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Review

The frontal lobe role in memory: a review of convergent evidence and implications for the Wada memory test

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Abstract

Functional imaging studies have implicated the frontal lobe in many of the memory processes often thought to be the domain of medial temporal structures. Results from fMRI studies of normal subjects have suggested that some components of memory formation, including those components tested during the Wada memory test, may involve frontal lobe regions. Specific behavioral disruptions during carotid amytal injections support a model for frontal lobe anesthesia in explaining results of the Wada memory test. Cortical stimulation data suggest that frontal lobe disruption is sufficient to cause memory disturbances. The convergence of evidence suggests frontal lobe memory may limit the predictive value of the Wada memory test in defining the risk of memory loss following temporal lobectomy. © 2002 Elsevier Science (USA). All rights reserved.

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1. Introduction

Recent advances in functional imaging, especially functional magnetic resonance imaging (fMRI), have refocused attention on the role of the frontal lobes in memory processes [1]. Additional evidence from lesion studies, and from cortical stimulation mapping, supports such roles for the frontal lobe. Results from material-by-hemisphere interactions when different stimuli are presented during the Wada memory test are also reviewed. The collective data and anatomic basis for the Wada test leave open the possibility that the results of the Wada memory test may be driven by impairment of frontal lobe function. Portions of this review have been presented in abstract form [2].

2. Functional imaging of frontal and medial temporal regions

Attempts to localize anatomical substrates for memory processes have historically focused on the role of the medial temporal lobe (MTL). This broad anatomical term incorporates the hippocampus, parahippocampal gyrus, entorhinal cortex, and, in some uses, the amygdala. The severe amnesia observed following bilateral MTL injury, as in the famous case of H.M., who underwent surgical removal of both MTLs [3], has left no doubt as to the importance of this area in memory. Injury to other cortical areas has been suggested to impact memory [4] and particular interest in frontal lobe involvement in memory processes has increased with the advent of modern functional neuroimaging techniques [5]. Early functional imaging using positron emission tomography (PET) revealed frontal involvement in memory retrieval processes [6,7]. This has since been confirmed with fMRI and across a variety of types of memory, including memory formation, or encoding [5], and retrieval [7,8].

Recent evidence from functional magnetic resonance imaging (fMRI) of normal subjects suggests involvement of certain frontal lobe regions, along with MTL regions, during memory encoding, especially during successful encoding (i.e., tasks that lead to successful memory for an event). In one set of experiments from Washington University [9], subjects were presented items visually with

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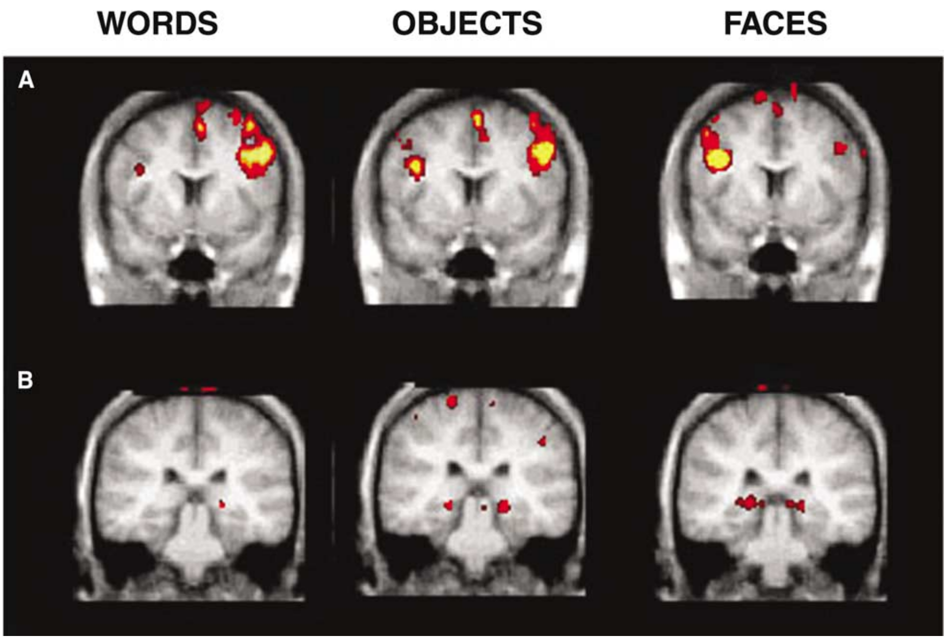

Fig. 1. fMRI data from normal subjects during encoding (superimposed on coronal anatomical images, right hemisphere on the left of each image). Memorization of words, pictures (objects), and faces altered lateralization of frontal lobe activation (A) to a greater extent than medial temporal activation (B).

the instructions to memorize these items. These tasks led to increased activation in frontal and medial temporal lobe regions. The lateralization of the observed frontal activations was influenced by the type of items encoded (Fig. 1). Specifically, memorization of words produced strong left-lateralized frontal activation, memorization of namable pictures produced strong bilateral frontal activation, and memorization of unfamiliar faces yielded strong right frontal activation. The activation pattern observed in medial temporal structures followed a similar pattern but with less clear lateralization. Left MTL activation was present through all of the different conditions, regardless of material type. In memorization of words, MTL activation was seen primarily in the left hemisphere, with no significant right MTL activation. Memorization of pictures gave increased MTL activation bilaterally. Strong right-hemisphere and persistent left-hemisphere MTL activation was the common pattern with memorization of faces. While the right-sided activation was considerably stronger than the left MTL activation during face memorization, the left-sided MTL activation for face memorization was as strong as the left MTL activation seen for memorization of words [9]. Thus, the lateralizing effect of stimulus material was more pronounced for frontal lobe than for MTL. A similar pattern of material-specific activation of frontal areas, with strong left lateralization for verbal material and right lateralization for nonverbal material, has been described by others [8,10,11].

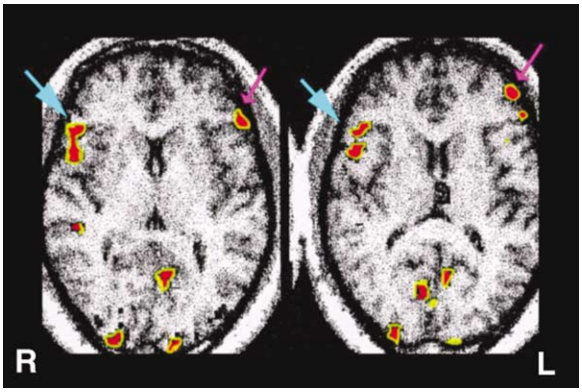

Fig. 4. fMRI during word encoding of a patient with right hemisphere dominance for language and left hemisphere dominance for verbal memory by Wada, corresponding to right inferior frontal activation (blue arrows) and left dorsolateral frontal activation (pink arrows). This supports the notion that the Wada memory results may be driven, at least in part, by the lateralization of dorsolateral frontal regions (W. Kelley and J. Ojemann, unpublished observations, NeuroImaging Laboratory, Washington University School of Medicine).

Further evidence supporting the importance of frontal contributions to memory formation comes from event-related fMRI studies examining item-by-item differences in memorization. Using these methods, activations in frontal cortex can be measured for each item presented during memorization. Performance on a subsequent memory test can then be used to compare encoding-related activity for items that are subsequently remembered well to activity for items that are later forgotten. If items that are subsequently remembered are compared with those not later remembered, frontal cortex activation is seen to be predictive of later successful retrieval of an item [10,12].

This combination of findings has led to speculation that frontal lobe activity provides a critical input to medial temporal lobe structures. The MTL may then bind together processed information from frontal and other cortical regions to form lasting, recollectable memory [5].

3. Preoperative prediction of memory loss: the cerebral amytal (Wada) test

Memory loss following neurosurgical procedures involving resection of the medial temporal lobes remains a feared complication. A common cause of medically intractable seizure disorders is mesial temporal sclerosis. Patients who suffer from epilepsy due to mesial temporal sclerosis may be candidates for surgical treatment if seizures can be demonstrated to arise from one temporal lobe. Surgery for intractable seizures requires extensive resection of medial temporal lobe structures, including hippocampus, parahippocampus, and enterorhinal cortex. Historically, patients who have suffered severe postoperative amnesia have been those who sustain, either from surgery or other causes, bilateral temporal lobe injury. The current method for testing the safety of medial temporal lobectomy with respect to postoperative amnesia, is the cerebral amytal, or Wada, test [13]. The Wada test attempts to establish lateralization of memory processes [14]. Along with determination of language dominance, memory formation is assessed during the injection through the internal carotid artery of the anesthetic agent sodium amytal. The ability to form memories while one hemisphere is anesthetized is taken as evidence of the ability of the contralateral hemisphere to support memory processes alone. The safety of resective surgery in the anesthetized hemisphere is thereby inferred from positive memory performance. The Wada test is not specific for medial temporal regions, however, and anesthetizes other ar32;eas of cortex as well. The test is performed by first advancing an angiographic catheter to the internal carotid artery. After the position is verified radiographically, an angiogram is performed to assess the intracranial circulation and to evaluate the degree of "crossover," or flow, to the contralateral hemisphere. In general, little or no flow to the posterior cerebral artery or posterior circulation is seen, although this may vary across individuals. Importantly, the medial temporal formation receives its blood supply principally from the posterior circulation and may not be directly anesthetized by an intracarotid injection (see below).

4. The Wada memory test

Two major methods exist for testing memory preoperatively with the Wada test [14]. Both methods involve the presentation of items following intracarotid injection of sodium amytal. After injection, the hemisphere injected becomes anesthetized. In the first method (the "Seattle" test), visually presented, namable objects are presented. Testing begins prior to injection of amytal. Patients are presented visual line drawings of objects and instructed to name them. A written sentence is then presented for patients to read. A card stating "Recall" cues patients to indicate what the previously presented object was. On the dominant hemisphere, patients will be initially unable to name the objects; however, the task is repeated through recovery of anesthesia. When naming has recovered, the task continues with more objects until successful performance reliably occurs, presumably corresponding to complete reversal of the anesthesia. If, during anesthesia of one hemisphere, a patient is unable to remember the items despite being able to name them, the hemisphere injected is then thought to be required for memory formation. In such a case, resection of the MTL on that side would be considered high risk for postoperative amnesia.

An alternative test (the "Montreal" test) assesses memory lateralization only by presenting items to be memorized during anesthesia of a hemisphere, and then testing for successful retrieval of the items after anesthesia of the hemisphere has worn off. Both words and pictures are typically presented for each injection of a hemisphere. Once function has fully returned after the injection, the patient is given a recognition test for the items presented. Both words and pictures are tested and successful recognition indicates that memory formation occurred despite anesthesia of the hemisphere. If recognition does not occur, this suggests that memory formation relies heavily on the hemisphere injected.

5. Limitations of the Wada test

In addition to anatomical questions about the validity of the Wada test to assess medial temporal lobe function, the documented predictive value of the Wada test is suboptimal. When postoperative neuropsychological tests are performed, the cerebral amytal test is found to be only partially predictive of postoperative verbal memory deficits [14]. Although severe global amnesia can be avoided if patients pass the cerebral amytal test prior to resection of medial temporal structures, significant material-specific memory losses (e.g., verbal memory loss but not visual memory loss) are not well predicted by these results. The Seattle method, using repeated stimulus presentation followed by recall after a short delay, had a 76% predictive value for correctly classifying patients who would or would not suffer significant verbal memory loss [14]. The Montreal method, using memory encoding during the cerebral amytal test with later testing of recognition, had only a 48% predictive value for postoperative verbal memory decline.

6. Areas of the brain anesthetized by the Wada test

Not only does the Wada test not accurately predict the effects of MTL removal, additional studies suggest the metabolic changes from amytal injection may not primarily involve MTL structures [15]. Medial temporal lobe structures receive perfusion from both the anterior (carotid) and posterior (vertebrobasilar) circulation [16]. Branches of the middle cerebral and anterior choroidal artery perfuse anterior medial temporal lobe structures, with posterior regions arising from branches of the posterior cerebral artery. The perfusion of these areas is variable across individuals [17].

Functionally, the degree of medial temporal perfusion may depend on the degree of flow through the posterior communicating artery [18]. Direct measurements of blood flow have suggested that little perfusion of the medial temporal lobe structures is achieved with intracarotid injections [19]. Although one study has suggested a partial reduction in blood flow is seen in medial temporal lobe following amytal injection [20], reliable perfusion requires a posterior cerebral injection [21]. Other studies have demonstrated inconsistent flow to anterior temporal lobe with nearly absent blood flow to posterior temporal lobe during intracarotid injections [22]. Interestingly, in another study that demonstrated poor medial temporal lobe distribution in the absence of filling through the posterior communicating artery, the Wada test was considered "successful" independent of flow to medial temporal regions [23]. In fact, attempts to selectively anesthetize medial temporal lobe regions with posterior cerebral injections suggested a much stronger ability to predict postsurgical memory outcome [24,25]. The technical difficulty of such selective injections, including anesthesia of other posterior circulation regions, such as the midbrain, and potential for severe complications have severely limited widespread acceptance of this method.

Conversely, injection of the internal carotid almost invariably perfuses ipsilateral frontal lobe [22]. A large anterior communicating artery, or other physiological shunt, may alter the distribution of anesthetic, but, if dosing is adequate, anesthesia of the ipsilateral frontal lobe is almost ensured, and is certain if a contralateral arm paresis is achieved. Thus, the assumption that testing memory following intracarotid injection is a reliable interrogation of medial temporal lobe function ignores potential effects of anesthetizing other cortical regions. In particular, a test that anesthetizes frontal lobe regions, that may be involved in various aspects of memory, may be driven by the role of these regions, even at the expense of any effect on anterior temporal lobe regions that may be perfused.

7. The frontal lobe affects memory when injured

Although many regions of cortex, including lateral temporal and parietal cortex, may also contribute to effects of an intracarotid amytal injection, the frontal lobe is, for reasons mentioned, especially of interest in considering effects of memory disruption. Lesion data, although classically focusing on frontal lobe functions such as speech production, response selection, and executive functions [1], also supports a role for frontal lobe in memory. Impairment of source memory [26], recognition memory [27], and particular susceptibility to false recognition [27,28] has been demonstrated in patients with frontal lobe lesions. Difficulty with retrieval has also been documented in frontal lobe lesions [29]. As suggested by the imaging literature [9], the degree of lateralization of frontal lobe involvement is highly driven by the material involved with dominant hemisphere mediation of verbal material and nondominant mediation of nonverbal material, such as faces [28].

8. Lateralization of memory functions: the Wada test and the frontal lobes

Thus, if disruption of frontal lobe function is a plausible source for memory changes seen during the Wada test, the lateralization of memory disruption should follow changes seen in frontal lobe activation for different tests. One paradigm used in the neuroimaging literature, encoding of words or faces during fMRI tasks, reveals very strong lateralization of frontal lobe activation in normal subjects [9], with poor lateralization of activation seen for medial temporal regions. If memory findings of the Wada test are, in part, driven by frontal lobe perfusion, a strong lateralizing effect should be seen with the Wada test if memory for faces is compared with memory for words. In a previously reported study [18], patients underwent a modification of the recognition portion of the Wada test, such that the memory test employed words and faces, similar to the neuroimaging study. Based on the fMRI activations, a strong degree of lateralization in the disruption of encoding of the test items would reflect the changes seen in frontal lobe activity. Thus, a strong left–right dissociation between memory formation for words and that for faces would correlate with the strong hemispheric interaction seen primarily for frontal lobe activation.

A total of 18 patients (all left dominant for language) were studied. Four words and four faces were presented during injection of each hemisphere. After recovery

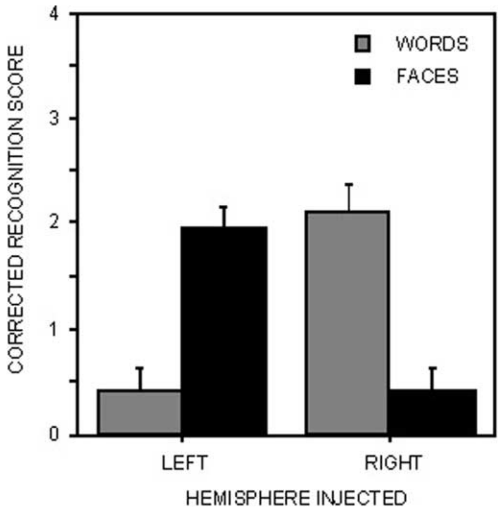

Fig. 2. Behavioral data during Wada testing of memory (see [18] for details). Right carotid injection selectively disrupted later recognition of faces while left carotid injections selectively disrupted later recognition of words. Study items were presented during the effects of amytal injection.

from the injection, recognition memory was tested with the four study items and four distracter items presented for both modalities (words and faces). Despite the relatively small number of items tested, a robust effect of later recognition was identified. A highly positive interaction between hemisphere injection and later recognition of item modality was seen (Fig. 2). Injection of the right hemisphere disrupted later recognition for faces, but not words, and injection of the left side preferentially disrupted later recognition for words over faces. Although an effect on medial temporal lobe function cannot be excluded in this case, the absence of highly lateralized fMRI changes in these areas with memory formation would appear discordant with the behavioral results during the Wada testing.

9. Is disruption of the frontal lobe sufficient?

Although the results of lateralization of memory formation during the Wada test are suggestive of frontal lobe involvement, and lesion literature suggests a role for the frontal lobe and memory, it is not clear that an acute "lesion" of the frontal lobe should cause the disruption in memory seen during the Wada testing. Another model of frontal lobe disruption, which provides anatomic specificity, is cortical stimulation. If focal frontal lobe disruption is sufficient to cause memory disruption, then anesthesia to this area is certainly a plausible explanation for the results seen during Wada memory testing.

In a second group of patients with frontal subdural grid electrodes placed for seizure monitoring, stimulation mapping during memory testing assessed whether frontal regions were essential (at least acutely) for memory processes [30]. This stimulation paradigm used object pictures as the test item for memory. In imaging studies, these items activate bilateral frontal lobe during memory formation. Thus, if disruption of frontal lobe regions is sufficient for memory impairment, cortical stimulation in either hemisphere should disrupt memory of these picture items.

Stimulation was performed in 10 patients (4 left-sided grids) through frontal grid electrodes (all left dominant for language) placed over the same dorsolateral frontal region as that involved in memory in imaging studies of normal subjects. Testing used a short-term memory paradigm highly similar to that used in the "Seattle protocol" of the Wada test. Patients were presented four slides (4 s each). The first was a picture of an object and patients were instructed to name and remember it. The next two slides were reading tasks and served as distracters. The fourth slide cued the patient to recall the initial picture. At each site mapped, stimuli were applied

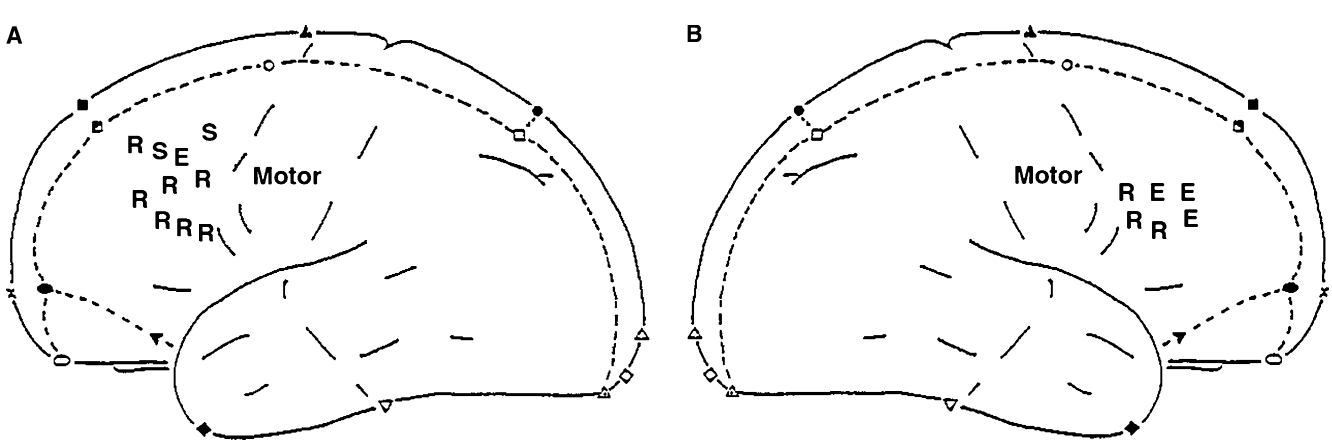

Fig. 3. Cortical stimulation mapping of frontal lobe memory. Stimulation disrupted successful recall when applied during encoding (E), storage (S), or retrieval (R). The summary of four patients with left-sided (A) and four with right-sided (B) stimulation are presented in relation to motor cortex. Left-sided sites were more common; none overlapped with naming sites. Similar premotor regions disrupted memory in both hemispheres, consistent with fMRI data.

either during encoding (naming of the picture), during the distracter task, or during recall. For each trial, stimulation was applied only once (during either encoding, distraction, or recall). One of the left-sided patients and one of the right-sided patients had a high baseline error rate, preventing mapping. Another of the right-sided patients made no errors with stimulation; he was found to have a region of cortical dysplasia in the dorsolateral frontal region where memory was disrupted memory in other patients. The remaining four left-sided (Fig. 3A) and three right-sided (Fig. 3B) patients all showed sites disrupting encoding or recall. Also consistent with imaging studies in normals, the number of sites of disruption was greater for the left than for the right hemisphere. Recall errors predominated on the left, with more balance between encoding and recall errors on the right. The location of these sites in premotor cortex, dorsal to classic Broca's area, closely matched the regions predicted from the imaging studies.

Stimulation studies have also implicated lateral temporal cortex in similar memory paradigms [31]. Transcranial magnetic stimulation has also demonstrated disruption of memory formation and retrieval with stimulation of frontal cortex [32].

10. Correlating fMRI and Wada test

In a separate patient, with left frontal seizures by surface recordings, Wada testing lateralized language to the left and verbal memory to the right (based on Montreal testing of verbal encoding). In the setting of this unusual dissociation, a fMRI was obtained (below) during tasks that activate both speech areas (e.g., classic Broca's area, inferior frontal gyrus) and the more dorsolateral frontal area discussed above as a region involved in memory. In this patient, inferior frontal actiavation during a langauge task was lateralized to the left. However, dorsolateral frontal activation during word encoding was lateralized to the right (Fig. 4). This unusual dissociation of language and verbal memory, determined by both Wada testing and fMRI in the same patient, further supports the model that dorsolateral frontal lobe activation is sufficient to explain memory lateralization on Wada testing.

A few studies have attempted to directly correlate fMRI and Wada testing. Primarily, these have demonstrated lateralized temporal-occipital activation, particularly in patients with mesial sclerosis [33,34]. Whether this represents lateralized memory processes or merely reflects lack of activation in abnormal (e.g., sclerotic) tissue remains to be determined. Ongoing studies with prospective fMRI, focusing on both frontal and mesial temporal activation, may determine whether frontal or temporal lobe activation is a better predictor of the results of the Wada test and, most importantly, which of these tools is the best predictor of cognitive outcome following temporal lobectomy.

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