong. Aging, Neuropsychology, and Cognition, 11(1), 1-11

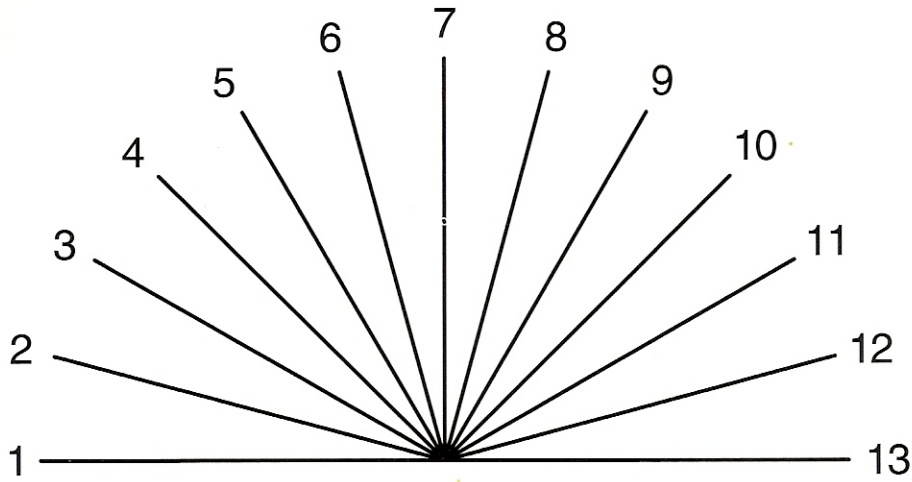
9.14.2. RBANS Battery Line Orientation Test

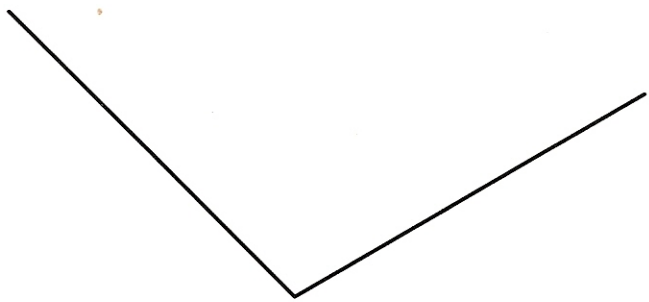

1
2
3
4
5
6
7
8
9
10
11
12
13

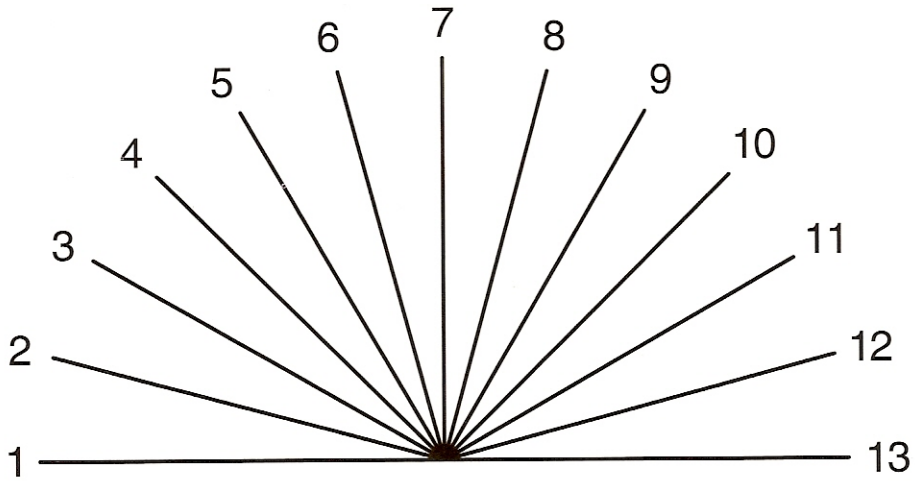


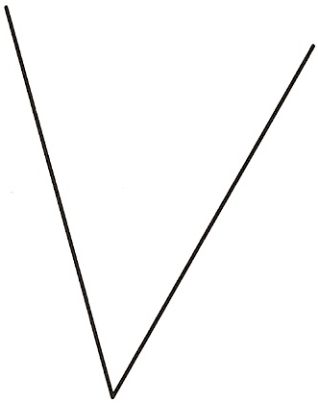

1
2
3
4
5
6
7
8
9
10
11
12
13

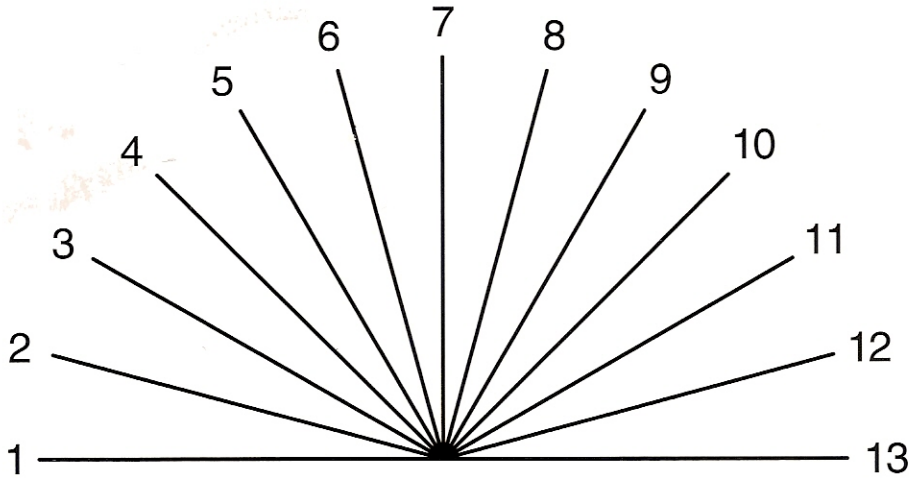


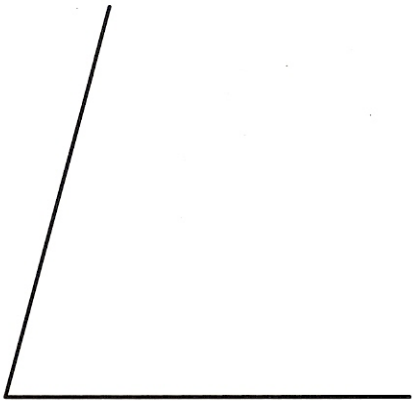

1
2
3
4
5
6
7
8
9
10
11
12
13

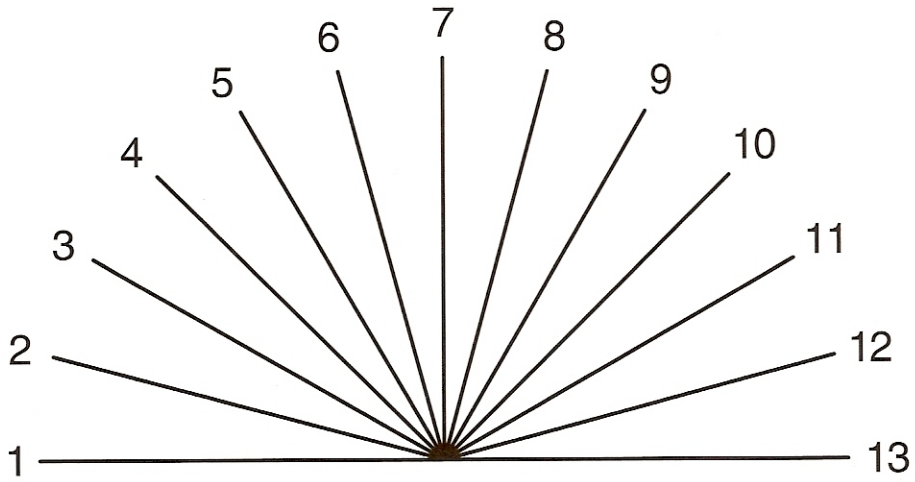


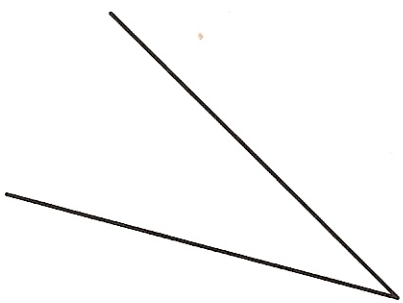

1
2
3
4
5
6
7
8
9
10
11
12
13

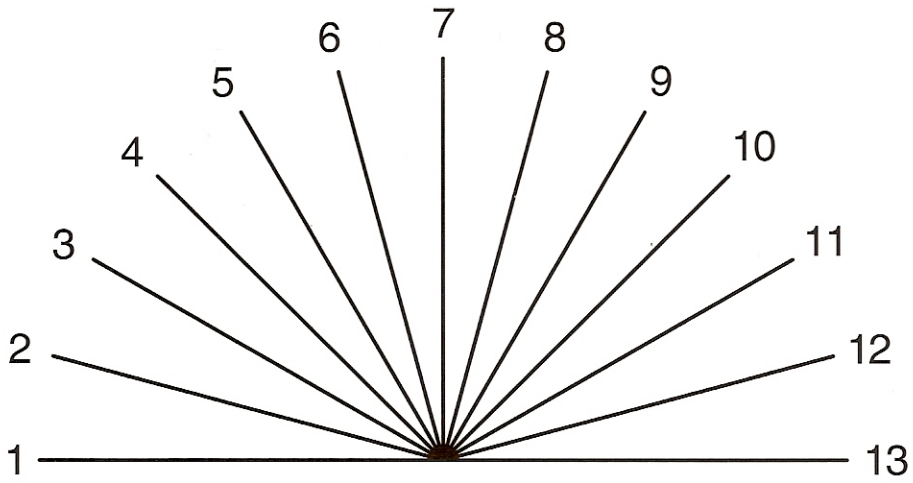


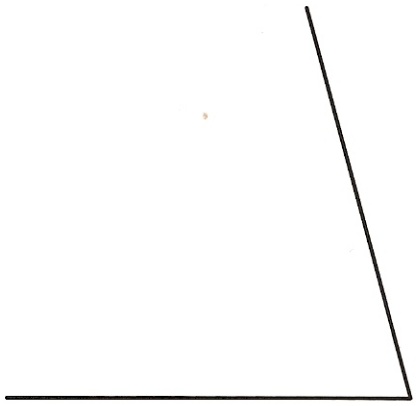

1
2
3
4
5
6
7
8
9
10
11
12
13

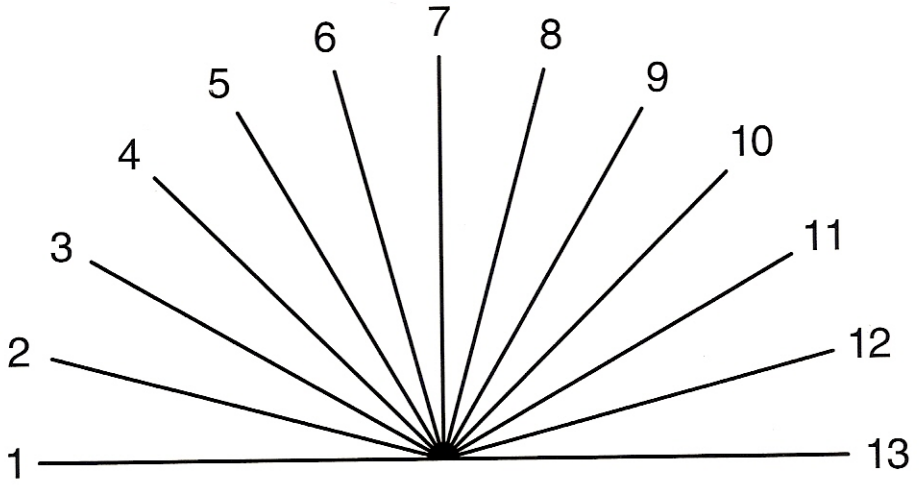


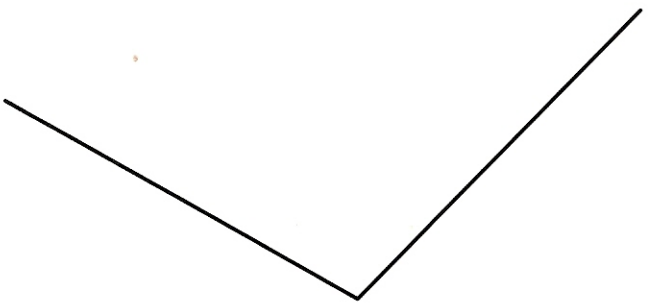

1
2
3
4
5
6
7
8
9
10
11
12
13

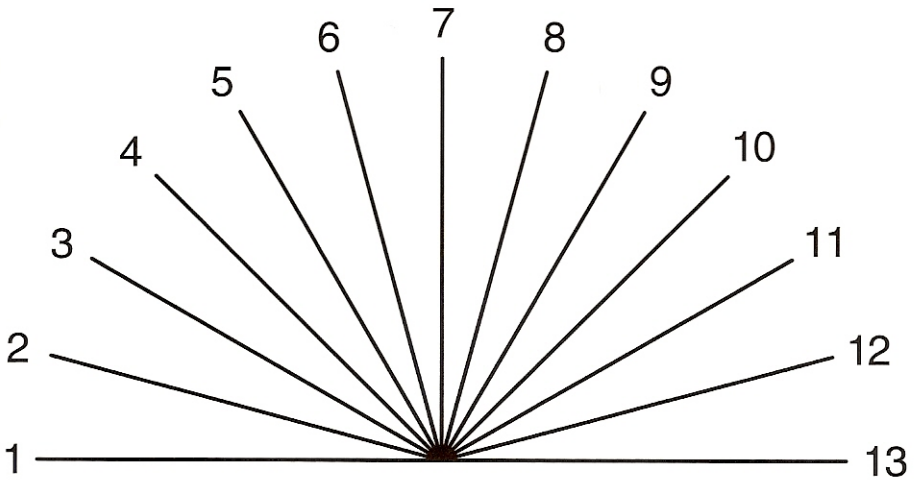


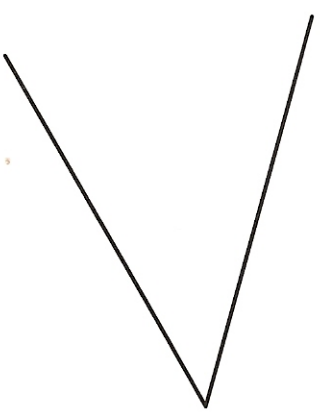

1
2
3
4
5
6
7
8
9
10
11
12
13

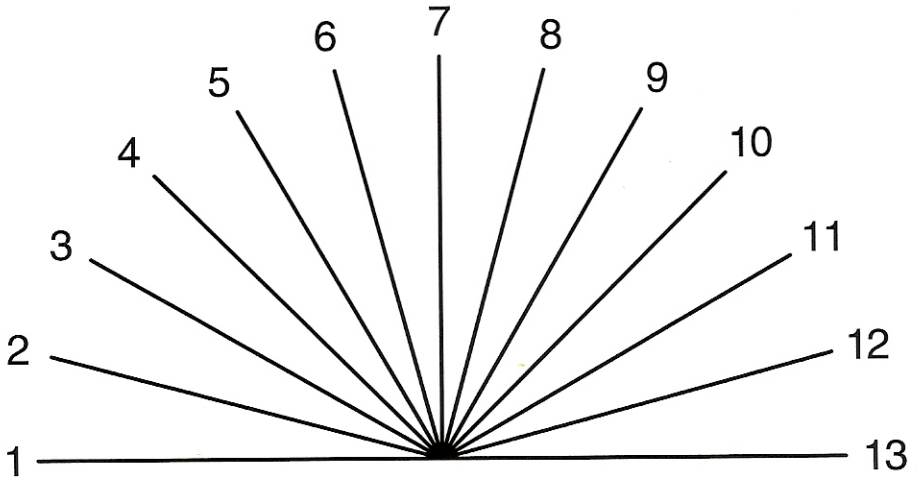


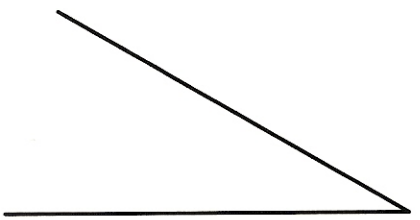

1
2
3
4
5
6
7
8
9
10
11
12
13

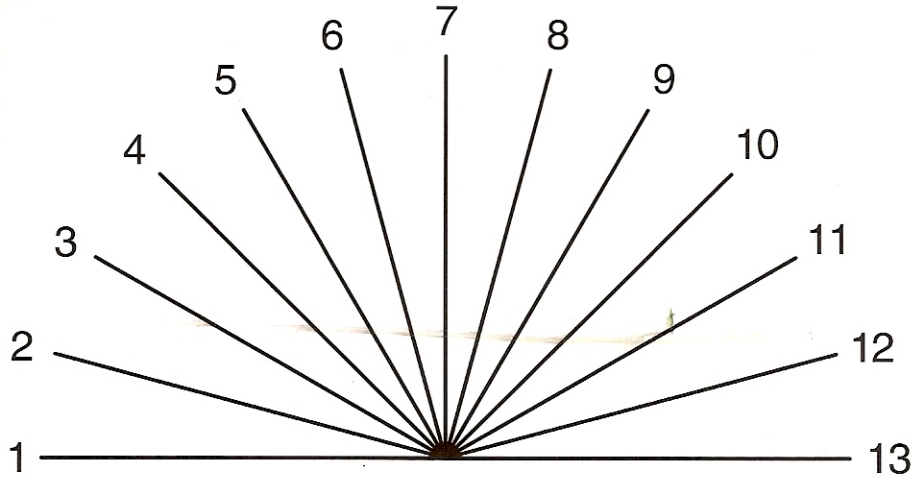


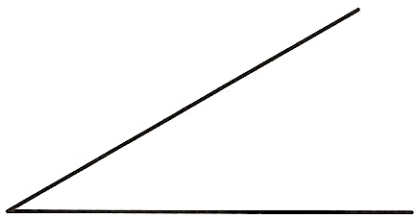

1
2
3
4
5
6
7
8
9
10
11
12
13



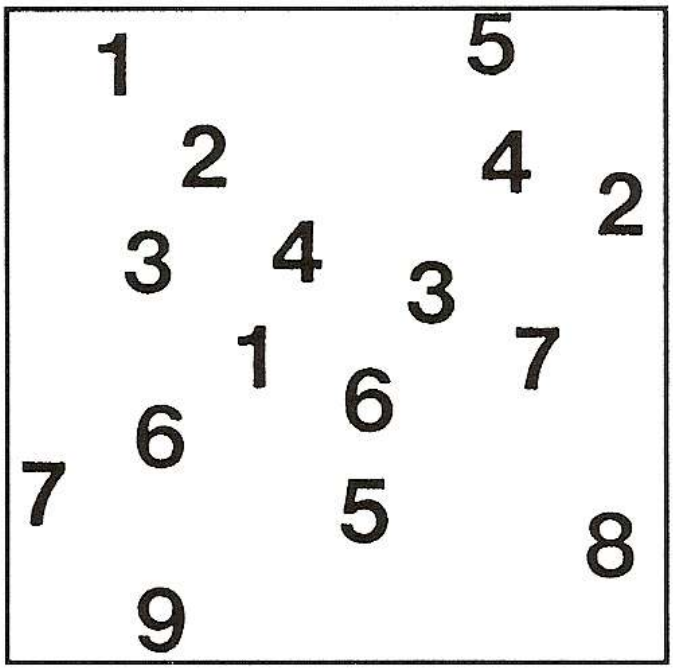

1
2
3
4
5
6
7
8
9
10
11
12
13

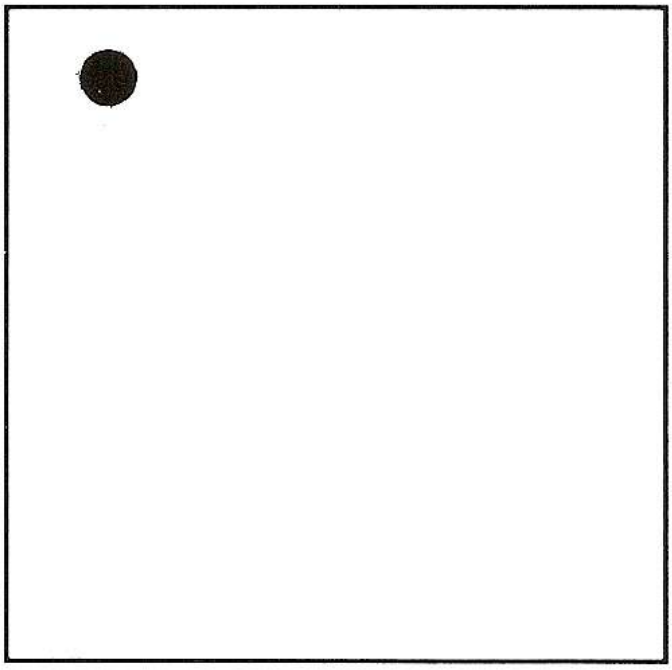


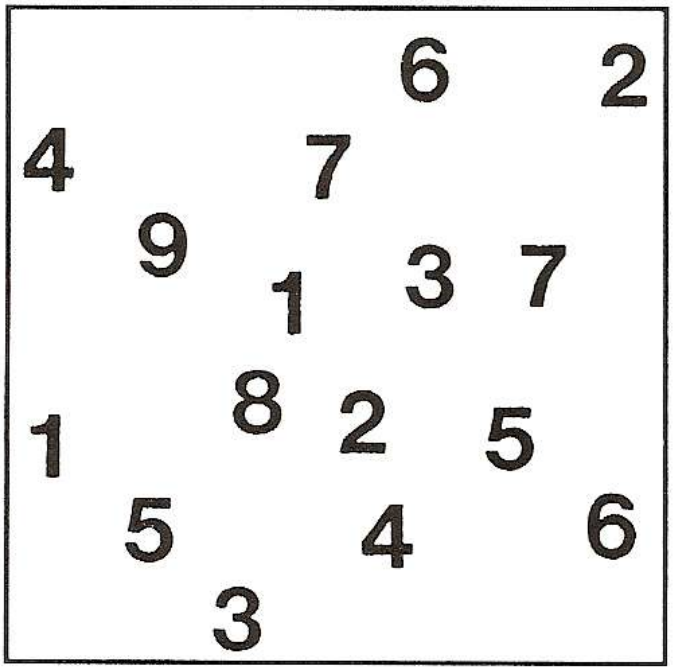

1
2
3
4
5
6
7
8
9
10
11
12
13

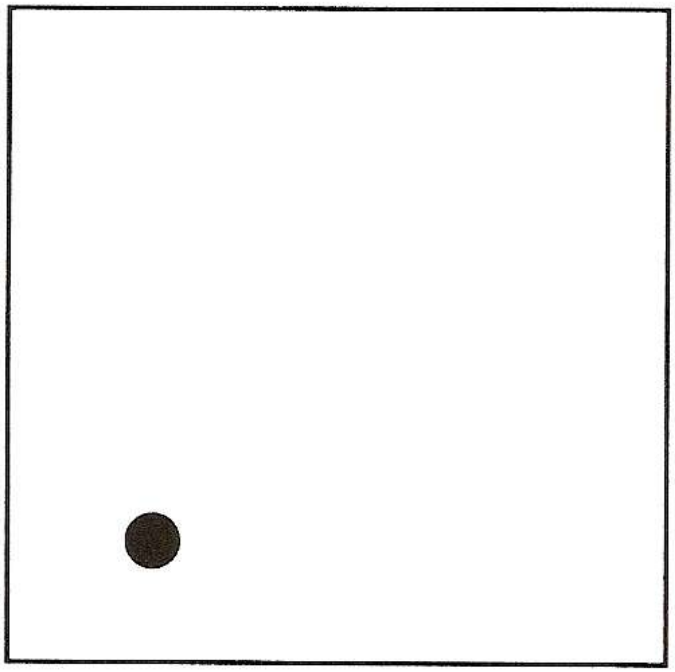


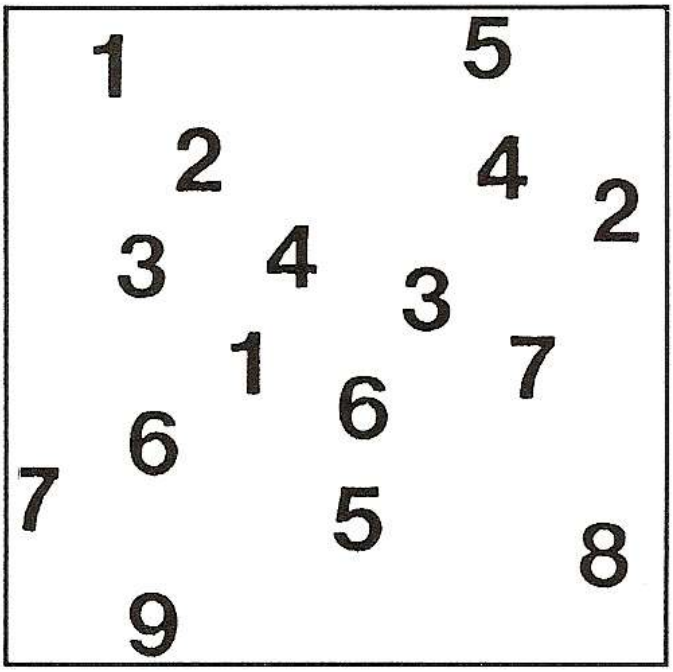
9.14.3. VOSP Battery Number Locator Test

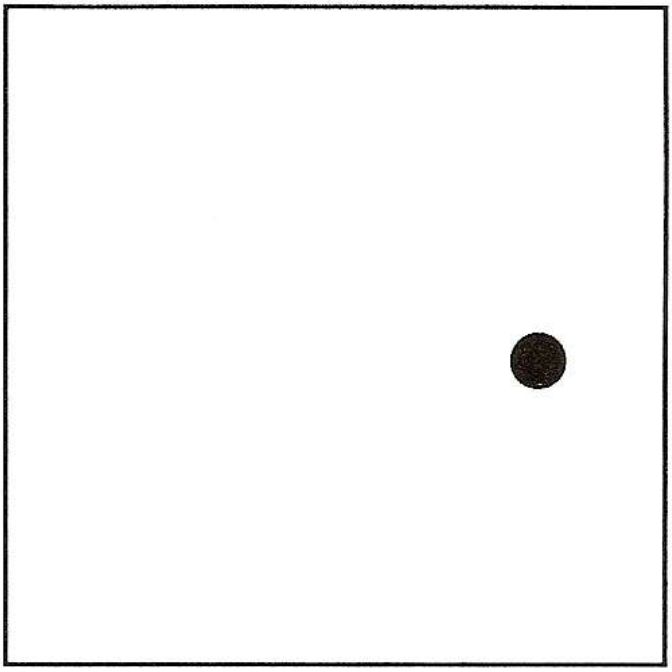

1
5
2
4
2
3
4
3
1
7
6
6
7
5
8
9

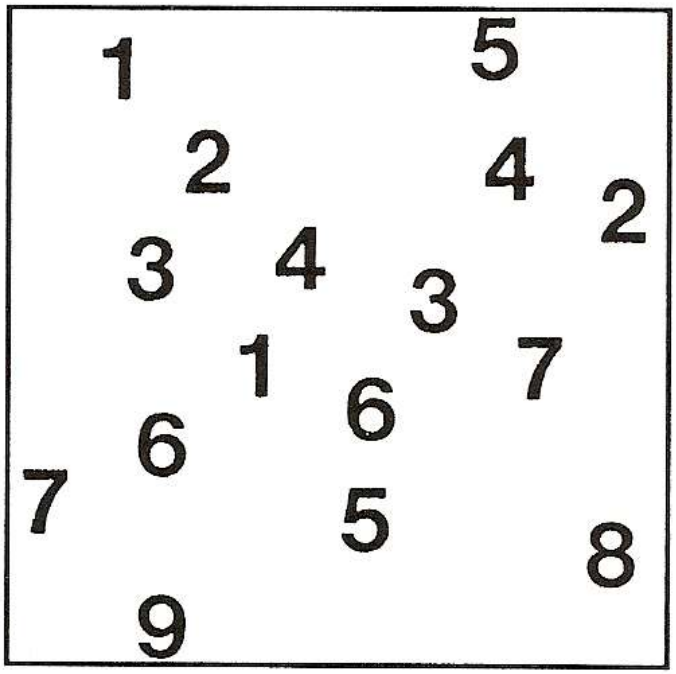


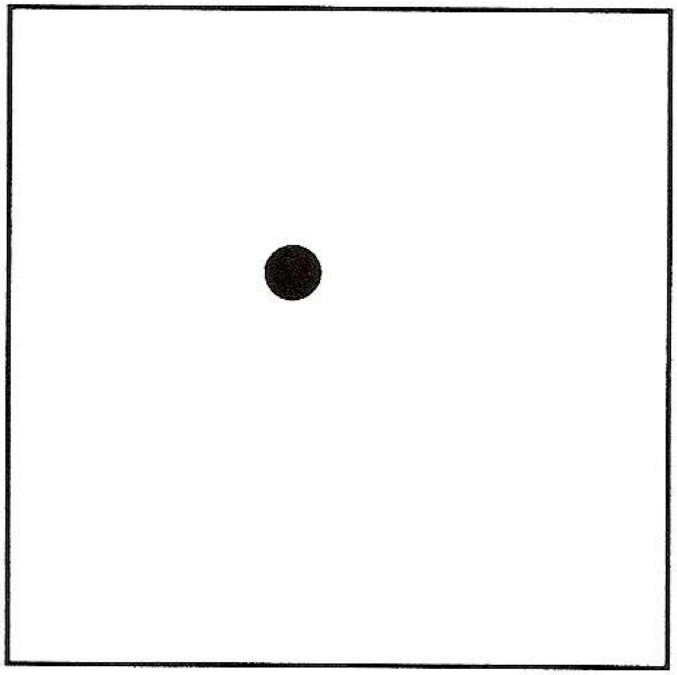

6
2
4
7
9
1
3
7
8
2
1
5
5
4
6
3

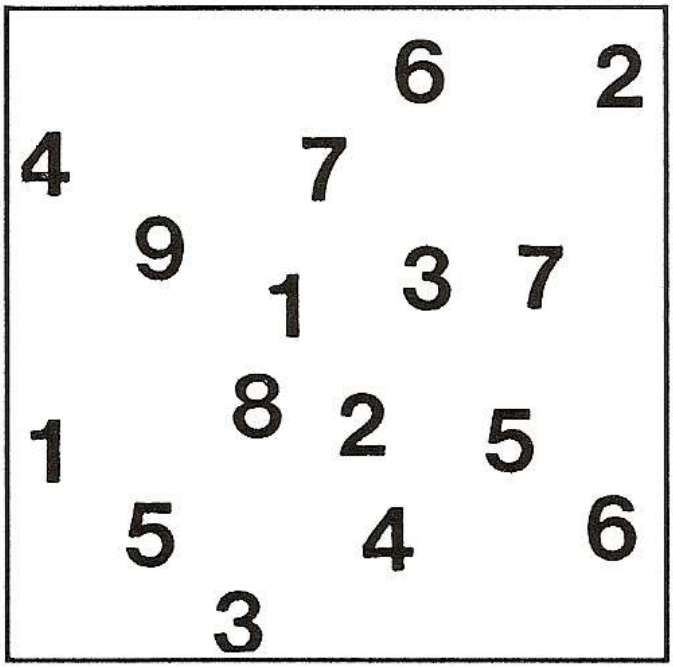


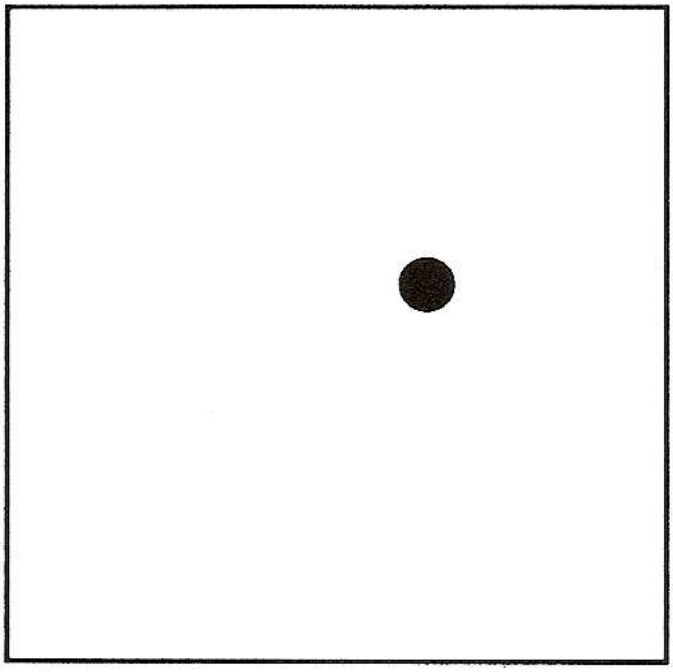

1
5
2
4
2
3
4
3
1
7
6
6
7
5
8
9

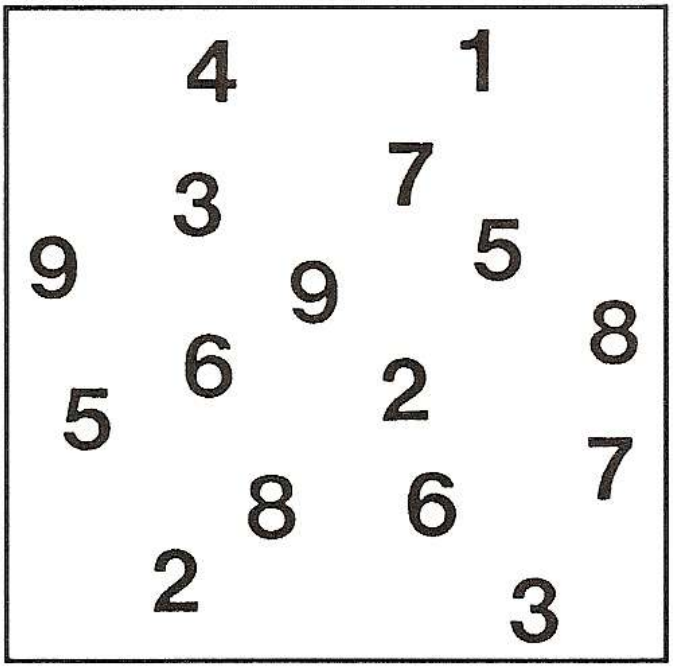


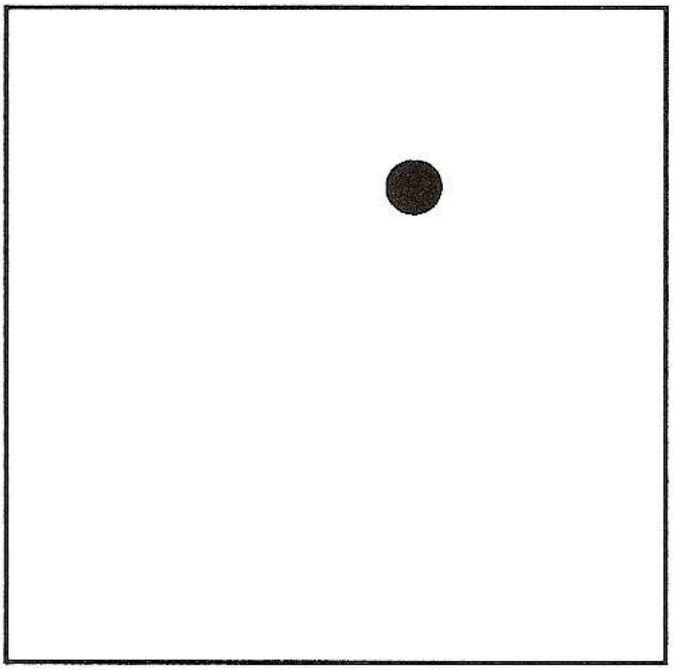

1
5
2
4
2
3
4
3
1
7
6
6
7
5
8
9

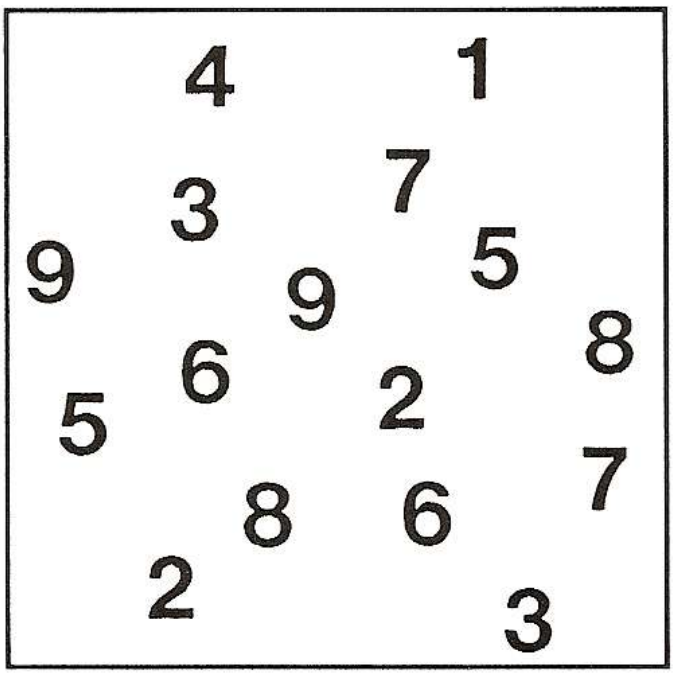


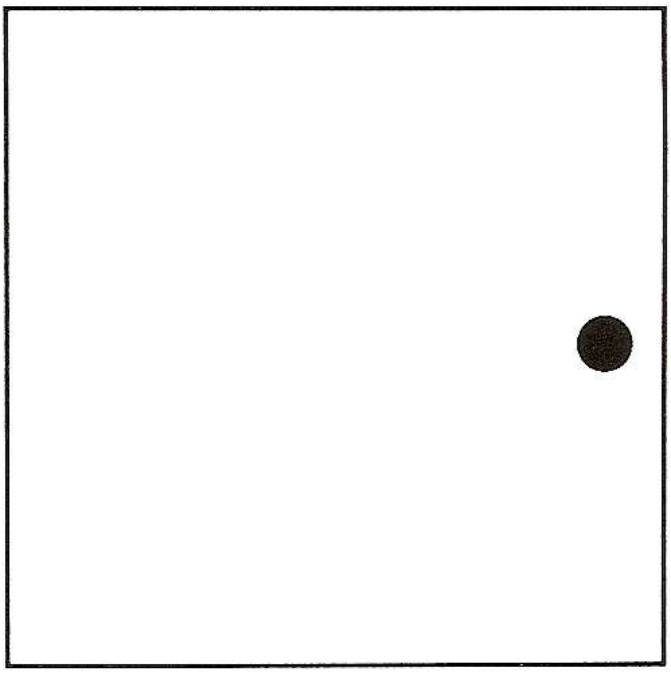

6
2
4
7
9
3
7
1
8
2
5
1
5
4
6
3

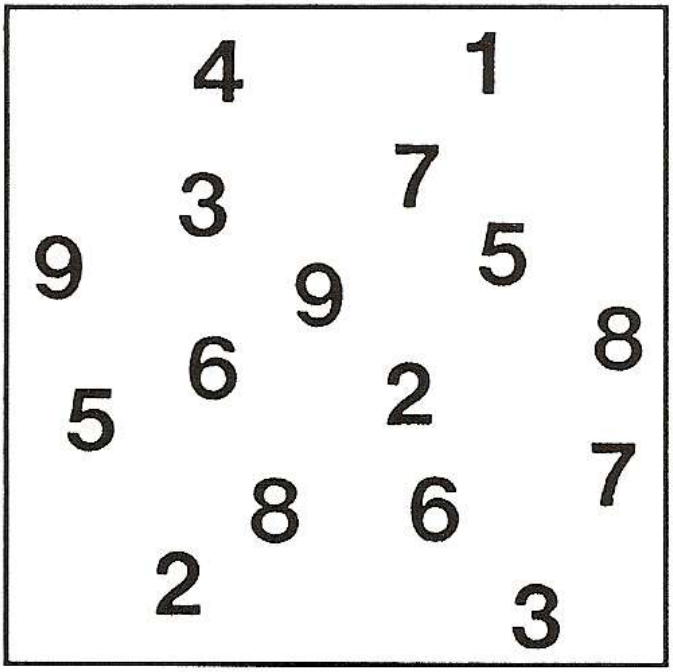


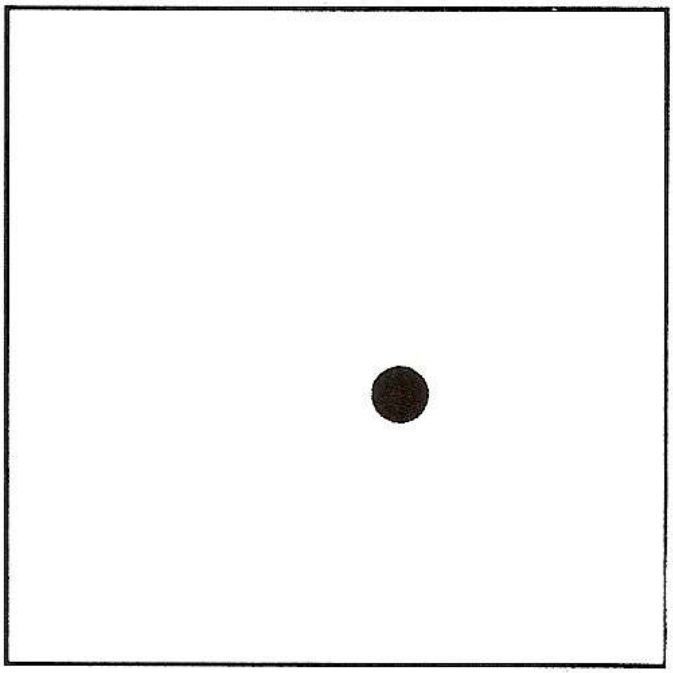

4
1
7
3
5
9
9
8
6
2
5
7
8
6
2
3

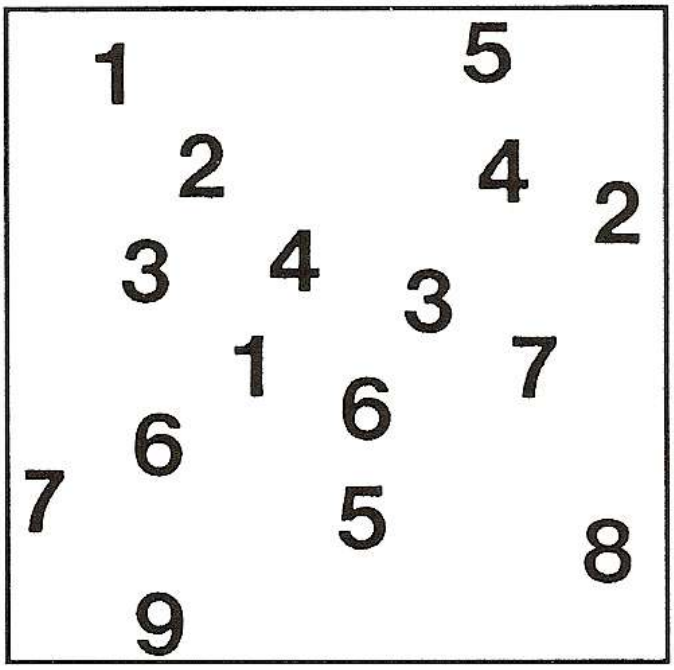


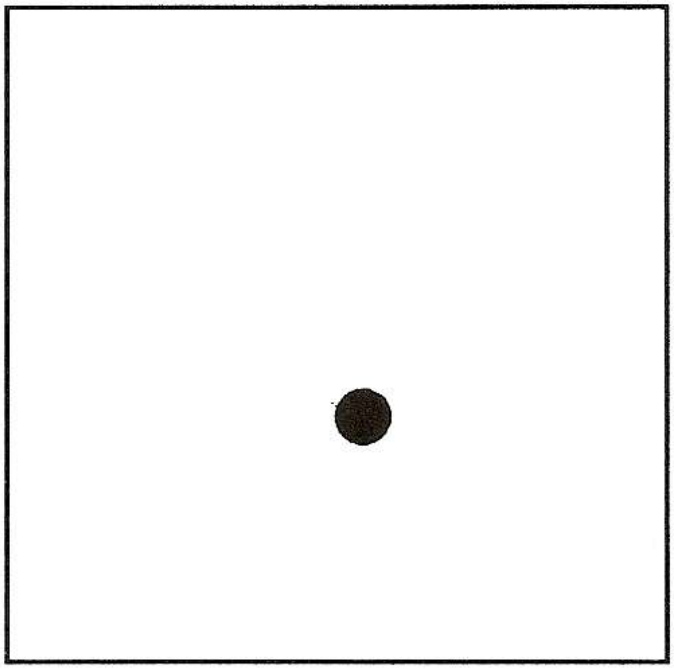

4
1
7
3
5
9
9
8
6
2
5
7
8
6
2
3

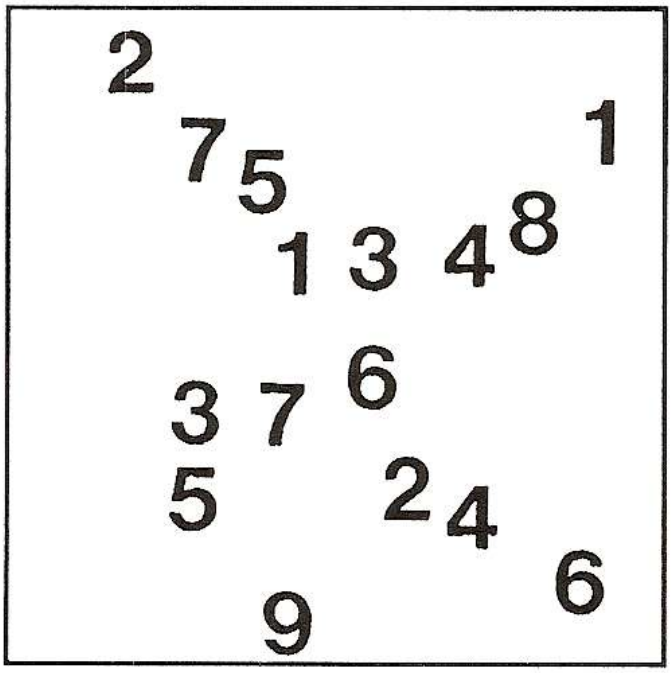


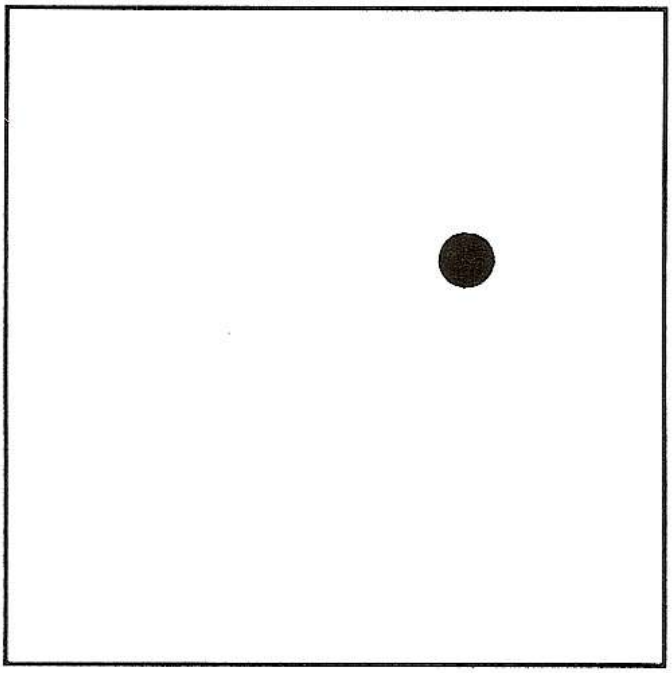

4
1
7
3
9
5
9
8
6
2
5
7
8
6
2
3

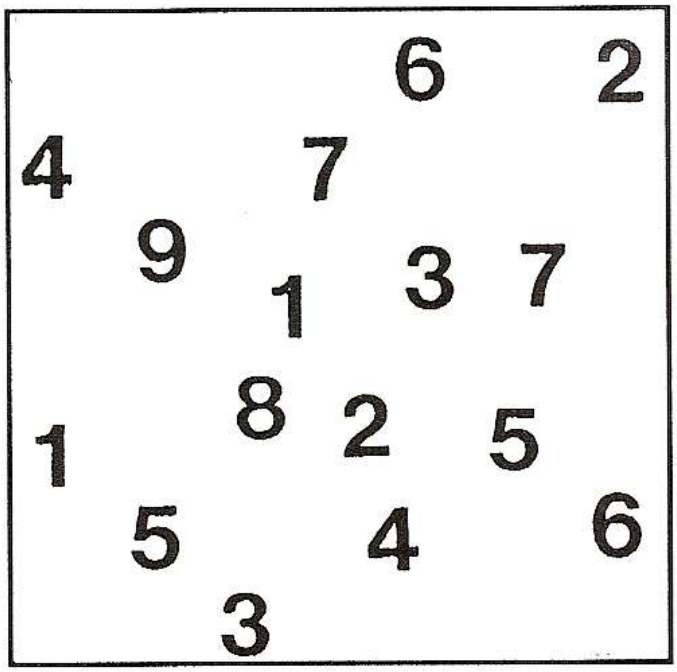


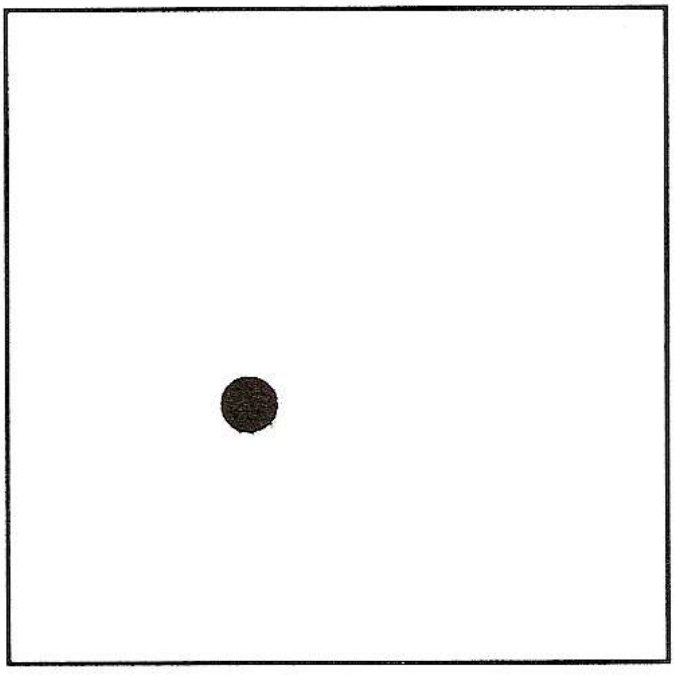

1
5
2
4
2
3
4
3
1
7
6
6
7
5
8
9

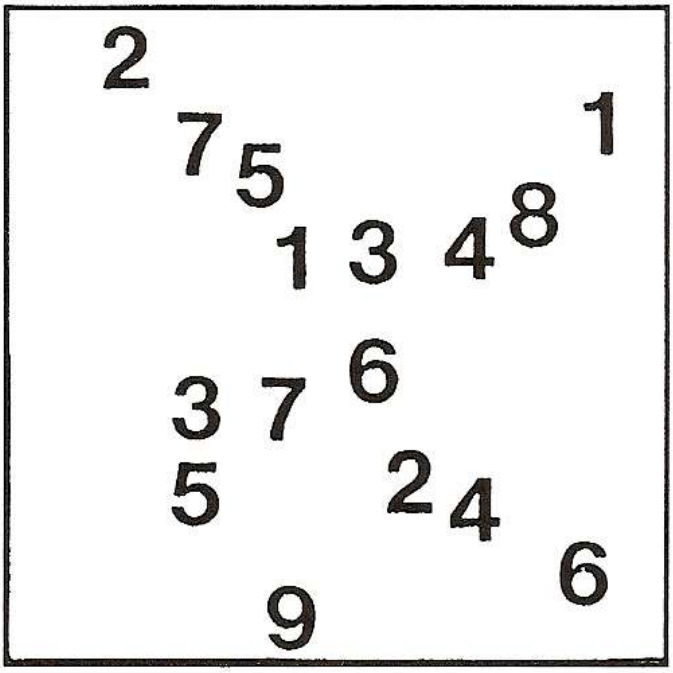



2
7
5
1
1
3
4
8
3
7
6
5
2
4
6
9




6
2
4
7
9
1
3
7
8
2
5
1
5
4
6
3




2
7
5
1
1
3
4
8
6
3
7
5
2
4
6
9