Un anno fa, la direzione di RPC aveva chiesto al collega Mario Guazzelli di inviarci un lavoro, suo e della sua scuola, da pubblicare sulla nostra Rivista. Oggi, purtroppo, Mario non è più tra noi. Pubblichiamo volentieri il prezioso contributo che i suoi allievi ci hanno inviato.
Per ricordare il collega ai nostri lettori; riandando alla dialettica, spesso simpatica, che ha caratterizzato il nostro rapporto.

The conceptual development of slight cognitive impairment as a diagnostic category: A historical perspective

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Abstract

Historically, many attempts have been made to classify the boundary state between physiological and pathological aging. After Kral (1962), who distinguished a physiological cognitive decline from a pathological one, the conceptual turnover was the classification of Mild Cognitive Impairment (MCI) by Petersen et al. in 1995. It represents the most important nosological entity currently adopted by clinicians to diagnose the preclinical phase of Alzheimer's Disease. However, clinical research on slight cognitive impairment did not provide inclusion/exclusion criteria and exact psychometric norms for such a diagnosis. In order to clarify the debate on the topic, we have given a comprehensive historical review of conceptual and nosographic aspects of all clinical entities which report a slight cognitive impairment, pointing out their neuropsychological features. As a result, we suggest a new categorization of slight cognitive impairment clinical entities, and we discuss some aspects of MCI evaluation that remain still unclear, in the light of recent revision of diagnostic criteria and psychiatric classification.

Key words: mild cognitive impairment, Alzheimer's Disease, cognitive decline, memory impairment, mild neuro-cognitive disorder, memory disturbance, memory loss, questionable dementia, cognitive impairment no-dementia, subclinical cognitive impairment

Introduction

Literature presents many attempts to classify slight cognitive impairment in order to distinguish between physiological and pathological aging. Every diagnostic category of slight cognitive impairment follows a timeline (Table 1). For each of these categories, scientific studies have produced supplementary diagnostic framing and/or new clinical entities over time.

Historical antecedents

Descriptions of cognitive impairment can be traced back to the last centuries. The first author who explained the loss of recent memory was James C. Prichard, who wrote *Treatise on Insanity and other Disorders affecting the Mind* (1837). He distinguished intellectual insanities from moral ones. He called *incoherence* or *dementia* a form of intellectual insanity that progresses slowly and gradually, where the first step was

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forgetfulness of recent impressions, while the memory retains a comparatively firm hold of ideas laid up in its recesses from times long past: the power of reasoning within the sphere of distinct recollection is not remarkably impaired, and the faculty of judgement is exercised in a sound manner when the attention can be sufficiently roused (p. 89).

Diseases of Memory (Ribot, 1887) proposed a preliminary classification of amnesia, pointing out those of progressive nature, thought to be a particular decline able to develop dementia. Ribot explicated the *law of regression or of dissolution* to clarify the gradual loss of memory, by establishing that recent memories are more likely to be lost than more remote ones:

we thus see that the progressive destruction of memory follows a logical order –a law. It advances progressively from the unstable to stable. It begins with the most recent recollections, which, lightly impressed upon the nervous elements, rarely repeated and consequently having no permanent associations, represent organization in its feeblest form. It ends with sensorial, instinctive memory, which, become a permanent and integral part of the organism, represents organization in its most highly developed stage (pp. 121-122).

The idea that deterioration of psychological functions through mental disorders or neurological impairments retraces in reverse the order of evolutionary development, was drawn on John H. Jackson's theory: recently acquired information will be lost before deeply stored older information (Taylor, 1931).

Pseudodementia

The term *pseudodementia* was used to describe a syndrome in which some psychiatric disorders could cause cognitive impairment. The impairment is not progressive and potentially reversible if the primary cause, for instance depression, is treated (e.g. with antidepressants). However, no neuropathological process could be identified or considered sufficient to explain the cognitive deficits. In their study conducted on a sample of 300 psychiatric patients, Madden et al. (1952) had noted that some symptoms ordinarily considered to indicate dementia, could be reversed with appropriate therapies. It was not until ten years later that Kiloh described a clinical state in which, initially, dementia may be very closely mimicked and associated with malingering, reactive or endogenous depression. He considered remote psychiatric disturbances, an abrupt psychiatric illness and the results of antidepressant therapies, as risk factors for pseudodementia (Kiloh, 1961). In 1965, Post discussed pseudodementia in the elderly but he did not suggest his association with psychiatric syndromes and in 1967 Lipowski documented a case of pseudodementia in a young highly educated woman with hysterical and depressive symptoms and amnestic syndrome. In 1978, Folstein and coll. hypothesized that pseudodementia develops when the neurobiological disturbances of individuals with affective disorders are superimposed on a compromised brain in aged people. They suggested that the cognitive impairment associated with depressive illness might be accurately described as the «demential syndrome of depression» (p. 93). One year later, Wells described 11 patients with a variety of psychiatric disorders, including depression (with or without personality disturbances), conversion reactions, posttraumatic neurosis and shizoaffective symptoms. He defined the syndrome as one in which «dementia is mimicked or caricatured by a functional psychiatric illness» (Wells, 1979, p. 163). He noted that pseudodementia could be associated with a variety of primary psychiatric illnesses, symptoms of short duration suffered before a request for help, rapid progression of symptoms, complaints of cognitive deficits, behavioural and cognitive performance inconsistent with the apparent degree of cognitive dysfunction, incapacity to reply to questions, and variability of performances. In 1981, Caine proposed the following diagnostic criteria that are currently used by clinicians to diagnose pseudodementia: 1) cognitive impairment as primary mental disorder; 2) superimposed symptoms or similar symptoms to primary organic disorder; 3) reversible disorder; 4) absence of organic pathology. An impairment of attention, processing information speed, spontaneous elaboration and analysis of details usually represent the main neuropsychological features of patients' cognition. Two years later, McAllister noted that pseudodementia could be associated with cerebral dysfunction or coexisted with a cerebral dysfunction. Cognitive impairment was usually a caricature

of dementia with exaggerated memory complaints and it was primarily observed in patients with personality disorders. By contrast, it was entirely indistinguishable from diffuse organic cerebral dysfunctions in other patients (McAllister, 1983). A few years later, he formulated specific diagnostic criteria for the diagnosis of pseudodementia:

> the most common features of cognitive impairment due to pseudodementia are a relatively acute onset, [with symptoms lasting from six to twelve months]; past psychiatric history, particularly depressive illness; age over 50; frequent *don't know* as opposed to *near miss* answers; normal electroencephalogram and computed tomographic scan of the brain, and absence of nocturnal worsening (McAllister, 1985, p. 175).

The persistence of cognitive symptoms after pharmacological treatment was related to a real organic pathology by Rabins, who in 1984 distinguished three subtypes of depressive pseudodementia: 1) depressed patients with subjective cognitive impairment (Type I); depressed patients with objective cognitive impairment (a Mini Mental State Examination score of <24) (Type II); patients with dementia complicated by a depressive disorder (Type III). As reported firstly by Reifler (1982) and secondly by Nussbaum (1994), the concept of *pseudodementia* has remained a permanent nosological entity in literature for over 100 years. In fact, the recognition that cognitive symptoms associated with reversible neuropsychiatric conditions can mimic or caricature irreversible disorders was known as early as the middle of 19th century. Nevertheless, the controversy regarding the validity and clinical use of the term is nowadays still present. In order to overcome this problem, Bianchetti and Pezzini (2001) have recently suggested deleting the term *pseudodementia* and introducing a temporal sequentiality between affective symptoms and cognitive ones, identifying two types of disorder (Type I and Type II), on the basis of four parameters: primary disorder, onset, initial course, cognitive state, prognosis. Type I is characterized by: depression (with consequent memory modifications); sub-acute onset (weeks or months); depressive symptoms usually appear in advance; subjective memory complaints without objective memory deficit; treatment alleviates both cognitive and affective symptoms. Conversly, Type II is characterized by: dementia (with superimposed depression); gradual onset for Alzheimer's Disease (AD) and sudden onset for vascular dementia; cognitive deficits usually appear in advance; objective cognitive deficit revealed by neuropsychological testing; treatment alleviate depression symptoms without modification of cognitive symptoms.

Senile psychosis

Primary Degenerative Dementia (original and revised criteria); Dementia and Mild Cognitive Impairment diagnostic criteria; Age-Related Cognitive Decline; Mild Neuro-cognitive Disorder

In 1955, Roth described the diagnostic criteria of senile psychosis by evaluating a total of 472 patients with different mental disorders that had necessitated the institutionalization in Graylingwell Hospital (Chichester, UK). He classified the mental disorders of later life into six different categories: affective psychosis, senile psychosis, late paraphrenia, arteriosclerotic psychosis, acute confusion and other mental disorders. He suggested that affective psychosis, late paraphrenia and acute confusion were distinct from senile and arteriosclerotic psychosis, thought as the two main causes of progressive dementia in old age. Senile psychosis was defined as:

> a condition with a history of gradual and continually progressive failure in the common activities of everyday life and a clinical picture dominated by failure of memory and intellect and disorganization of personality, where these were not attributable to specific causes as infection, neoplasm, chronic intoxication or cerebrovascular disease, known to have produced cerebral infarction (p. 283).

In 1980, the American Psychiatric Association (APA) revised diagnostic criteria for senile psychosis, by introducing *Primary Degenerative Dementia* as a diagnostic entity apt to describe the degeneration of dementia in elderly people. The APA recommended limiting the diagnosis of Primary Degenerative Dementia only to cases of notable evidence of progressive and significant decline of cognitive functions and compromised social activities. In 1983, a group of researchers

coordinated by Sulkava proposed new diagnostic criteria to improve the accuracy of identifying Primary Degenerative Dementia:

> Extensive progressive failure of intellectual capacity during adult life; deterioration of memory as the first symptom followed by disorientation; failure in the common activities of everyday life and flattening of personality; psychiatric symptoms, such as paranoid and depressive features possible; special neuropsychological symptoms such as apraxia, agnosia, aphasia and lowered alertness (p. 10).

The psychiatric classification for the diagnosis of dementia formulated by the *Diagnostic and Statistical Manual of Mental Disorders 3rd Revision* (DSM-III-R) (APA, 1987) provided valuable diagnostic criteria for Mild Cognitive Impairment which was divided into three groups: 1) MCI Type I, characterized by the only impairment of short- and long-term memory; 2) MCI Type II, characterized by short- and long-term memory impairment plus at least one of the following impaired functions: abstract thinking, judgement, aphasia, apraxia, agnosia or personality change; 3) MCI Type III, characterized by the interference of cognitive deterioration described both for MCI Type I and Type II with daily life activities. Within the *Diagnostic and Statistical Manual of Mental Disorders 4th Revision* (DSM-IV) section *Additional conditions that may be a focus of clinical attention*, *Age-Related Cognitive Decline* and *Mild Neuro-cognitive Disorder* were also mentioned (APA, 1994). The Age-Related Cognitive Decline diagnosis was used when the object of clinical attention was a clear decline of cognitive functions in aging, compared to normal limits. Subjects do not remember names or dates and they have difficulty in problem solving. Such a category was considered if a specific mental disorder and a neurological condition were excluded. Conversely, the diagnosis of *Mild Neuro-cognitive Disorder* was entirely reported by DSM-IV (APA, 1994) as *Cognitive Disorder Not Otherwise Specified* without exact psychometric norms for its neuropsychological diagnosis. The disorder was characterized by:

> The presence of two (or more) of the following conditions of cognitive impairment for most of the time over a period of at least two weeks (on the basis of the subject's report or a reliable informant): 1) impairment of memory, as evidenced by decreased ability to acquire or retrieve information; 2) abnormal operative functioning (e.g. planning, organizing, sorting, inferring); 3) alteration of attention or spread of processing information; 4) impairment of perceptual-motor skills; 5) impairment of language (e.g. understanding, searching for words). The cognitive deficits cause discomfort or marked impairment in social functioning, working, or in other important areas, and represent a decline compared to previous level of operation living (code 294.9).

More recently, the Neurocognitive Disorder Work Group (Jeste et al., 2011) has proposed a reclassification of Mild Neuro-cognitive Disorder for the fifth edition of *Diagnostic and Statistical Manual of Mental Disorders* (DSM-V), indicating a specific range for patient's cognitive performance on formal testing or equivalent clinical evaluation, that is between 1 and 2 standard deviations (*SD*) below appropriate norms (i.e., between the 3rd and 16th percentile).

Senescent Forgetfulness
Benign Senescent Forgetfulness versus Malignant Senescent Forgetfulness

Even though literature reports different terms referring to mild changes in elderly cognition like *normal senility* (Bleuler, 1924), *normal senescent decline* (Dörken & Kral, 1951), and *mild senescent memory decline* (Kral, 1958), the principal attempt to define the normal tail-end of the continuum normal aging/dementia dates back to 1962, when Kral introduced the term *Benign Senescent Forgetfulness* (BSF) to describe a loss of remote memories in elderly healthy people. As neuropsychiatric consultant at the *Hebrew Old People's and Sheltering Home* in Montreal, Kral conducted two surveys (1956-1957; 1957-1961) to evaluate mental health among patients and to recommend measures to improve their conditions (for historical reviews see Heinik, 2006; 2010). In the first survey, based on DSM-I (APA, 1952) and Roth's categorization (1955), Kral classified 162 inpatients according to the following observational criteria: degree of personality preservation, judgment ability and emotional responsiveness; presence (or absence) and type of memory impairment; presence (or absence) of psychotic/neurotic symptoms. Kral adopted a meticulous

comprehensive neurological examination, a psychiatric interview focused on particular aspects of mental functioning and intelligence, memory and visuo-perceptive tests, administered by his colleague B.T. Wigdor. Based on these criteria, Kral (1962) suggested a classification of the entire population of the Home identifying two main types of senescent forgetfulness:

> The first type of senescent memory dysfunction is characterized by the inability of the subject to recall relatively unimportant data and parts of an experience, like a name, a place, a date, whereas the experience of which the forgotten data from a part can be recalled. However, the same data which are not available for recollection on one occasion may be recalled at another time [...]. The forgotten data seem to belong to the remote rather than to the recent past. Also, the subjects are aware of their shortcoming and try to compensate for it by circumlocution and may apologize for it. This type of memory dysfunction [...] seems to progress relatively slowly [...]. It is proposed to term this type of memory dysfunction benign [senescent forgetfulness] [...]. The second type of senescent memory impairment is characterized by the inability of the subject to recall events of the recent past, whereby not only relatively unimportant data and parts of an experience but the experience as such cannot be recalled [...]. The loss of recent memories leads to two important consequences: disorientation, at first in time and place and later also to disorientation as to personal data; and, secondly, because of the missing cues, to retrogressive loss of remote memories [...]. The subjects with this type of senescent memory impairment remain unaware of their defect [and frequently produce confabulations at the beginning] [...]. The second type of memory dysfunction bears the essential characteristics of the amnestic syndrome [...]. It is identical with the senile amnestic syndrome, or as it sometimes called, *senile Korsakoff*, and forms the axis syndrome of what used to be called senile dementia and now is being termed chronic brain syndrome associated with senile brain disease [...]. For these reasons the term *malignant* is suggested for the second type of senescent memory dysfunction (pp. 257-258).

As Heinik has recently suggested (2010), the construct of benign senescent forgetfulness characterized by «(a) all well-preserved aged people, (b) normal (dull) level of general intelligence as demonstrated by IQs; (c) subnormal performance on a specific memory test, (d) subnormal performance on a specific perceptual/organization test and (e) no significant signs of malignant amnestic syndrome» (p. 6), has very similar results to those of Mild Cognitive Impairment (MCI), which is widely used today (cf. Petersen et al., 1995; 1999; Petersen, 2004).

Mild cognitive impairment not amounting to dementia
Dementia and Mild Cognitive Impairment diagnostic criteria; Mild Cognitive Disorder; Mild Memory Disturbance; Memory Loss

In 1978, the category of *Mild cognitive impairment not amounting to dementia* was formulated by the World Health Organization (WHO) in the *International Classification of Disease 9th Clinical Modification* (ICD-9-CM), as a condition of mild memory disturbance associated with aging. The *10th Revision of the International Classification of Mental and Behavioural Disorders* (ICD-10) (WHO, 1990) established diagnostic criteria for *Mild Cognitive Impairment*, by selecting them from those of dementia. The diagnosis of MCI excluded the clouding of consciousness and a significant interference with the activities of daily living. MCI was also divided into three subtypes: MCI Type 1 (with memory impairment), MCI Type 2 (with decline of intellectual abilities), and MCI Type 3 (memory impairment and decline of intellectual disabilities caused impaired functioning of daily living, from mild to severe) (cf. APA, 1987). In 1993, the WHO established the diagnostic criteria for *Mild Cognitive Disorder*, a syndrome characterized by the

> A. Presence of objective evidence (from physical and neurological examinations and laboratory tests) and/or history of brain disorder, damage or dysfunction, or *a* systemic physical disorder known to be the cause of cerebral dysfunction [...]; B. Presence of a disorder in cognitive functioning for most of the time for at least two weeks, as reported by [the] subject or by a reliable informant [...] [in each of the following areas]: (1) New learning; (2) Memory (e.g. recall); (3) Concentration; (4) Thinking (e.g. slowing); (5) Language (e.g. comprehension, word finding, etc.); C. Presence of an abnormal decline in quantitative performance detectable by means of cognitive assessment (neuropsychological or mental status examination) [...] (F06.7).

Mild Cognitive Disorder was restated by the WHO in the *International Statistical Classification of Diseases and Related Health Problems 10th Revision Version for 2007*. It was defined as:

A disorder characterized by impairment of memory, learning difficulties, and reduced ability to concentrate on a task for more than brief periods. There is often a marked feeling of mental fatigue when mental tasks are attempted, and new learning is found to be subjectively difficult even when objectively successful. None of these symptoms is so severe that a diagnosis of either dementia (F00-F03) or delirium (F05.-) can be made. This diagnosis should be made only in association with a specified physical disorder, and should not be made in the presence of any of the mental or behavioural disorders classified to F10-F99. The disorder may precede, accompany, or follow a wide variety of infections and physical disorders, both cerebral and systemic, but direct evidence of cerebral involvement is not necessarily present (F06.7).

The classification of *Mild Cognitive Impairment* was formulated as a clinical entity in the *International Classification of Diseases 9th Revision Clinical Modification* (ICD-9-CM) (WHO, 2002). This included memory complaints (preferably corroborate), objective memory impairment, relatively preserved general cognition and essentially intact activities of daily living. *Mild Cognitive Impairment* was not the only diagnostic category of cognitive decline. In fact, diagnostic criteria for *Mild Memory Disturbance* and *Memory Loss* were quoted, too. Mild Memory Disturbance was classified as another specific non-psychotic mental disorder, following organic brain damage (WHO, 2002). Alternatively, Memory Loss was characterized by the organic or psychogenetic loss of the ability to recall information, retrograde and anterograde amnesia, and a temporary or permanent loss of recent memory caused by organic or psychological factors (WHO, 2002). Some years before, Bowen and coll. (1997) had studied the progression to dementia of a group of patients with *Isolated Memory Loss*, because they thought that the history of severe memory loss in patients without other cognitive impairments was poorly understood. These patients performed 2 Standard Deviations (*SD*) below the average of memory tests administered while other areas of cognition were preserved, and they did not meet DSM-III criteria for dementia. They recognized the isolated memory loss as the incipient symptom of AD and recommended following similar patients over time to verify conversion into frank dementia.

Limited Dementia or Limited Cognitive Disturbance

In 1982, Gurland and coll. formulated the criteria for the diagnosis of dementia, by adopting the Geriatric Mental State Examination (Copeland et al., 1976) and the Comprehensive Assessment and Referral Evaluation (Gurland et al., 1977), a basically semi-structured interview guide with specific criteria for diagnosis and severity of dementia. *Limited Dementia* or *Limited Cognitive Disturbance* represents the first level of dementia severity, characterized by

a subjective report of memory decline; increased reliance on notes as reminders; occasional (less than once a week) forgetfulness of name, appointments and misplacing of objects; occasional (less than once a month) destructive or dangerous memory lapses (e.g. burning cooking or leaving on gas tap); one or two errors on cognitive testing (e.g. the subject forgets current or past president, exact date, phone number, zip code, dates of marriage or moving to present location) (Gurland et al., 1977, p. 184).

Questionable Dementia

In the same year, two other independent tests for the evaluation of dementia were published. The first one was the Clinical Dementia Rating (CDR) of Hughes et al. (1982) that identified five stages of cognitive deterioration, including the 0.5 stage, termed *Questionable Dementia*. According to Morris et al. (1993), it depicted specific deficits for each of the following domains: (A) Memory, (B) Orientation; (C) Judgement and Problem Solving; (D) Community Affairs; (E) Home and Hobbies; (F) Personal Care:

(A) Consistent slight forgetfulness; partial recollection of events; benign forgetfulness; (B) Fully oriented [patient] except for slight difficulty with time relationships; (C) Slight impairment in solving problems, similarities, and differences; (D) Slight impairment in these activities; (E) Life at home, hobbies, and intellectual interests slightly impaired; (F) Fully capable of self-care (p. 2413).

Mild Cognitive Decline

The second test published in 1982 was the Global Deterioration Scale (GDS) created by Reisberg and coll. for the evaluation of Primary Degenerative Dementia (cf. APA, 1980). It consisted of seven stages of cognitive impairment, where stage 0 corresponded to no cognitive decline and stage 7 corresponded to severe cognitive decline (for a historical review see Reisberg et al., 2008). GDS stage 3 termed *Mild Cognitive Decline*, was adopted to describe patients with specific clinical characteristics, such as

> Concentration deficit [that] may be evident on clinical testing. Also, the [patients] may demonstrate decreased facility in remembering the names of people [they have just] been introduced to. The *patients* at this stage may read a passage in a book and retain relatively little material. Decreased performance becomes manifest in demanding employment and social situations: co-workers become aware of the patient's relatively poor performance, difficulties in finding words and names may become evident to intimates, the [patients] may lose or misplace an object of value. Frequently, for the first time, the [patients] may get seriously lost when traveling to an unfamiliar location. The subtlety of the clinical symptoms may be increased by the denial that often begins to become manifest in these patients. Mild to moderate anxiety also accompanies the symptoms (Reisberg et al., 1982, p. 1137).

Age-associated Memory Impairment
Age-associated Memory Impairment (revised criteria); Age-consistent Memory Impairment; Late-Life Forgetfulness

In 1986, the National Institute of Mental Health (NIMH) Work Group proposed specific diagnostic criteria to describe memory loss in elderly healthy people with three main goals: firstly, to reach an agreement on memory decline research in aging, secondly, to investigate the neurobiological basis of memory decline and thirdly, to develop effective pharmacological and non-pharmacological treatments. For this purpose, Crook and coll. established diagnostic criteria for *Age-associated Memory Impairment* (AAMI):

> Complaints of memory loss reflected in everyday problems [such] as difficulty [in] remembering names or individuals following introduction, misplacing objects, difficulty remembering multiple items to be purchased or multiple tasks to be performed, problems remembering telephone numbers or zip codes, and difficulties recalling information quickly or following distraction. Onset of memory loss must be described as gradual, without sudden worsening in recent months (p. 270).

In 1989, Blackford and La Rue revised the NIHM diagnostic criteria for AAMI and distinguished it from *Age-consistent Memory Impairment* (ACMI), and *Late-Life forgetfulness* (LLF), according to specific psychometric concomitants (for a comparison see Table 2).

Minimal Dementia

The term Minimal Dementia was used in the Cambridge Mental Disorders in the Elderly Examination (CAMDEX) created by Roth and coll. in 1986. It was a structured psychiatric interview intended for use in studies of prevalence and incidence of dementia, especially mild. Patients were graded for dementia severity (minimal, mild, moderate and severe). Minimal Dementia was defined as «a limited and variable impairment of recall, minor and variable errors in orientation, a blunted capacity to follow arguments and solve problems and occasional errors in everyday tasks» (p.704).

Mild Cognitive Impairment
Mild Cognitive Impairment (previous and original criteria); Mild Cognitive Impairment (subtypes); Mild Cognitive Impairment with subcortical vascular features

In 1991, the term Mild Cognitive Impairment was introduced into literature by Fliker and coll. to describe patients obtaining a score of 3 on the Global Deterioration Scale, who were not demented and who exhibited at least two of the following symptoms:

(1) getting lost when travelling to an unfamiliar location; (2) decline in work performance apparent to co-workers; (3) word- and name- finding deficit apparent to intimates; (4) relatively little retention of material read in a passage of a book; (5) decreased facility remembering the names of newly introduced people; (6) losing or misplacing an object of value or (7) a concentration deficit apparent upon clinical testing (p. 41).

The diagnosis of Mild Cognitive Impairment was also formulated on the basis of performance resulting in the neuropsychological battery of the Structured Interview for the Diagnosis of Dementia of Alzheimer's Type, multi-infarct dementia, and dementias of other aetiology according to DSM-III and ICD-10 (SIDAM) (Zaudig, 1992). It was a short diagnostic screening instrument to service each item of DSM-III and ICD-10 criteria for dementia. All items of the SIDAM could be summed up as resulting in the SIDAM score (SISCO), which ranged from 0 (the worst cognitive impairment) to 55 (no cognitive impairment). The psychometric norms for the diagnosis of Mild Cognitive Impairment were a SISCO score of 34-47 and a Mini Mental State Examination score of 23-27. In 1995, Petersen and coll. created the primary diagnostic criteria for Mild Cognitive Impairment but only in 1999 researchers came to a better description of MCI diagnostic criteria: «(a) complaint of defective memory; (b) normal activities of daily living; (c) normal general cognitive function; (d) abnormal memory function for age and (e) absence of dementia» (p. 67). They studied a sample of 75 subjects during a period of five years who reported memory decline from 1.5-2.0 SD below the mean of individuals with similar age and education level. These patients were less able than healthy controls to benefit maximally from the use of semantic cues during a recall task; they also showed an impairment of delayed recall without a general cognitive function deficit. The first major study focusing on the clinical characterization and outcome of MCI was published in 1999 by Petersen and coll. to identify individuals at high risk for severe cognitive decline and progression to dementia. They recruited a sample of 76 subjects with MCI, 243 healthy normal controls and 106 patients with mild AD. MCI subjects showed a poorer performance than healthy controls in memory tests but the results were similar if compared to very mild AD patients. However, these patients were more impaired in other cognitive domains than MCI subjects. In conclusion, Petersen and coll. formulated the original criteria to diagnose MCI:

memory complaint, preferably corroborated by an informant; memory impairment documented according to appropriate reference values; essentially normal performance in non-memory cognitive domains; generally preserved activity of daily living; non dementia (Petersen et al., 1999, p. 304).

In 2001, given the great interest in this clinical category, the American Academy of Neurology (AAN) included MCI as a clinic parameter on early detection of dementia (Knopman et al., 2001). In 2004, based on the belief that memory symptoms were not unique in different MCI clinical presentations, an international conference convened in Stockholm and expanded MCI diagnostic criteria, pointing out two MCI subtypes (Winbald et al., 2004; Petersen, 2004): Amnestic MCI and Non-Amnestic MCI. In turn, both aMCI and naMCI were divided into two subgroups: Single and Multiple Domain. Amnestic MCI Single Domain, was characterized by the isolated memory impairment while Amnestic MCI Multiple Domain was characterized by the impairment of multiple cognitive domains, including memory. Non-Amnestic MCI Single Domain was characterized by a specific cognitive domain impairment, other than memory, while Non-Amnestic MCI Multiple Domain was characterized by the impairment of multiple cognitive domains, without associated memory deficits. From Petersen's reclassification of MCI, research has focused on amnestic subtype, which is believed to be a high risk condition for developing AD, or a prodromal state of this condition (Dubois et al., 2004). Patients with other dementing typologies might pass through similar pre-clinical stages. A group of patients with pre-dementia stage of Subcortical Vascular Dementia (SVD), known as *subcortical vascular MCI* (svMCI) was described by Frisoni and coll. in 2002. These patients present an executive dysfunction (mainly characterized by the impairment in goal formulation, planning, organizing, sequencing, executing, set-shifting, self-maintenance, and abstracting), alongside a mild memory deficit (impaired recall, relative intact recognition, less severe forgetting, benefit from cues) not interfering with occupational and social activities.

Age-associated Cognitive Decline

In 1994, a task force of the International Psychogeriatric Association (IPA) in collaboration with the WHO proposed diagnostic criteria for *Aging-associated Cognitive Decline* (AACD) (Levy, 1994), a clinical condition characterized by the lack of evidence of cerebral or systemic diseases or other conditions known to cause cerebral dysfunction (cf. Mild Cognitive Disorder, WHO, 1993), absence of dementia, and difficulties in any one of the following areas: memory, language, attention, concentration, thinking, and visuo-spatial functioning. Levy and coll. did not include an age restriction in order to consider a cognitive decline occurring in the last decades of adulthood. An individual or reliable informant report confirms the cognitive functioning decline which must be described as gradual and be present for at least 6 months. Subjects with AACD are required to score 1 *SD* below age and education norms in neuropsychological tests or mental state evaluation.

Cognitive Impairment no-Dementia

In 1995, Elby and coll. identified a significant number of elderly people, recruited for the Canadian Study of Health and Aging, whose problems of memory and/or relative to another cognitive domain were not sufficient to meet DSM-III-R criteria for dementia (APA, 1987). They were categorized as Cognitively Impaired Not Demented (CIND), a wide category encompassing different clinical conditions, such as Age-associated Memory Impairment (Crook et al., 1986), Age-related Cognitive Decline (APA, 1994), Age-consistent Memory Impairment (Blackford & La Rue, 1989), Late-Life Forgetfulness (Blackford & La Rue, 1989), cerebrovascular disease, depression, general vascular disease, psychiatric disorders and other non-specific conditions. In 1997, Graham et al. reported that the most common impairment of people with CIND was represented by a circumscribed memory deficit and a low score (24/30) in the Mini Mental State Examination (Folstein et al., 1975).

Subclinical Cognitive Impairment

Research has recently focused on the stage that should precede Mild Cognitive Impairment, named *Subclinical Cognitive Impairment* (SCI) (Ritchie et al., 1996). The importance of identifying SCI lies in the potential role that it can play in dementia prevention (especially AD). SCI refers to subjective memory complaints of aged people and it can be associated with other conditions, especially depression and physical illness (Reisberg & Gauthier, 2008). In 1993, on the basis of the Eugeria Study, Ritchie et al. tested 397 individuals with SCI, thought to be a heterogeneous condition with an elevated risk for dementia (with an estimated 18% incidence rate of conversion over three years). Only 13% of the sample with minimum cognitive impairment appeared to have a totally benign and transient syndrome. In 2000, Ritchie et al. investigated SCI to determine if such a nosographic label could represent normal aging, early senile dementia or a separate clinical entity. Over the 1-year period, 48% of the sample had some degree of observable cognitive deterioration on attention, language performance and working memory. Over the 3-year period, 18% of the sample developed a form of senile dementia. It appeared that SCI was a highly heterogeneous group for which previous classification such as Benign Senescent Forgetfulness (Kral, 1962) or Age-associated Memory Impairment (Crook et al., 1986), were largely inappropriate. Other criteria for SCI diagnosis were referred by Reisberg and Gauthier (2008) to the GDS Stage 2:

> This is the phase of forgetfulness. The patients complain of memory deficit. Most frequently patients in this phase complain of forgetting where familiar objects have been placed and of forgetting names that they formerly knew well. There is no objective evidence of memory deficit in the clinical interview and no objective deficit in employment or social situations. The individual in this phase displays appropriates concern about symptoms (Reisberg et al., 1982, p.182).

The hypothesis that SCI was a first step in the course from SCI->MCI->AD continuum was supported by an important longitudinal study by Prichep et al. in 2006. The researchers followed 44 SCI subjects (all GDS Stage 2 at baseline) and examined outcomes after a minimum of seven

years. The decline was defined as a cognitive change that met diagnostic criteria for MCI or dementia over the follow-up interval. They observed that 27 out of 44 individuals progressed to MCI over a period of 9 years.

Conclusions

Once the nosological and conceptual aspects of slight cognitive impairment in the context of its historic evolution have been reviewed, we conclude that despite the great number of articles describing this category, only the following clinical entities, some of which reported in ICD or DSM classifications, have specific inclusion/exclusion criteria for diagnosis: Primary Degenerative Dementia (Sulkava et al., 1983), Age-associated Memory Impairment (Crook et al., 1982; Blackford & La Rue, 1989), Age-consistent Memory Impairment and Late-Life Forgetfulness (Blackford & La Rue, 1989), Age-associated Cognitive Decline (Levy, 1994), Mild Cognitive Impairment (APA, 1990; WHO, 1993), Mild Cognitive Disorder (WHO, 1993), Mild Neuro-cognitive Disorder (APA, 1994), and Mild Cognitive Impairment with subcortical vascular features (Frisoni et al., 2002). The history of slight cognitive impairment as a diagnostic category shows that it is a heterogeneous condition representing the complex nature of cognitive decline in aging. Starting from this consideration, the clinical entities we have described in the paper should be divided into five sub-categories, according to their clinical features and prognosis (Table 2): 1) normal aging, as a benign cognitive impairment occurring in late life; 2) clinical preface of mild cognitive impairment; 3) mild cognitive decline, as a transitional state between physiological aging and early dementia; 4) dementia progression; 5) other pathological conditions related to psychiatric disorders. The various set of psychodiagnostic instruments, usually used without exact psychometric norms to analytically and specifically evaluate impaired cognitive domains, also reflect the historical development and the different theoretical perspectives that have been proposed to address the issue of *slight cognitive impairment*. In our opinion, the assessment of slight cognitive impairment should be able to pursue the following targets: 1) distinguish age-related cognitive impairment from early dementia; 2) evaluate objective memory deficits reported by elderly people with subjective memory complaints; 3) clarify if Subclinical Cognitive Impairment can be considered a stage prior to Mild Cognitive Impairment; 4) manage decision in the care of the patient, including cognitive therapies, such as Cognitive Stimulation Therapy (CST) (Spector et al., 2003) or Activation Therapy (AT) (Mondini & Bergamaschi, 2005); 5) differentiate mood and cognitive symptoms in pseudodementia. With reference to the fourth point, beyond the pharmacological treatment of dementia, many non-pharmacological interventions have been adopted by health care professionals to improve cognitive functioning of people with mild to moderate cognitive decline over time, from holistic-type techniques (i.e. Reality Orientation Therapy, Validation Therapy) to neuropsychological strategies. CST and AT currently represent effective cognitive rehabilitation strategies set up for patients suffering from MCI and mild dementia, based on neuropsychological principles (Stott & Spector, 2011). They consist of the specific stimulation of each cognitive domain in order to maximize current cognitive functioning and to reduce the risk of increasing decline. They improve patient's cognitive abilities in daily life activities, alleviating the burden of the care-giver (Cammisuli et al., 2009; 2011). With regards to the fifth point, we suggest that alongside a neuropsychological test battery apt to detect cognitive decline, clinicians should adopt psychodiagnostic instruments to evaluate depression as well as the other psychopathological symptoms that recent studies have reported in MCI patients, such as irritability, dysphoria, apathy, and anxiety (Ellison et al., 2008; Apostolova & Cummings, 2008). We do also stress that the diagnosis of Cognitive Impairment no-Dementia (CIND) in clinical practice appears, at the moment, the most adequate to diagnose different types of slight cognitive impairment, from normal aging to mild cognitive decline. In recent years, some investigators have defined a subset of persons with CIND who more closely resembled MCI subjects (Fisk et al., 2003): MCI seems to be a subtype of CIND that can include impairments of single or multiple domains. We hope that further studies will investigate more deeply the relationship between CIND and MCI to clarify whether or not MCI might represent a specific subtype of CIND. MCI has received a great deal of interest concerning the topic of a transitional state between normal aging and dementia, or more specifically AD (Petersen et al., 2009). However, the controversy over the correct definition and assessment of

MCI is still present. Petersen and O'Brien (2006) promoted the inclusion of MCI as a separate category into the DSM-V, pointing out the presence of clear criteria for identification and description, such as a distinct course of its subcategories, a specific treatment response (cholinesterase inhibitors), neurobiological abnormalities compared to normal subjects, and a genetic pattern (apolipoprotein E). Nevertheless, MCI is being included in the category of *Minor Neurocognitive Disorder* by the DSM-V Neurocognitive Disorder Work Group (Jeste et al., 2011). It represents a condition that can evolve into dementia, named *Major Neurocognitive Disorder*, with a greater degree of cognitive impairment in at least one (typically two) or more cognitive domains, such as complex attention (impairment of sustained attention, selective attention, divided attention and processing speed), executive abilities (planning, decision-making, working memory, feedback/error correction, overriding habits, mental flexibility), learning and memory (immediate memory span, recent memory, free recall, cued recall, recognition memory), language (confrontation naming, fluency, receptive language), visuoconstructional-perceptual abilities, and social cognition (emotion recognition and behavioural regulation), with a complete loss of independence in instrumental activities of daily living. Minor Neurocognitive Disorder has been opposed to Major Neurocognitive Disorder in order to recognize individuals with slight cognitive deficits in one or more of the same domains, with intact instrumental activities of daily living, even if a great effort and compensatory strategies may be required of individuals to maintain autonomy. Such an entity should include subjects currently coded in DSM-IV as *Cognitive Disorders Not Otherwise Specified* (Cognitive Disorders NOS) (i.e. MCI, CIND, ARCD). In order to make a diagnosis of Minor Neurocognitive Disorder, performance on objective cognitive assessment must be typically from 1 to 2 *SD* below the mean (or in the 2.5th to 16 th percentile) in relation to an appropriate range population (age, gender, education, premorbid intellect, culture adjusted). Such a new psychometric rule disconfirms the convention of 1-1.5 *SD* below the mean on measures of delayed recall (cf. Petersen et. al, 1999) adopted by many researchers to diagnose MCI; in clinical practice, this convention appears to be limited and also inadequate, especially for the evaluation of memory system, attention system and executive functioning. Although we are aware that the committee is in the process of redefining the diagnostic criteria for Minor and Major Neurocognitive Disorder, in our opinion, specific and sensitive neuropsychological instruments for diagnosis need to be indicated, the confusion within the category of 'Minor' remains and the problem of neuropsychological evaluation has not as yet been solved. It is thus desirable that future research will specify neuropsychological profiles of each MCI subtype and use comprehensive neuropsychological tests for exploring all cognitive domains. Finally, according to the recent criteria both for preclinical and symptomatic phases of AD by the National Institute on Aging and Alzheimer's Association (Sperling et al., 2011), clinicians should adopt a multidimensional assessment also focusing on biomarkers to support the neuropsychological diagnosis.

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CATEGORIES AND CLINICAL ENTITIES OF SLIGHT COGNITIVE IMPAIRMENT	DATE	AUTHOR/S	ASSESSMENT
Pseudodementia	**1952**	**Madden et al.**	Clinical observation
	1961	Kiloh	Clinical observation
	1979	Wells	Check-List Differentiating Pseudodementia from Dementia (Wells, 1979)
	1981	Caine	Neuropsychological Screening Test (Caine, 1981)
	1984	Rabins	Clinical observation plus Mini Mental State Examination (Folstein et al., 1975)
	1985	McAllister	Clinical observation
	2001	Bianchetti & Pezzini	Clinical observation plus neuropsychological testing (not specified)
Senile Psychosis	**1955**	**Roth**	Psychiatric Classification of Disease
Primary Degenerative Dementia	1980	American Psychiatric Association	Psychiatric Classification of Disease
Primary Degenerative Dementia (revised criteria)	1983	Sulkava et al.	Psychiatric Classification of Disease
Mild Cognitive Impairment diagnostic criteria selected from Dementia Criteria	1987	American Psychiatric Association	Psychiatric Classification of Disease
Age-Related Cognitive Decline	1994	American Psychiatric Association	Psychiatric Classification of Disease
Mild Neuro-cognitive Disorder	1994	American Psychiatric Association	Psychiatric Classification of Disease
Mild Neuro-cognitive Disorder (revised criteria)	2011	Jeste et al.	Psychiatric Classification of Disease
Benign/Malignant Senescent Forgetfulness	**1962**	**Kral**	Wechsler Memory Scale (Wechsler, 1945); Wechsler Belleview Intelligence Scale I (Wechsler, 1939); Bender Gestalt Visual Motor Test (Bender, 1938); Rorschach Test (Rorschach, 1942)
Mild Cognitive Impairment not amounting to dementia	**1978**	**World Health Organization**	Psychiatric Classification of Disease
Mild Cognitive Impairment diagnostic criteria selected from Dementia Criteria	1990	World Health Organization	Psychiatric Classification of Disease
Mild Cognitive Disorder	1993	World Health Organization	Psychiatric Classification of Disease
Isolated Memory Loss	1997	Bowen et al.	Mini Mental State Examination (Folstein et al., 1975); Mattis Dementia Rating (Mattis, 1988); Fuld Object Memory Evaluation (Fuld, 1981); Boston Naming Test (Kaplan et al., 1983); Wechsler Adult Intelligence Scale Revised (Wechsler, 1981); Trail Making Test (Reitan, 1985)
Mild Cognitive Impairment	2002	World Health Organization	Psychiatric Classification of Disease
Mild Memory Disturbance	2002	World Health Organization	Psychiatric Classification of Disease
Memory Loss	2002	World Health Organization	Psychiatric Classification of Disease
Mild Cognitive Disorder (revised criteria)	2007	World Health Organization	Psychiatric Classification of Disease
Limited Dementia or Limited Cognitive Disturbances	**1982**	**Gurland et al.**	Geriatric Mental State Examination (Copeland et al., 1976); Comprehensive Assessment and Referral Evaluation (Gurland et al., 1977)

SLIGHT COGNITIVE IMPAIRMENT SUBCATEGORIES	CLINICAL ENTITIES	NEUROPSYCHOLOGICAL FEATURES/PSYCHOMETRIC CRITERIA

Questionable Dementia	**1982**	**Hughes et al.**	Clinical Dementia Rating (Hughes et al., 1982)
Mild Cognitive Decline	**1982**	**Reisberg et al.**	Global Deterioration Scale (Reisberg et al., 1982)
Age-Associated Memory Impairment	**1986**	**Crook et al.**	Benton Visual Retention Test (Benton, 1974); Wechsler Memory Scale (Wechsler, 1975); Memory Complaints Questionnaire (Crook et al., 1992); Wechsler Adult Intelligence Scale Revised (1981); Mini Mental State Examination (Folstein et al., 1975)
Age-Associated Memory Impairment (revised criteria)	1989	Blackford & La Rue	Wechsler Memory Scale-Revised (1987); Benton Visual Retention Test (1974); Rey-Osterreith Complex Figure (1964); Visual Recognition Memory Test (Fliker et al., 1987); Guild Memory Test (Gilbert & Ferris, 1980); Randt Memory Test (Randt & Brown, 1986); Rey Auditory Verbal Learning Test (Rey, 1964); Buschke-Fuld Selective Reminding Test; Wechsler Adult Intelligence Scale-Revised (1981)
Age-Consistent Memory Impairment	1989	Blackford & La Rue	
Late-Life Forgetfulness	1989	Blackford & La Rue	
Minimal Dementia	**1986**	**Roth et al.**	Cambridge Mental Disorders in the Elderly Examination (Roth et al., 1986)
Mild Cognitive Impairment	**1991**	**Fliker et al.**	Global Deterioration Scale (Reisberg et al., 1982)
Mild Cognitive Impairment (diagnosis according to ICD-10 and DSM-III-R criteria)	1992	Zaudig	Structured Interview for the Diagnosis of Dementia of Alzheimer Type, multi-infarct dementia, and dementias of other aetiology according to DSM-III and ICD-10 (Zaudig, 1992)
Mild Cognitive Impairment (primary criteria)	1995	Petersen et al.	Free and Cued Selective Reminding Test (Buschke, 1984); Rey Auditory Verbal Learning Test (Rey, 1964); Mini Mental State Examination (Folstein et al., 1975); Wechsler Adult Intelligence Scale Revised (Wechsler, 1981); Dementia Rating Scale (Mattis, 1988)
Mild Cognitive Impairment (original criteria)	1999	Petersen et al.	Wechsler Adult Intelligence Scale Revised (Wechsler, 1981); Rey Auditory Verbal Learning Test (Rey, 1964); Wide-Range Achievement Test (Jastak & Jastak, 1978); Mini Mental State Examination (Folstein et al., 1975); Dementia Rating Scale (Mattis, 1988); Free and Cued Selective Reminding Test (Buschke, 1984); Boston Naming Test (Kaplan et al., 1983); Controlled Oral Word Association Test (Benton & Hamsher, 1978); Category Fluency Test (Monsch et al., 1984); Clinical Dementia Rating (Hughes et al., 1982); Global Deterioration Scale (Reisberg et al., 1982)
Mild Cognitive Impairment with subcortical vascular features	2002	Frisoni et al.	Category Fluency (Novelli et al., 1986); Wisconsin Card Sorting Tets (Heaton, 1981); Digit Span (Orsini et al., 1987); Memory of Prose (Spinner & Tognoni, 1987); Corsi Span (Orsini et al., 1987); Token Test (De Renzi & Vignolo, 1962); Limb Apraxia Test (De Renzi et al., 1968)
Mild Cognitive Impairment (revised criteria)	2004	Petersen	Diagnostic algorithm to differentiate between MCI subtypes
Age-Associated Cognitive Decline	**1994**	**Levy et al.**	Psychiatric Classification of Disease
Cognitive Impairment no-Dementia	**1995**	Elby et al.	Mini Mental State Examination (Folstein et al., 1975)
	1997	Graham et al.	Mini Mental State Examination (Folstein et al., 1975)
Subclinical Cognitive Impairment	**1996**	**Ritchie et al.**	Détérioration Cognitive Observée structured questionnaire (Ritchie & Fuhrer, 1994); Examen Cognitif par Ordinateur (Ritchie et. al., 1993)
	2006	Prichep et al.	Global Deterioration Scale (Reisberg et al., 1982)
	2008	Reisberg & Gauthier	Global Deterioration Scale (Reisberg et al., 1982)

Table 1. Categories of slight cognitive impairment and related clinical entities: A timeline.

NORMAL AGING	Benign Senescent Forgetfulness (Kral, 1962)	Remote memories loss that progress slowly, followed by recent memories loss
	Mild cognitive decline not amounting to dementia (WHO, 1978)	Mild memory disturbance associated with aging
	Age-Related Cognitive Decline (APA, 1994)	Slight impairment of several cognitive domains occurring in aging
	Age-Associated Memory Impairment (Crook et al., 1986)	Gradual memory loss without sudden worsening. Performance at least 1 *SD* below the means established for young adults on standardized tests of recent memory
	Age-Associated Memory Impairment (revised criteria) (Blackford & La Rue, 1989)	People between age range 50-79 years. Perceived decrease of day-to-day memory functioning verified by standardized self-report memory questionnaires. Performance at least 1 *SD* below the mean established for young adults on one or more recommended tests. Evidence of adequate intellectual function as determined by a scaled score of at least 9 on the Vocabulary subtest of the Wechsler Adult Intelligence Scale
	Age-Consistent Memory Impairment (Blackford & La Rue, 1989)	Performance within ± 1 *SD* of the mean established for age on 75% or more of verbal and non-verbal memory tests. Verbal and performance IQ scores between 90 and 130, as determined by either the Wechsler Adult Intelligence Scale and Wechsler Adult Intelligence Scale Revised.
	Late-Life forgetfulness (Backford & La Rue, 1989)	Performance between 1 and 2 *SD* below the means established for age on 50% of verbal and non-verbal memory tests. Verbal and performance IQ scores between 90 and 130, as determined by either the Wechsler Adult Intelligence Scale and Wechsler Adult Intelligence Scale Revised.
	Age-Associated Cognitive Decline (Levy et al., 1994)	Performance must be at least 1 *SD below* the mean value for appropriate population in quantitative cognitive assessment (neuropsychological test or mental state evaluation)
CLINICAL PREFACE OF MILD COGNITIVE IMPAIRMENT	Subclinical Cognitive Impairment (Prichep et al., 2006; Reisberg & Gauthier, 2008)	GDS Stage 2. The patient performs below average for his or her age and Wechsler Adult Intelligence Scale vocabulary score on 3 of the 5 Guild Memory subtests
MILD COGNITIVE DECLINE	Malignant Senescent Forgetfulness (Kral, 1962)	Loss of recent memories and conversion into dementia in the span of a few years
	Limited Dementia or Limited Cognitive Disturbances (Gurland et al., 1982)	Subjective reports of memory decline and declarative memory impairment
	Questionable Dementia (Hughes et al., 1982)	CDR 0.5
	Mild Cognitive Decline (Reisberg et al., 1982)	GDS Stage. The patient perform 1 *SD* or more below average for their age and Wechsler Adult Intelligence Scale vocabulary score on at least 3 of the 5 Guild Memory subtests. They may still make no errors on the 10-item Mental Status Questionnaire.
	Minimal Dementia (Roth, 1986)	Limited and variable impairment in memory, attention, orientation and executive functioning, revealed by CAMDEX
	Mild Cognitive Impairment (APA, 1987)	Memory Impairment (MCI Type I); impairment of memory, executive functioning, language, praxis, and gnosis (MCI Type II)
	Mild Cognitive Impairment (WHO, 1990)	Memory Impairment (MCI Type I); memory impairment and intellectual abilities decline (MCI Type II); memory impairment, intellectual abilities decline and deteriorated activities of daily living (MCY Type III)
	Mild Cognitive Impairment (Fliker et al., 1991)	Short- and long- term memory, language, visuo-spatial praxis, and executive functioning impairment. GDS Stage 3
	Mild Cognitive Impairment (Zaudig, 1992)	Diagnosis of Mild Cognitive Impairment (APA, 1987) plus a SIDAM (Structured Interview for the Diagnosis of Dementia of Alzheimer Type, multi-infarct dementia, and dementias of other aetiology according to DSM-III and ICD-10) score of 34-47 and Mini Mental Examination Score of 23-27
	Mild Cognitive Impairment (Petersen et al, 1995; 1999)	Memory decline from 1.5-2.0 *SD* compared to individual of similar age and education level. Intact general cognitive function
	Mild Cognitive Disorder (WHO, 1993)	Cognitive disturbance referred to difficulties in memory, attention, language, visuo-spatial and executive functioning. Abnormal decline detectable by neuropsychological or mental status examination
	Mild Neuro-cognitive Disorder (APA, 1994)	Cognitive impairment in memory (decreased ability to acquire or retrieve information), executive functioning (planning, organizing, sorting, and interfering), attention and information processing, perceptual-motor skills, and language (understanding, searching of words)
	Cognitive Impairment no-Dementia (Graham et al., 1997)	Circumscribed memory deficit and low Mini Mental State Examination (24/30)
	Isolated Memory Loss (Bowen et al., 1997)	Pure memory deficit without impairment of other cognitive domain. Performance below 2 *SD* of memory tests administered
	Mild Cognitive Impairment with subcortical vascular features (Frisoni et al., 2002)	Mild memory deficit (impaired recall, relative intact recognition, less severe forgetfulness, benefit form cues) and disexecutive syndrome (impairment in goal formulation, planning, organizing, set-shifting and self-maintenance, and abstracting)
DEMENTIA PROGRESSION	Primary Degenerative Dementia (Sulkava et al., 1983)	Deterioration of memory, orientation, attention, praxis, and gnosis. Failure in common activities of everyday life
OTHER PATHOLOGIES RELATED TO PSYCHIATRIC SYNDROME	Pseudodementia (Rabins, 1982)	A Mini Mental State Examination score of <24 in patients with depression
	Pseudodementia(Bianchetti & Pezzini, 2001)	Depression with consequent memory modifications *versus* dementia with superimposed depression

Table 2. Slight cognitive impairment clinical entities: A proposal for a new classification model