Review

Drawing Disorders in Alzheimer’s Disease and Other Forms of Dementia

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Abstract. Drawing is a multicomponental process that can be impaired by many kinds of brain lesions. Drawing disorders are very common in Alzheimer’s disease and other forms of dementia, and can provide clinical information for the distinction of the different dementing diseases. In our review we started from an overview of the neural and cognitive bases of drawing, and from a recollection of the drawing tasks more frequently used for assessing individuals with dementia. Then, we analyzed drawing disorders in dementia, paying special attention to those observed in Alzheimer’s disease, from the prodromal stages of the amnesic mild cognitive impairment to the stages of full-blown dementia, both in the sporadic forms with late onset in the entorhino-hippocampal structures and in those with early onset in the posterior neocortical structures. We reviewed the drawing features that could differentiate Alzheimer’s disease from vascular dementia and from the most frequent forms of degenerative dementia, namely frontotemporal dementia and Lewy body disease. Finally, we examined some peculiar aspects of drawing disorders in dementia, such as perseverations, rotations, and closing-in. We argue that a careful analysis of drawing errors helps to differentiate the different forms of dementia more than overall accuracy in drawing.

Keywords: Alzheimer’s disease, constructional apraxia, drawing disorders, frontotemporal dementia, Lewy body disease, vascular dementia

INTRODUCTION

Different kinds of drawing disorders can be found in Alzheimer’s disease (AD) and other forms of dementia. Investigations conducted in patients with focal brain lesions and with different forms of dementia have shown that many of these disorders can be traced back to visual-spatial [1–5] or planning disturbances [6–10]. These disorders are the main determinants of constructional apraxia (CA) and are subsumed by lesions affecting the parietal [11–14] and frontal regions of the brain [15]. They are usually observed in copying tasks, whereas other forms of drawing disorders, mainly due to disruption of (or impaired access to) the pictorial representations of objects, can be observed on tasks of drawing from memory [16–19]. The relationships between clinical forms of dementia and the corresponding patterns of drawing disabilities stem, therefore, from the fact that (at least in the early stages of the disease) the brain pathology affects different brain networks preferentially. Furthermore, the need for simple tasks with rich informational content in the assessment of dementia prompted the implementation of drawing...
tasks, such as the clock drawing test (CDT) [20–22], in place of those classically used for assessing CA in patients with focal brain lesions. The present review will therefore include several sections. The first will illustrate the neural and cognitive bases of drawing. The second will focus on the drawing tasks more frequently used in dementia. In the third section we will pass to analyze drawing disorders in AD, from the prodromal stages of the amnesic mild cognitive impairment (aMCI) to the stages of full-blown dementia, both in the sporadic forms with late onset in the entorhino-hippocampal structures and in those with early onset in the posterior neocortical structures. In the next section we will consider the features of drawing disorders that could differentiate AD from vascular dementia and from the most frequent forms of degenerative dementia, namely frontotemporal dementia (FTD) and the Lewy body disease (LBD). In the last section we will take into account some peculiar aspects of drawing disorders in dementia, paying particular attention to the ‘closing-in’ phenomenon [23–25].

Drawing disorders in dementia are a field that is attracting an ever-growing interest. A 2005-2015 literature search limited to Pubmed database and to papers in English language, with the terms ['drawing' OR 'copying' OR 'constructional'] AND 'dementia' as keywords, identified 793 papers. Faced with this huge amount of studies, we realized that our work could not be ‘exhaustive’ but wanted to provide a ‘comprehensive’ review including the most relevant clinical aspects of drawing disorders.

NEURAL AND COGNITIVE BASES OF DRAWING

Poppelreuter [26] observed that some brain damaged patients may be impaired in a series of activities requiring a careful control of vision on action, such as drawing, but Kleist [27] was the first to propose the term CA to designate a specific disturbance ‘which appears in formative activities (such as assembling, building, or drawing) in which the spatial form of the task is missed, although there is no apraxia of the single movements’. In subsequent years, drawing became progressively more popular in neuropsychological assessment, and a lot of studies have been aimed at comprehending its neural and cognitive bases in brain-damaged patients and in healthy subjects [22, 28].

Studies on focal brain-damaged patients have often provided contrasting evidence about the lateralization and the intra-hemispheric locus of lesions provoking drawing disabilities. Since 1962 Benton [29] underlined that the subjective nature of the clinical method, and the large variability of the tasks used to study constructive disorders could account for the inconsistencies found in previous literature. Benton thus suggested using graded tests with precise scoring procedures to improve the comparability of results [29]. The ensuing authors adopted this pivotal standpoint in trying to comprehend the neural basis of drawing through the analysis of performance in focal brain damaged patients. However, many studies did not detect differences in prevalence or severity of drawing disorders after right or left hemispheric lesions (e.g., [2, 6, 30–32]). Then, prompted by a first systematic survey about the relationships between visuoconstructive disabilities and hemispheric locus of lesion [12], several authors attempted to ascertain whether qualitative differences existed between the drawing disorders resulting from right or left hemispheric lesions. On one hand, correlational studies demonstrated that drawing disabilities are tightly related with scores on visual-perceptual tasks in right but not in left brain-damaged patients, e.g., [3, 4, 18, 33]. On the other hand, drawing disorders in left-brain damaged patients have been ascribed to a planning disorder [6, 7], but this interpretation has not been supported by several empirical studies [8, 9, 17, 18, 33]. In one of the most recent systematic attempts at identifying the neural structures involved in copying a complex figure, in a wide sample of focal brain-damaged patients, voxel-based morphometry revealed that different lesions in the two hemispheres were significantly correlated with different aspects of the drawing production [34].

Rather inconclusive results on the neural bases of drawing in healthy individuals have also been obtained by functional neuroimaging studies, because the latter have been constrained by the relevant artifacts induced by the hand and arm movements, and have often assessed drawing-related tasks rather than actual drawing [14, 35–38]. Only very recently the development of functional MRI-compatible graphic tablets [39] allowed the analysis of hand movements during drawing, and one recent study using such a device reported activation within a wide network extending from the temporo-occipital to parietal and lateral frontal areas bilaterally in healthy participants while they were drawing faces or abstract patterns with different levels of visual details [40].
Neuropsychological and functional neuroimaging data are thus consistent with the view that the process of drawing involves different cognitive components. Several authors tried to single out these components within comprehensive models [41–43]. All these models share the idea that visuospatial processes, dedicated planning abilities, and general control processes are involved in drawing [44], although the models differ from each other in terms of formal characteristics, depth of analysis and some theoretical aspects. It is worth mentioning that Grossi [43], drawing on the distinction between lexical and sub-lexical components of language, proposed the existence of two copying procedures: a “lexical” route and a “line-by-line” procedure. The former would consist of motor subroutines, which could be considered as part of a “constructional lexicon”, could be used to draw well-known figures (such as a square or a face), and would develop as a result of formal education and personal aptitudes [45–46]. The latter procedure would not rely on previously acquired constructional representations, could be used for copying novel stimuli, and would be only based on a piecemeal spatial analysis. Both procedures could be adopted for copying complex pictures, but some patients might be constrained to use either one or the other. For instance, patients with visual agnosia tend to adopt a slavish “line-by-line” copying procedure for familiar objects they cannot recognize [47–48]. On the contrary some demented patients can draw simple figures successfully but fail at integrating correctly shaped simple elements in a coherent whole, because of planning or visuospatial defects.

**DRAWING TASKS FREQUENTLY USED IN DEMENTIA**

Drawing tasks have been widely used in patients with dementia, but drawing performance may vary greatly, as a function of the task (copying and free drawing cannot be considered equivalent), of the stimuli used in the task (more complex stimuli pose greater load on visuospatial and planning functions), and even of the patients’ pre-morbid abilities (largely dependent on age, educational level, and even cultural background) [49].

Free drawing, in which the patient is required to draw a named object (e.g., a clock, a face, an animal, or a tool), can reveal information about the patients’ ability to draw complete shapes or a tendency to omit parts and about their ability to organize the figure as a whole, with its component elements in the correct spatial relationships. However, this task also relies on non-constructural cognitive abilities, particularly on lexical-semantic knowledge, pictorial representations, and mental imagery [16, 50, 51].

A clear instance of the complexity of free drawing tasks is CDT which is widely used in the first steps of detecting cognitive impairment and dementia (Fig. 1), as a stand-alone neuropsychological test or
included into composite screening batteries [20, 21, 52]. In its classical form [53], CDT requires patients to ‘draw the face of a clock with all the numbers and set the two hands to 10 after 11’. Many administration procedures and scoring systems have been proposed [54–57], but there is no consensus as to which is the most useful for dementia screening [20, 54]. The scoring systems share commonalities, but each may reflect different cognitive components and be correlated with different areas of brain atrophy [58]. However, independently from the scoring procedure, performance on CDT in demented patients seems to be inversely correlated with lesion load in medial temporal lobe, in subcortical structures and in periventricular white matter [59, 60] and might provide prognostic information for cognitive decline [61, 62]. Some authors suggested that qualitative analysis of errors in clock drawing might improve diagnostic accuracy for dementia screening. For instance, errors such as inaccurate time setting, missing hands or numbers, and number substitutions or repetitions might be particularly useful for dementia screening [63]. Using appropriate error classification criteria, error analysis in CDT might be useful to differentiate different types of dementia, since conceptual errors are quite frequent in AD [64], but in a recent longitudinal study conceptual errors and perseverations were more frequently observed in the advanced stages of all dementing diseases, whereas spatial and planning errors were more frequent in mild-to-moderate dementia [65]. Errors in setting numbers and clock hands seem to correlate with regional hypometabolism in bilateral parietal and posterior temporal areas and in the right middle frontal gyrus [58]. By the same token, it has been recently observed that different types of errors in demented patients’ performance on CDT might correlate with atrophy in different brain regions, within frontal, parietal and temporal lobes [66].

One strategy for improving diagnostic accuracy of CDT is to compare the drawing to command condition with a copy condition, in which subjects are required to copy a pre-drawn clock, since in the copy condition performance would rely only on a subset of cognitive functions centered on visuoperceptual and constructional skills [16]. However, although copying seems to directly assess the patient’s ability to reproduce a figure, even this task can imply problem solving and executive abilities and is affected by age, educational level, and even cultural background [49]. In assessing copying abilities, it is therefore necessary to adopt standardized tasks, with solid normative data, and to use different kinds of stimuli, well-known or novel, of graded complexity from simple shapes, such as circles and squares, to complex figures, such as a cube (Fig. 2), two intersecting pentagons (Fig. 3), or the Rey-Osterreith Complex Figure (ROCF) [67, 68]. In particular, the copy of the ROCF (Fig. 4) is often used in the assessment of drawing abilities in individuals with dementia, and provides an opportunity to assess copying procedures together with copying accuracy [67]. The copy of the ROCF has high sensitivity for detecting brain damage, since it involves a wide network of brain areas, including...
Fig. 3. Copying of geometrical drawings: interlocking pentagons (model on the top). Simplifications and distortions in drawings by patients with Alzheimer’s disease (first row); perseverations, simplifications and spatial distortions in patients with vascular dementia (second row); simplification, distortions and planning errors in patients with moderate to severe frontotemporal dementia (third row). The bottom figures show instances of near closing-in (on the left) and of adherent closing-in (on the right).

Frontal, superior temporal, posterior parietal and middle occipital cortex, more extensively in the right hemisphere [69]. Several neurofunctional studies in dementia showed that accuracy in copying the ROCF is related to metabolic rate in bilateral temporal-parietal cortex and occipital lobe, and in right frontal lobe, whereas the procedure used to copy, namely the tendency to draw first the main organizational lines of the model, correlated with metabolism in right lateral temporal cortex [70]. It is worth mentioning, however, that neurofunctional studies comparing copying of different kinds of stimuli in dementia demonstrated that the neural correlates of copying tasks, mainly centered in the posterior brain regions, differ as a function of task complexity [71]. Moreover, it appears advisable to complement evaluation of accuracy in copying tasks with a qualitative analysis of the patients’ production, which can provide further relevant information. This fact has been underlined above with respect to the CDT, and is also suggested by recent studies showing that the copy of two intersecting pentagons (Fig. 3), included in the Mini-Mental State Examination [72], may provide elements for differential diagnosis of degenerative
dementias [73, 74]. Although error analysis in drawing tasks may not reveal a straightforward procedure [22, 28, 44], several clinical phenomena observed in drawing have been sufficiently characterized in their possible cognitive and neural correlates, as it will be discussed in the next sections of this paper.

It is important to stress, however, that in the diagnostic work-up for dementia performance on drawing tasks, and qualitative error analysis should be complemented by assessment of the deficits that can contribute to constructional apraxia. As it will be discussed later, mechanisms inducing errors in spontaneous drawing or in copying may encompass basic deficits in visuoperceptual abilities, spatial attention, spatial working memory or spatial planning, and executive function [75]. Particularly crucial in this respect seem to be visuospatial perception whose different aspects can be impaired in dementia [44, 75–78]. Visuospatial perception is often assessed by tests such as Benton’s Judgement of Line Orientation, in which subjects are required to identify the lines that have the same angulation as those presented as stimuli [79], or by comprehensive test batteries assessing several aspects of visuoperceptual processing, such as Warrington and James’ Visual Object and Space Perception Battery [80], or the Battery for Visuospatial Abilities [4, 81]. The mechanisms underlying drawing disorders can also be clarified by means of planning/executive tests, such as Trail Making test [82] or tests for figural fluency [83], and other tests that have been used to assess patients with focal lesions. Motor programming deficits have also to be considered,
because patients with dementia may be affected by limb apraxia or optic ataxia with a strong impact on drawing. Therefore, these types of tests have to be used to evaluate the different forms and the different stages of dementia. To establish an algorithm for assessing the different abilities involved in the drawing process and the disorders than can cause constructional apraxia might provide a powerful tool to understand the genesis of drawing disorders and of the different types of errors in dementia.

**DRAWING DISORDERS IN ALZHEIMER’S DISEASE**

The observation that many brain regions in both hemispheres are involved in different aspects of drawing, and by implication of constructional tasks, can provide an explanation of the classical findings suggesting that CA is related to poor intellectual abilities in focal brain damaged patients [30], and can represent an index for diffuse cognitive deterioration, after lesions in either left [84] or right hemisphere [85]. According to old studies, in AD drawing disorders are present since early stages of the disease, and their severity increases as the illness progresses [22, 28]. However, more recent investigations have shown that in the early stages of AD simple models can be easily copied and that drawing disabilities are observed only with rather complex tasks [86, 87]. Several variables must therefore be taken into account in the study of drawing disorders in AD. Among these variables we will separately consider: (1) the drawing tasks used in AD and the corresponding kinds of errors; (2) the drawing disorders in prodromic (aMCI) and clinical forms of AD; (3) the drawing disabilities in early and late onset forms of AD; (4) the drawing disorders in different stages of evolution of AD; (5) the mechanisms giving rise to drawing disorders in AD patients.

**The drawing tasks used in AD and the corresponding kinds of errors**

An attempt at a systematic description of AD patients’ errors in a free drawing task [9] has shown frequent occurrence of simplifications, spatial alterations, and lack of perspective. However, since free drawing poses a heavy load on semantic memory, errors on this task (e.g., simplifications in drawing a house) may derive from impaired access to semantic knowledge or to impaired visuo perceptual processing [51]. For this reason, spontaneous drawing could be impaired in the early stages of AD, while copying may deteriorate later [88]. The reproduction of complex figures is particularly sensitive to the progression of the disease [89]. For instance, the ROCF may be reproduced in a simplified way, with single constitutive elements put one after the other, even in early AD (see Fig. 4). In these cases, patients seem able to recognize and reproduce single well-known elements but are unable to reproduce complex spatial relationships correctly. Another “simplification” error may consist in the reproduction of more familiar or simpler figures instead of more complex ones (e.g., a square instead of a diamond). As the disease progresses, patients usually become unable even to draw simple figures correctly, as they no longer had access to well-consolidated motor subroutines and can show peculiar patterns of behavior, such as the ‘closing-in’ phenomenon [23–25, 90, 91] (see Figs. 2 and 3).

**Drawing disorders in prodromic (aMCI) and clinical forms of AD**

Drawing disorders are not usually present in MCI, at least when copying of simple figures is employed [92]. However, a different pattern might emerge as a function of clinical characteristics of MCI patients, particularly if more complex tasks are used. In a study on brain morphometric correlates of CDT in patients with MCI or AD, Thomann et al. [86] observed that, even though MCI patients did not differ from matched healthy controls in copying simple geometrical drawings, CDT could discriminate MCI from matched healthy controls and AD from MCI patients. Analogously, in a further brain morphometric study on MCI and AD patients, accuracy in copying the ROCF was significantly different in MCI patients, AD patients, and healthy controls [87]. It has also been observed that MCI patients make conceptual and graphic errors more often than matched healthy controls, and that AD patients score lower and make significantly more conceptual, graphic, and spatial-planning mistakes than MCI individuals, thus suggesting that a detailed scoring system is necessary to differentiate individuals with MCI from healthy adults [63, 93]. Scoring systems focusing on hand and number placement might better differentiate MCI individuals from healthy controls, but the solidity of CDT as a screening tool for MCI has been questioned [94]. In this respect, it should be taken into account that cognitive characteristics of MCI individuals might correlate with performance on CDT. It is very important to acknowledge at this point that MCI
patients are rather heterogeneous from the etiological point of view and that amnesic MCI can be considered as that most related to development of AD [95, 96]. In line with this caveat is the fact that, a recent study contrasting MCI of the amnesic, dysexecutive, or multi-domain type on CDT [97] confirmed that aMCI did not differ from a matched sample of healthy controls, whereas both dysexecutive and multi-domain MCI individuals made a significantly larger number of errors in hand and number placement.

Poor performance on CDT might predict future development of AD in patients with MCI [98], but not all qualitative scoring procedures might be sufficiently sensitive to capture longitudinal changes of CDT from MCI to AD [61]. A recent meta-analysis [99] confirmed that the CDT might reveal a useful measure of cognitive decline over time, and that conceptual clock drawing errors (with particular mention of hand and number placement) would be most informative about the cognitive decline. This meta-analysis also showed that CDT might differentiate individuals who are going to develop dementia in subsequent years, thus being included among the tests helpful for predicting conversion to dementia.

**Drawing disabilities in early and late onset forms of AD**

The structural or functional neuroimaging abnormalities correlate with the clinical features in AD patients, because typical late-onset patients (LOAD) present with memory disorders and medial temporal lobe atrophy, whereas focal neocortical forms of AD prevail in early-onset patients (EOAD) and are associated with executive-behavioral disorders, logopenic aphasia, and (in posterior cortical atrophy) with progressive visual-spatial disorders [100, 101]. Some authors have, therefore, examined the relations between age at onset and drawing disorders in AD patients, and reported a high prevalence of visuocognitive and visuospatial disabilities, assessed with the copy of the ROCF, in EOAD patients [101–103]. In some studies [102, 103], but not in others [101], these drawing disorders were associated with visuospatial deficits. Furthermore, some studies [104] have shown that the presence and severity of drawing disorders certainly depends on the AD phenotype and not just on age. Most frequent and severe drawing disorders are found in posterior cortical atrophy (PCA), which is associated with prominent visuospatial impairments and relative preservation of memory, insight, and judgment [105]. PCA is associated with atrophy in the occipital, parietal, and posterior temporal lobes [106], but recently a lesion of long white-matter tracts, including the superior and inferior longitudinal fasciculi and the inferior fronto-occipital fasciculus, has also been documented [107]. This early-onset AD-related pathology gives rise to complex visuospatial difficulties, such as visuoperceptual impairments [108], optic ataxia, simultanagnosia, gaze apraxia (often with the complete spectrum of Balint syndrome), and even egocentric [109, 110] or allocentric [111] unilateral spatial neglect. All such visuospatial impairments have a strong impact on drawing abilities, affecting both spontaneous drawing and copying, and giving rise to severe displacements of drawing elements, omissions, gross spatial distortions (‘exploded drawings’) [112].

**Drawing disorders in different stages of evolution of AD**

A strict relation between the progression of visuoperceptual spatial impairment and the progressive deterioration of the performance on copying the ROCF has been reported [89] (see Fig. 4). Mild AD usually spares basic visual sensory processes and affects all high-order visual processes, along both the ventral (occipito-temporal) and the dorsal (occipito-parietal) visual streams [113]. Caine and Hodges [114] carried out two separated studies to investigate ventral and dorsal visual functions in a sample of AD patients at early stages of the disease. A small subsample of patients were impaired on visuospatial tasks, thus suggesting that this small group of patients with prominent visuospatial disorders might represent one end of a continuous spectrum at the other end of which are patients affected by a focal degenerative dementia involving occipito-parietal cortex, the so-called PCA [115–118]. In the moderate stages of AD, visuospatial impairments would become more evident, and play a relevant role in the development of constructional disorders [89]. More recently, Guerin et al. [119] examined the cognitive mechanisms underlying the constructional performance of AD patients at different stages of the disease by means of a copying task and of visuospatial tasks measuring spatial exploration (visual search tasks), judgment of spatial relations, and planning abilities. The results suggested that cognitive defects underlying constructional impairment in patients with AD involved the early phases of spatial-constructional processing, likely exploration and judgment of
spatial relationships, rather than the late stage of planning.

**Mechanisms giving rise to drawing disorders in AD patients**

The drawing impairment in AD patients may stem from different cognitive mechanisms.

In recent years, the different mechanisms giving rise to CA in AD patients have been tackled with by means of morphometric and neurofunctional studies. However, such studies did not provide strongly convergent data. For instance, in a sample of AD and MCI patients overall score on CDT was inversely correlated with atrophy in the middle and superior temporal gyri (BA 21 and 22) bilaterally, but more strongly in the left hemisphere, and in the left entorhinal area (BA 28) [86], whereas in a sample of AD patients impaired performance on the ROCF copying task was correlated with atrophy in several fronto-temporo-parietal regions in the right hemisphere [87]. In a further study [5], impaired performance on a copying test was correlated with bilateral parietal atrophy. The differences between such studies might originate from different characteristic of the AD samples, and from the neuropsychological measure used for assessing drawing. Several recent neurofunctional studies are illustrative of these sources of variability. In a large sample of AD patients, Shon et al. [120] found that drawing from memory or copying a clock face correlated with regional glucose metabolism in bilateral temporo-parietal regions, but these correlations changed as a function of dementia severity. Matsuoka et al. [58] reported another representative instance of variability in neurofunctional findings depending on the neuropsychological measure used for assessing drawing. Several recent neurofunctional studies are illustrative of these sources of variability. In a large sample of AD patients, Shon et al. [120] found that drawing from memory or copying a clock face correlated with regional glucose metabolism in bilateral temporo-parietal regions, but these correlations changed as a function of dementia severity. Matsuoka et al. [58] reported another representative instance of variability in neurofunctional findings depending on the neuropsychological measure used for assessing drawing. Several recent neurofunctional studies are illustrative of these sources of variability.

These data explain why the diffuse involvement of several areas of the brain in AD can impair drawing performance since the early clinical stages of the diseases, particularly when drawing is assessed by means of complex stimuli. Therefore, drawing is characteristically affected in AD, as well as visuospatial cognition in general [76].

**DRAWING DISORDERS IN THE DIFFERENTIAL DIAGNOSIS BETWEEN AD AND VASCULAR DEMENTIA OR OTHER DEGENERATIVE FORMS OF DEMENTIA**

The widespread involvement of several neural networks in drawing can explain the clinical observation of frequent drawing disorders in AD, but also implies that drawing disorders are often found in other forms of dementia, both of vascular and degenerative nature. In recent years a growing number of studies attempted to ascertain whether drawing could provide additional information for refining clinical differentiation among dementias. Here we will provide an overview of such studies, from which it will emerge that it can be difficult to disentangle drawing disorders in AD from those shown by patients affected by other kinds of progressive dementias, and that some distinctive features might be captured by qualitative analysis of drawing productions (see Table 1). As it will be argued in the next section, it is possible, however, that if patients with different etiologies share some neural lesions or cognitive defect, they will produce similar error types in drawing.

**Drawing disorders in vascular dementia**

Since AD and vascular dementia (VaD) are the most common forms of dementia in old age, several investigations tried to evaluate if drawing disorders can be useful to differentiate the two forms of dementia. In particular, a few studies compared drawing abilities in AD and VaD patients on the CDT, without providing convergent evidence. Looi and Sachdev [122] reviewed 11 studies comparing the performance of VaD and AD patients in the area of constructional apraxia, using tests such as block design, CDT, or the ROCF copying task. Eight of the studies showed no difference between the two groups, while in three it was found that the AD were less impaired. Wiechmann et al. [123] did not find difference in accuracy on CDT between AD and VaD. Instead, in a study on a sample of outpatients matched for general cognitive impairment VaD scored significantly lower than AD on CDT, likely related to poorer executive abilities in VaD patients [124]. Kitabayashi et al. [125] stratified their patient sample by general cognitive impairment, and found
that CDT can well discriminate patients with AD or VaD from healthy controls, but the main differences between AD and VaD patients were in the relative percentage of error types, with AD patients showing many conceptual and spatial/planning errors independently from disease severity and VaD patients showing conceptual errors and graphic difficulties only in presence of moderate cognitive impairment (see Fig. 1). Partially different findings have been reported by Fukui et al. [126], who did not find differences between AD and subcortical VaD patients at mild disease stages on CDT and figure copying, whereas at later stages VaD achieved significantly lower scores than AD in both drawing tasks. Graham et al. [127] also found that patients with subcortical VaD were more impaired than AD patients on executive/attentional functioning, and visuospatial and perceptual skills. In their recent review on CDT, Tan et al. [128] concluded that the overall accuracy on CDT in AD versus VaD patients did not provide consistent results, whereas qualitative analyses might reveal useful differences between the two groups of patients, since VaD patients tended to show more frequent executive/planning or perseveration errors than AD patients.

**Drawing disorders in frontotemporal dementia**

The relative preservation of visuospatial and constructional abilities is a feature capable of distinguishing FTD from other degenerative dementias and, notably, AD [129, 130]. Consistent with this idea, a few studies observed better performance on CDT in FTD compared with AD. For instance, in

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<td>Cormack [165]</td>
<td>Pentagon copying</td>
<td>LBD worse than AD</td>
</tr>
<tr>
<td>Connor [166]</td>
<td>Pentagon copying</td>
<td>No difference</td>
</tr>
<tr>
<td>Caffarra [167]</td>
<td>Figure copying, pentagon copying</td>
<td>No difference in figure copying, worse scores in LBD on an analytic scoring system for pentagon copying</td>
</tr>
</tbody>
</table>

AD, Alzheimer’s disease; VaD, vascular dementia; sVaD, subcortical VaD; FTD, frontotemporal dementia; bvFTD, behavioral variant of FTD; PA, progressive aphasia; LBD, Lewy body dementia; CDT, Clock drawing test; ROCF, copy of Rey’s complex figure. Reference numbering follows the text.
a sample of autopsy-verified FTD patients, Rascovsky et al. [131] observed similar scores in copying geometrical figures, but higher overall CDT scores in FTD compared to AD patients. In the same vein, Blair et al. [132] reported higher overall scores, and fewer conceptual, spatial, and planning errors in FTD compared to the AD group (see Fig. 1). Comparing the performance of AD patients with those of patients with right and left FTD, Razani et al. [133] also confirmed that the AD group displayed significant impairments in visual-constructional ability relative to the two FTD groups. On the other hand, Thompson et al. [134] observed that FTD patients outperformed AD patients in copying geometrical figures of different complexity showing a lower number of spatial errors, but were also characterized by a higher number of perseverative errors and of poor organization of the copy than AD patients. Thus spatial errors were highly predictive of AD, whereas organizational errors, perseverations, or overelaborated copies were significant predictors of FTD (see Figs. 2–4). Data supporting the hypothesis of a prevalence of spatial errors in the drawings of AD patients and of executive disorders in those of FTD patients have also been obtained in a study by Possin et al. [135], that used quantitative morphometric evaluation of structural MRI. These authors reported that a sample of patients with the behavioral variant of FTD performed better than a sample of AD patients in copying drawings, but also observed that the drawing impairments in the two patient groups might be based on different cognitive and neuroanatomical correlates. In AD patients poor figure copy was associated with performance on spatial perception and attention tasks, and correlated significantly with volumes in the right parietal cortex. In FTD patients, instead, performance on figure copy correlated significantly with scores on spatial planning and working memory tasks, and correlated with right dorsolateral prefrontal cortex volumes. These observations would fit clinical data demonstrating that patients with prominent behavioral disorders and frontal involvement (behavioral variant FTD, bvFTD) outperformed AD patients on ROCF immediate reproduction [136], and that patients with prominent language deficits (non-fluent progressive aphasia) did not differ from healthy controls on cube copying and ROCF immediate reproduction [137]. Therefore, several studies confirmed the basic tenet that visuospatial skills and drawing abilities are relatively preserved in FTD, but at least two studies did not detect differences in overall accuracy on ROCF immediate reproduction between bvFTD and AD patients [138, 139]. It is thus possible that during the disease course FTD patients might show relatively preserved constructional abilities in the early stages, whereas differences between FTD and AD might blur in late stages of the disease. This hypothesis might find support in a meta-analysis showing that constructional abilities assessed on copying the ROCF quickly deteriorate as FTD progresses [140], and in a longitudinal study showing a trend toward a steeper decline of copying abilities in FTD with respect to AD [141, but for contrasting findings see 142].

Divergences about drawing disorders in FTD will be reconciled by taking into account the heterogeneous clinical features and neuropathological underpinnings of the disease [108, 143]. In this respect, it is important to underline a distinction between the drawing abilities of patients with bvFTD and those of patients with the temporal variant or semantic dementia (SD), because both are relatively spared on copying tasks, but the latter can be either enhanced or selectively impaired in drawing from memory the typical shape of common objects. The seemingly contradictory data about drawing abilities in SD are illustrated by results obtained by Bozeat et al. [19] and by Miller et al. [144–146]. Bozeat et al. [19] showed that when SD patients are required to produce drawings of concrete objects from dictation of their names, their drawings are characterized by a loss of the distinctive features of the represented objects. On the other hand, Miller et al. [144–146] showed that in the early stages of SD, patients with left anterior temporal lobe atrophy and severe anomia increase their drawing abilities, copying with great precision and sometimes become artists. In these cases, artworks show an enhanced tendency to realism. This improvement of visuocinstructive and artistic abilities was attributed by Miller et al. [144, 146] to the sparing of the parietal lobe and of the right hemisphere, considered as dominant for the three key features of visual artistry: visual constructive ability, spatial attention, and internal representation. The apparent contradiction between data reported by Bozeat et al. [19] and by Miller et al. [144–146] is probably due to the different stage of evolution of SD in which observations were gathered (an early stage of SD, with unilateral left sided atrophy in the case of Miller et al. [144–146]) and a later stages of SD, with bilateral anterior temporal lobe (ATL) atrophy in the case of Bozeat et al. [19] observations. Gainotti [147, 148] has, indeed, recently reviewed data showing that the format of
conceptual representations is mainly verbal in the left ATL and mainly sensorial/pictorial in the right ATL. In the early stages of SD the atrophy of the left ATL can release, with a mechanism labeled “paradoxical functional facilitation” by Kapur [149] the pictorial representations stored in the right ATL, leading to an enhancement of pictorial abilities. On the other hand, in later stages the bilateral spread of atrophy provokes a complete disruption of verbal and non-verbal conceptual representations of concrete entities, leading to an inability to draw them from memory.

**Drawing disorders in Lewy body dementia**

In LBD, visuospatial difficulties are often early and prominent [150]. Visuospatial tasks, such as object size discrimination, form discrimination, overlapping figure identification, and visual counting tasks, may reveal more impaired performances in LBD than in AD, and these defects likely contribute to the disproportionate impairment in constructional tasks in LBD patients [151–153].

In line with these observations, case series of pathologically verified LBD [154] as well as systematic reviews [155] confirmed that an impairment in copying geometrical figures is very common in LBD patients since early stages of the disease. However, several studies employing CDT did not report significant differences in overall total score in LBD and AD patients [156, 157], likely because of the complexity of the task (see Fig. 1), although at least two studies reported significantly lower overall scores in LBD versus AD patients [158, 159]. A SPECT study showed that LBD patients with defective performance on CDT presented more marked hypoperfusion in a frontal-subcortical network, involving frontal eye fields and the thalamus, with respect to LBD patients with normal CDT scores [160]. A more recent neurofunctional investigation by PET revealed, instead, that, after controlling for overall cognitive impairment, there was a direct association between frontoparietal dysfunction and impaired CDT performance in LBD [161].

The early and prominent impairment of visuospatial processing in LBD led some authors to suggest that LBD patients may be characterized by parallel impairments in free drawing and in figure copying since early stages of the disease whereas AD patients would show relative sparing of figure copying ([162], but also see [163]). Likely because of its complexity, copy of the ROCF has not been extensively used in LBD, whereas some interesting observations came from the copy of the intersecting pentagons included in the MMSE. Ala et al. [164] found that patients with LBD were more likely than those with AD to copy pentagons incorrectly, and suggested that this impairment might help identifying LBD patients. Cormack et al. [165] confirmed that LBD patients show significantly lower performance in copying pentagons with respect to AD; moreover, while in AD the impairment in copying figures was correlated with general cognitive deterioration, in LBD drawing was correlated with visuospatial tasks only, thus suggesting the existence of a specific defect in this disease. Different scoring criteria can likely explain the reason why other authors failed to detect differences between LBD and AD in pentagon copying [108, 159], but more extended copying tests might show significantly lower performance in LBD with respect to AD patients [108]. Recently, it has been demonstrated that qualitative analysis of the pentagon copy test can provide additional information for distinguishing AD from LBD patients, since a lower number of angles is among distinctive features of LBD patients' copies [73, 167], and can also predict development of LBD in MCI patients [74].

**PECULIAR ASPECTS OF DRAWING DISORDERS IN DEMENTIA**

In the previous sections of this paper it has been repeatedly underlined that qualitative observations (i.e., errors analysis) might provide additional information about the cognitive and neural correlates of drawing disorders in patients with different kinds of dementia. Some qualitative phenomena observed in drawing tasks in demented patients deserve, therefore, some brief comments, because of their frequency and their potential clinical usefulness.

**Graphic perseveration**

Generally speaking, perseveration can be considered among “productive” (or “positive”) pathological signs, since it consists in iterative behavioral responses, not adequate to the current stimulus (see Figs. 2–4) [168]. Patients may produce the same figure repeatedly, in response to only one stimulus, or replicate stimuli’s elements (continuous perseveration, according to Sandson and Albert) [169]; on other occasions, patients may inappropriately draw figures already drawn in previous trials, instead of reproducing the current stimulus (recurrent perseveration) [169].
A specific kind of perseveration is observed in CDT, when patients produce repeatedly the same numbers, or start numeration over and over (see Fig. 1). These errors in clock drawings have been described in a small proportion of AD patients [170], but would increase in moderate to severe stages of the disease [88]. Ryan et al. [171] compared graphic performances on the Bender visuomotor Gestalt test and on CDT in AD with or without wandering, and observed a larger number of both continuous and recurrent perseverative errors in individuals showing the wandering phenomenon, thus suggesting a relationship between perseveration and behavioral control.

A first study specifically comparing graphic perseverations in patients with AD or VaD showed that perseverations were more frequent in VaD than in AD [172]. However, Cosentino et al. [173] assessed patients on CDT and on copying clocks and observed that perseveration and closing-in (see below) in AD and VaD were more frequent in patients with higher number of white matter lesions and with more marked impairment on executive frontal tasks. These findings suggested that the executive impairment associated with frontal-subcortical dysfunction contributes to the genesis of perseveration in clock drawing in dementia. Recently, in a retrospective study on a large patient sample, De Lucia et al. [174] observed that frequency of graphic perseverative errors was similar in AD and VaD, and that patients with moderate-to-severe dementia produced a significantly higher number of perseverations than individuals with mild dementia. In both groups graphic perseverations were related with frontal and visuoconstructional impairments, thus supporting the view that frontal-executive defects can hamper inhibition of iterative graphic productions.

In FTD, patients’ perseveration in drawing would be often present also in early stages of the disease while specific disturbances in reproducing spatial relationships would become evident later during the course of the disease [175]. In a systematic analysis of errors observed in a wide range of neuropsychological tests, Thompson et al. [134] reported that FTD patients outperformed a group of AD patients in drawing tasks, but the most distinctive feature between the two groups were perseveration and poor overall organization (see Figs. 2–4). It is worth mentioning, however, that other studies focusing on qualitative error analysis [138, 139] did not observe a higher proportion of graphic perseverations in FTD with respect to AD.

In LBD, several authors suggested that less efficient executive control might induce higher frequency of perseveration than in AD [176, 177], but perseverations in drawing tasks have not been assessed specifically.

Rotation

In copying stimuli, some patients may respect spatial relationships among constituent elements but reproduce a model with general orientation different from the stimulus, e.g., rotating the reproduction by 90 or 180 degrees (see Fig. 4). This behavior has been already mentioned in early studies on constructional apraxia [27, 178, 179] and has been reported, although rarely, in subsequent descriptions of patients with brain lesions. From a consecutive unselected series of 240 neurological patients, Solms et al. [180] identified 16 patients who reproduced the ROCF with its major axis vertically rotated, independently from accuracy in reproducing spatial relationships among inner elements. Seven of these patients had diffuse cerebral involvement, but all remaining cases showed a lesion involving frontal regions. The authors suggested that this behavior could reflect the lack of planning and verification abilities of frontal patients [180]. In another case series of patients with AD, rotation of one or both items of the MMSE interlocking pentagons was reported in about 5% of patients [181]. Frequency of rotation of MMSE pentagons would not differ in AD or LBD patients, but it could progressively increase during the disease course in LBD patients [73].

Recent findings suggest that complex visuoperceptual and planning mechanisms might contribute to the genesis of rotations. In a retrospective study on a large sample of patients with MCI or degenerative dementia who underwent copy and recall of the ROCF, Isella et al. [182] observed rotation on the copy condition in 2.7% patients and on recall in 3.3% patients. In a subsequent prospective study on a mixed sample of patients with degenerative dementias or focal brain lesions, rotation at the copy of the ROCF was associated with visuospatial and selective attention impairments, and with more severe temporoparieto-occipital atrophy or hypometabolism, whereas no specific profile of cognitive impairment distinguished patients with rotation at recall of the ROCF, in whom frontal abnormalities were more frequent [183].

Closing-in

In copying tasks, demented patients often show the tendency either to put the pencil directly over the model, producing a scrawl, or to overlap the lines...
of the model with those of the copy or to draw very near to the model’s elements (see Figs. 2 and 3). Such behavior, termed ‘closing-in’ (CI), has been first described by Mayer-Gross [23], and later reported more often in demented patients than after focal brain lesions [24, 90]. Among dementing syndromes, CI is predominant in AD [88, 184], suggesting the idea that CI might represent a neuropsychological marker for clinical diagnosis of AD [90, 185, 186]. One more recent study [25] confirmed that CI is more frequent in AD with respect to VaD patients, and that the two patient groups also tended to show qualitatively different CI phenomena, as AD patients more often overlapped at least one element of the copy onto the model (adherent-CI), whereas VaD patients more often drew close to the model without overlapping onto it (near-CI). On the other hand, one retrospective study showed that CI is as frequent in AD as in FTD patients, but did not provide qualitative observations [187]. Indeed, in a sample of patients with the behavioral variant of FTD, near-CI has been observed more frequently than adherent-CI, and was often (but not obligatorily) associated with other imitation and utilization behaviors [188]. Notably, more frequent occurrence of near-CI than of adherent-CI has been recently reported also in non-demented Parkinson’s disease patients [189], whereas the reverse pattern has been observed in Parkinson’s disease dementia [190].

In his original description, Mayer-Gross [23] ascribed CI to a disturbance in performing spatial movements of hands and fingers correctly. In later years, interpretative accounts of CI clustered within two main streams. The first interpretative framework might originate from the consideration that CI could occur when patients who are unable to structure an empty space look for a reference point to solve difficult constructional dilemmas [191]. Following this suggestion, in recent years CI has been considered as a compensatory behavior implemented to overcome deficits of visuospatial skills or of visuospatial working memory [192]. In this ‘compensation’ account, patients with impaired ability to represent the model, or to hold its representation for the need of reproducing it, might tend to close-in to the model in the attempt to reduce their difficulties. Such a ‘compensation hypothesis’ received support by the finding that complex models could increase severity of CI in AD patients [192], and by the observation that AD patients with CI showed more severe impairments on several visuospatial tests, compared to AD patients without CI [193].

The alternative account might be traced back to the hypothesis that CI might represent a primitive reflex leading patients to be strongly attracted by the model and be unable to detach from it [24]. In this ‘attraction’ framework, CI would represent a ‘default’ behavior released by defects of attentional-executive abilities [194, 195]. In analogy with the ‘compensation’ hypothesis, the ‘attraction’ hypothesis predicts that CI would be enhanced in dealing with complex models because they likely imply high attentional load, and reduce available resources for monitoring graphic productions. However, this hypothesis specifically envisages strong correlations between CI and frontal/executive dysfunction, and foresees that CI can be triggered by high attention-demanding task conditions. Several converging pieces of evidence supported this account, since a significant correlation between CI and frontal/executive dysfunction has been reported in MCI [196], AD [191], VaD [25], and Parkinson’s disease [189], and in such diseases copying geometrical figures in dual-task conditions enhanced CI [25, 184, 197]. A first attempt at identifying the neural correlates of CI in AD patients would point to bilateral orbito-frontal cortex as the area in which atrophy was significantly associated to presence of CI [198].

Most available evidence would thus suggest that, independently from the diagnosis, patients sharing similar cognitive impairment might produce CI, and this attempt at identifying cognitive and neural correlates of specific errors might provide further insights on drawing disorders in dementia.

CONCLUDING REMARKS

It is interesting to note, at the end of this review, that several patients reported by Kleist [27] and Mayer Gross [23] in the earliest studies on constructional apraxia and drawing disorders were affected by dementia. It is also worth of note that, in one of the first studies aiming to investigate with standardized tests the patterns of neuropsychological impairment shown by various diagnostic groups of dementia, Gainotti et al. [199] showed that AD patients were particularly impaired on memory and drawing tasks. In another study, Villa et al. [200] showed that the coexistence of mental deterioration was more strongly associated with drawing disabilities than laterality of lesion, or intrahemispheric locus of lesion in focal brain-damaged patients. More recent investigations have followed two complementary strategies.
to clarify incidence and qualitative aspects of drawing disabilities observed in AD and other forms of dementia. The first strategy consisted in adopting short drawing tasks that could be particularly appropriate to investigate drawing disorders in demented patients. The second strategy consisted in taking into account the nature and the qualitative aspects of drawing disorders that could facilitate the differential diagnosis between AD and the other clinical forms of dementia.

The most popular task adopted according to the first strategic approach is CDT, which allows obtaining different scores (e.g., graphic difficulties, related to elementary motor disorders; stimulus bound responses, resulting from inhibition difficulties; conceptual deficits, related to semantic memory disorders; perseverations resulting from frontal lobe dysfunctions and visuospatial disorders), and can be administered both under verbal command and in the copy of a pre-drawn clock (see Fig. 1). According to several authors [21, 88, 125, 126, 201, 202], the CDT is able to distinguish different severity levels and clinical forms of dementia, but other authors reported contrasting findings with respect to both issues [65, 123, 128, 131, 158]. These deceiving results are probably due to two main factors. The first is that the CTD is basically a screening test and as such cannot provide a large and diversified amount of clinical information. The second is that many administration procedures and scoring systems have been proposed [20, 21, 54–57], but there is no consensus as to which is the most useful for dementia screening or for providing hints at a differential diagnosis among different forms of dementia. It is also possible that different diagnostic criteria and, above all, rate of diagnostic uncertainty might have biased some of the findings reported in the present review, and this concern might only be overcome by gathering further well-designed and accurate studies on drawing disorders in the different forms of dementia.

More substantial results have been obtained following the second research strategy, because several studies have shown that the discrepancy between command and copy condition (in the CDT or in other drawing tasks) is greater in forms of dementia, such as the subcortical vascular dementia [127, 128] or the bvFTD [129, 130, 134–136], which are characterized by a prominent deficit of the executive and control functions. Furthermore, the drawing impairments in AD and FTD patients could be based on different cognitive and neuroanatomical mechanisms in agreement with the different prevalence of lesions in these two diseases [134, 135]: in AD patients poor figure copy was associated with performance on spatial perception and attentional tasks, and correlated significantly with volumes in the right parietal cortex; in FTD patients, instead, performance on figure copy correlated significantly with scores on spatial planning and working memory tasks, and was connected with right dorsolateral prefrontal cortex volumes. Very different from the drawing disorders of patients with bvFTD are those of patients with semantic dementia, because both obtain good performances on copying tasks, but the latter are selectively impaired in drawing from memory the typical shape of common objects. In fact, when SD patients were given the names of concrete objects and asked to produce the corresponding designs, these drawings were characterized by a loss of the distinctive features of the represented objects, resulting from disruption of the corresponding conceptual representations, stored in the right and left anterior temporal lobes [19, 203]. Copying tasks are, therefore, substantially spared in degenerative diseases mainly involving the frontal (bvFTD) or temporal (SD) cortices and might be mildly impaired in the early stages of standard forms of AD, in which atrophy prevails in the medial temporal lobes, although neuroimaging studies repeatedly showed a pattern of reduced metabolism in the tempo-parietal areas [204, 205]. A much more severe impairment in copying tasks is observed in early-onset AD patients and in PCA variant, in which severe constructive defects are usually associated with progressive visual-spatial disorders, including Balint syndrome and unilateral spatial neglect [100, 101]. A very similar pattern of drawing impairment is found in LBD, in which visuospatial difficulties are often early and prominent and contribute to the disproportionate impairment in constructional tasks observed in these patients [151–153].

We can, therefore, conclude that the pattern of drawing disorders observed in AD and in other forms of dementia can be explained if we take into account the main functions of the neural networks involved in these forms of degenerative diseases. Among the peculiar drawing patterns that are observed in the moderate-to-severe forms of dementia the most important is certainly the ‘closing-in’ symptom (see Figs. 2 and 3), which is significantly more frequent and more severe in AD than in vascular forms of dementia [24, 25], because AD patients tend to put the pencil directly over the model, or to overlap the lines of the model with those of the copy (adherent-CI), whereas VaD patients more often draw in close proximity to the model without overlapping onto it.
(near-CI). If the CI might improve the differential diagnosis between AD and VaD, it is less useful in the distinction between AD and FTD, because the role of the frontal lobes in its production is still under scrutiny. From this point of view, it is important that recent neuroanatomical data demonstrated the selective association of atrophy in the orbito-frontal areas, implied in inhibiting primitive reflexes, with the closing-in phenomenon [198]. Even if several neuropsychological and pathophysiological aspects of drawing disorders in AD and other forms of dementia require further clarification, the clinical interest of drawing tasks in the differential diagnosis among different forms of dementia can, therefore, be considered as firmly established, particularly taking into account analysis of drawing errors more than overall accuracy in drawing.

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