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Recall, recognition, and the hippocampus: Reply to Yonelinas et al. (2004)

JOHN T. WIXTED and LARRY R. SQUIRE
University of California, San Diego, La Jolla, California

Two recent studies disagree about whether recall is more impaired than recognition (and recollection more than familiarity) in patients with damage limited to the hippocampus (Manns, Hopkins, Reed, Kitchener, & Squire, 2003; Yonelinas et al., 2002). Wixted and Squire (2004) pointed out that the disagreement about recall and recognition stems entirely from an outlying recognition score obtained by 1 of 55 control subjects in Yonelinas et al. (2002). In their comment on our paper, Yonelinas et al. (2004) minimize the importance of this result and argue that the role of the hippocampus in recollection and familiarity is best studied using different methods that attempt to measure these processes directly. Here, we argue that the recall versus recognition comparison is the strongest test because it relies on the fewest controversial assumptions, and that the other approaches explored by Yonelinas et al. (2002) rely on questionable, theory-laden assumptions. We also rehearse the reasons for concluding that the patients studied by Manns et al. (2003) have damage limited to the hippocampal region (hippocampus proper, dentate gyrus, and subiculum), and we emphasize that important questions remain about the patients studied by Yonelinas et al. (2002), inasmuch as no neuroanatomical information and minimal neuropsychological information has been provided.

Wixted and Squire (2004) recently evaluated two similar studies, one carried out by Yonelinas et al. (2002) and the other by Manns, Hopkins, Reed, Kitchener, and Squire (2003). Both studies were intended to test the effects of selective hippocampal damage on recall and recognition (and, indirectly, on recollection and familiarity), and both studies used the Rey Auditory Verbal Learning Test (RAVLT) to assess memory performance. In spite of their surface similarities, the two studies arrived at different conclusions. Yonelinas et al. (2002) interpreted their results to mean that selective hippocampal damage impairs recall more than recognition (and, theoretically, recollection more than familiarity). Manns et al. interpreted their results to mean that selective hippocampal damage impairs recall and recognition similarly.

Although both studies used the RAVLT, the procedures were not identical in every respect. In Manns et al. (2003), a list of 15 words was presented five times, and recall was tested after each list. Separately, a different list was presented five times, and recognition was tested after each. To evaluate the relative degree of impairment, Wixted and Squire (2004) compared recall and recognition performance following the first trial of each five-trial sequence. As can be seen in Figure 1 of Wixted and Squire, these data were uncontaminated by ceiling effects or outliers. Performance following Trials 2–5 was not analyzed, because, as might be expected, control recognition performance on these trials exhibited ceiling effects.

By contrast, Yonelinas et al. (2002) tested recall and recognition performance for the same list of words (recall first, then recognition) 20 min after that list had already been studied and recalled five times. The fact that the lists had been previously tested multiple times probably contributed to the ceiling effects in their data (effects that are evident in Figure 2 of Wixted & Squire, 2004), and the fact that recall and recognition were tested for the same previously studied (and previously recalled) words raises the possibility that recall contaminated recognition performance. Thus, the procedure used by Yonelinas et al. (2002) was not ideally suited to making comparisons between recall and recognition.

Nevertheless, Wixted and Squire (2004) argued that the results obtained by Yonelinas et al. (2002) agreed with those reported by Manns et al. (2003) despite appearances to the contrary. Specifically, Wixted and Squire noted that 1 of the 55 control subjects in Yonelinas et al. (2002) produced a recognition score that was an extreme outlier. When that single score was removed, the recall impairment exhibited by the patients was no longer more severe than their recognition impairment. The influence of the outlier was decisive. Had the removal of the outlying score shifted the p value for impaired recall versus impaired recognition only slightly (e.g., from $p = .027$ to .06), one would not argue that the effect was determined by this single subject. Yet when we removed the deviant score from the analysis, the p value changed from .027 to .56. Thus, in the data set presented by the authors, the outlying score of 1 out of 55 subjects determined the outcome

Correspondence concerning this article should be addressed to either J. T. Wixted or L. R. Squire, Department of Psychology, University of California at San Diego, La Jolla, CA 92093 (e-mail: jwixted@ucsd.edu, lsquire@ucsd.edu).

entirely. When that subject's outlying score is included, the results suggest one conclusion. When that subject's score is excluded, the results suggest a different conclusion. Accordingly, these data cannot be taken in support of the particular conclusion advanced by the authors.

In response, Yonelinas et al. (2004) now argue that a direct comparison between recall and recognition in amnesic patients is not a particularly useful way to test a dual-process model of recollection and familiarity. As they now see it, "even a failure to find a significantly greater recall deficit than recognition deficit would not be particularly informative for the model. Such a finding could simply be due to the fact that both the recall and recognition tests relied heavily on recollection" (p. 380).

This stance represents a significant departure from their original position. In their original report, Yonelinas et al. (2002) state:

> The hypoxics performed more poorly than the control subjects on the recall and recognition tests, and their recall deficit was significantly ($p < .05$) greater than their recognition deficit (Figure 1A and B). Recall requires recollection, whereas recognition judgments can be based on either recollection or on assessments of test-item familiarity . . . Therefore, the results indicate that recollection was more disrupted by hypoxia than was familiarity. (p. 1236)

Thus, the authors now offer a revised view about recall and recognition, which allows them to accept their model in the face of results that are different from what was originally predicted. We believe that their original logic and their original prediction were sound. Confidence in their model should increase if the results turn out as predicted, and should decrease if the results turn out otherwise. Once an obvious outlier was removed, the results reported by Yonelinas et al. (2002), like those reported by Manns et al. (2003), differed from the model's prediction. As such, confidence in their dual-process model and its application to hippocampal function should decrease.

Model-Based Estimates of Recollection and Familiarity

Because they no longer find the direct comparison between recall and recognition scores to be informative, Yonelinas et al. (2004) now argue that a better strategy is to rely on methods that treat recollection and familiarity as latent variables (e.g., structural equation modeling) or on methods that attempt to measure those two processes directly. However, the direct comparison between recall and recognition deficits is theoretically straightforward in that it relies on widely shared assumptions, whereas all of the other approaches considered by the authors (Yonelinas et al., 2002; Yonelinas et al., 2004) depend on more controversial assumptions. Some of the tests are compromised by ceiling effects as well.

Structural equation modeling. Yonelinas et al. (2002) used structural equation modeling to estimate the influence of recollection and familiarity (as latent variables) on the recall and recognition performance of the 56 patients as a function of coma duration. The analysis is somewhat odd in that it uses coma duration (ranging up to 32 h) rather than hypoxia duration (ranging up to 7 min) as the predictor variable for estimating recollection and familiarity. Because hypoxia is presumably the causative factor in determining brain damage, the use of hypoxia duration as the predictor variable would seem to make more sense. In any case, the coma duration analysis reported by Yonelinas et al. (2002) suggested that, as coma duration increases, recollection (but not familiarity) decreases.

Wixted and Squire (2004) questioned this analysis because it includes debatable assumptions (e.g., that recollection and familiarity are statistically uncorrelated from patient to patient) and because the patient data are compromised by marked ceiling effects. In response, Yonelinas et al. (2004) present a simulation to suggest that ceiling effects would not artifactually imply a two-process model when the data are actually generated by a one-process model. However, our criticism was not that the data might be explained better by a one-process model than by a two-process model (which is the criticism addressed by their simulation). Our concern was that the specific two-process model on which the analysis is based involves questionable assumptions and that, even if one accepts the model, ceiling effects might influence the conclusions drawn from fitting it to the patient data. The authors offer no new information about how the presence of ceiling effects might have affected their conclusions.

Remember/know and ROC analyses. Two additional methods used by Yonelinas et al. (2002) attempt to measure recollection and familiarity directly—namely, the remember/know procedure and an analysis of the receiver operating characteristic (ROC). These tests involved only a few patients (only 4 patients were studied using the remember/know procedure and only 3 were involved in the ROC analysis). Furthermore, these tests rely on one specific version of the generally accepted idea that recognition is based on recollection and familiarity. According to their version of a dual-process model, (1) recognition decisions are based either on recollection or on familiarity (i.e., both processes never contribute to a recognition decision), (2) recollection and familiarity are statistically independent processes, and (3) recollection is governed by a threshold process, whereas familiarity is governed by a signal-detection process.

These are not preposterous ideas, but they have been tested empirically and have often been questioned. For example, with regard to the remember/know procedure, remember responses were assumed by Yonelinas et al. (2002) to reflect the threshold recollection process directly, whereas know responses were assumed to reflect the signal-detection familiarity process directly. However, as first suggested by Donaldson (1996), a growing number of researchers argue that remember responses are also best characterized by a signal-detection process, not a threshold process (Dunn, 2004; Rotello, Macmillan, &

Reeder, 2004; Wixted & Stretch, in press). Wixted and Stretch, for example, have shown that hit and false alarm rates for remember responses are strongly correlated across subjects. That is, some subjects have relatively high remember hit and false alarm rates, suggesting a liberal remember criterion, whereas others have low remember hit and false alarm rates, suggesting a conservative remember criterion. Criterion effects like these offer compelling evidence that even remember responses are based on a detection process, not a threshold process (also see Hirshman & Henzler, 1998).

These considerations help to explain what is otherwise a seemingly inexplicable anomaly in the remember/know results reported by both Yonelinas et al. (2002) and Manns et al. (2003). The amnesic patients in both of these studies had remember false alarm rates of 16%, a finding that leads to the unlikely conclusion that the patients recollected 16% of the words that did not appear on the list. A remember false alarm rate as low as 11% has been described by prominent remember/know theorists as being "exceptionally high" (Gardiner & Richardson-Klavehn, 2000, p. 241n.2), but the patients in both Yonelinas et al. (2002) and Manns et al. had a remember false alarm rate 45% higher than even that exceptionally high standard.

Wixted and Squire (2004) suggested that this extraordinarily high remember false alarm rate could be understood within a signal-detection framework. From this point of view, remember responses are given to items that exceed a criterion level of memory strength. Usually, subjects use a strict setting for the remember criterion such that only items that are high in strength exceed the criterion. However, because patients do not have many high-strength memories, they may use a lower criterion setting that allows at least some of the target items to exceed it. A lower setting for the remember criterion would also allow more lures to exceed the criterion, thereby increasing the remember false alarm rate (Curran, Schacter, Norman, & Galluccio, 1997; Schacter, Curran, Galluccio, Milberg, & Bates, 1996; Wixted & Stretch, in press). This is the same explanation that is typically offered for the generally high false alarm rate exhibited by amnesic patients (e.g., Snodgrass & Corwin, 1988). And if this explanation is correct, the estimates of recollection and familiarity derived from the remember/know procedure are invalid, because the theory used to derive those estimates does not construe remember responses within a signal-detection framework.

Another possible explanation for the high remember false alarm rate is that the amnesic patients failed to understand the remember/know instructions fully. According to this view, the high remember false alarm rate essentially reflects random responding. In their response, Yonelinas et al. (2004) argue that they went to great lengths to ensure that their patients understood the remember/know instructions, but they offer no explanation for the extraordinarily high remember false alarm rate exhibited by their amnesic patients. Without a reasonable explanation as to why patients would claim to recollect 16% of the items that did not appear on the list, drawing strong conclusions from the remember/know data of 4 patients seems unwise.

The dual-process model of recognition memory advocated by Yonelinas and colleagues (Yonelinas, Kroll, Dobbins, Lazzara, & Knight, 1998; Yonelinas et al., 2002) also provides a method for extracting recollection and familiarity estimates from ROC data. In their original report (Yonelinas et al., 2002), 3 patients who had suffered cardiac arrest were found with this method to have selectively impaired recollection. Traditionally, ROC data are interpreted in terms of an unequal-variance signal-detection model (e.g., Ratcliff, Sheu, & Gronlund, 1992), a model that neither endorses nor rejects the idea that recognition is based on familiarity and recollection. However, this unequal-variance signal-detection model is inconsistent with the specific dual-process model that Yonelinas et al. (2002) used to estimate recollection and familiarity. Heathcote (2003) recently compared the two models in terms of their ability to fit ROC data and found that although the fits of both are both typically quite good, the standard detection model almost always provides a better fit (also see Glanzer, Kim, Hilford, & Adams, 1999). A good fit, per se, does not provide compelling evidence in favor of any quantitative model because, as discussed by Roberts and Pashler (2000), incorrect models often fit data quite well. Heathcote showed that the detection model provides not only a good fit but a reliably better fit than does the dual-process model proposed by Yonelinas (1994). Moreover, specific predictions made by this dual-process model concerning the shape of the ROC were not confirmed. Heathcote concluded that "These findings strongly reject Yonelinas's DPS (1994) theory as a model of item recognition ROCs and as an explanation of findings about *z*-ROC slope in item recognition. They do not, however, rule out some other type of dual-process theory or a role for recollection in item recognition" (p. 1224). That is, the general idea that recognition memory is based on recollection and familiarity may be correct, but the specific model used by Yonelinas et al. (2002) to extract estimates of those two processes may not be correct. If this dual-process model is incorrect, the estimates of recollection and familiarity that it provides from ROC data are unlikely to be accurate.

We are not suggesting that the available evidence disproves the dual-process model that Yonelinas et al. (2002) used to obtain their estimates. Instead, our point is simply that the model on which they relied is widely disputed. Accordingly, we regard the model-based analyses that they have reported as less compelling than the more straightforward comparison of the relative severity of recall and recognition deficits. The assumptions underlying that test are widely shared, and the results from that test suggest that recall and recognition (and, therefore, recollection and familiarity) are similarly impaired in the patients studied by Yonelinas et al. (2002) and Manns et al. (2003).

Patient Populations

Although the utility of the remember/know procedure can be questioned, Yonelinas et al. (2002) and Manns et al. (2003) both used this procedure to obtain estimates of recollection and familiarity in their patients. If the remember/know data are taken at face value in spite of the high remember false alarm rates, then the results reported by Yonelinas et al. (2002) suggest that recollection was differentially impaired in the 4 patients whom they studied. In contrast, the results reported by Manns et al. suggest that recollection and familiarity were similarly impaired in the 7 patients whom they studied. Thus, the data from the two studies are in conflict and they are in conflict even if they are analyzed using the independence remember/know method, which is based directly on the dual-process model advanced by Yonelinas and colleagues (Yonelinas, 1994; Yonelinas & Jacoby, 1995; Yonelinas et al., 2002). Given the small sample size (n = 4 patients) used by Yonelinas et al. (2002), Wixted and Squire (2004) suggested that the difference may simply be attributable to measurement error. Yonelinas et al. (2004) instead point to differences in the patient populations used in the two studies. Specifically, they suggest that the familiarity deficits observed by Manns et al. may indicate that their patients had damage to regions outside the hippocampus.

This is a bold claim, given that Manns et al. (2003) screened their patients for selective hippocampal damage and provided quantitative volumetric data from MRI scans. Yonelinas et al. (2002) provided no anatomical information at all. Speculation about the extent of brain damage in the patients studied by Manns et al. may have been invited by the fact that measurements were not reported for regions outside the medial temporal lobe. However, additional measurements from MR images have since been completed for the fusiform gyrus, insula, lateral temporal lobes, frontal lobes, parietal lobes, and occipital lobes. With one exception, these measurements yielded entirely normal values. The one exception was Patient R.S., who had a small parietal lobe. However, normalized to total brain size, even this value fell within two standard deviations of normal controls. Thus, for the patients studied by Manns et al., radiological evidence suggests that damage was limited to the hippocampal region.

Yonelinas et al. (2004) state that "the patients in the Manns et al. study appear to have been selected to include only patients with extremely low memory scores" and that "the patients in the Yonelinas et al. study were not selected on the basis of their memory impairments, but rather on the basis of their etiology: cardiac arrest" (p. 381). These comments highlight a critical difference between these two studies in their approach to testing a hypothesis concerning the effects of damage limited to the hippocampus. Yonelinas et al. (2002) chose to study patients with a common etiology and implied that Manns et al. (2003) chose their patients on the basis of the severity of memory impairment. In fact, the patients in Manns et al. were selected on the basis of independent evidence that damage was limited to the hippocampal region. The first sentence of the Participants section of Manns et al. reads "Seven amnesic patients (six men and one woman) with damage limited primarily to the hippocampal region (CA fields, dentate gyrus, and subicular complex) participated" (p. 177). Choosing patients on that basis makes sense given that the hypothesis in question concerns the effect of hippocampal damage on memory, not the effect of cardiac arrest, per se. It is true that, on average, the patients studied by Manns et al. had more severe memory impairment than did those studied by Yonelinas et al. (2002). Yet, in comparison with the profound impairments of patients like E.P. and H.M. (Scoville & Milner, 1957; Stefanacci, Buffalo, Schmolck, & Squire, 2000), the memory impairments of patients with limited hippocampal damage are best described as only moderately severe.

The patients studied by Yonelinas et al. (2002) experienced a period of hypoxia ranging from 30 sec to 7 min, but they were not screened for evidence of hippocampal damage. Accordingly, many of these patients (e.g., the ones who suffered from brief periods of hypoxia) may have had damage so slight that it would not have been evident in neuroimaging. These may be the same patients whose recall and recognition scores were on the ceiling. Had the authors been able to acquire brain images of their patients, it is easy to imagine that many would have been excluded on the basis of the absence of detectable hippocampal damage. But these patients were not excluded, and the average memory impairment exhibited by the patient group as a whole was mild.

Although many of the patients studied by Yonelinas et al. (2002) performed very well on the recall and recognition tests, a minority of the patients exhibited more severely impaired scores. Presumably, these were the patients who experienced longer periods of hypoxia. Some of these memory-impaired patients may have had damage limited to the hippocampus, but the idea that they all did is not a safe assumption. According to Grubb et al. (2000),

> The results of the structural and functional studies suggest that in patients who suffer from memory impairment after cardiac arrest, hypoxic injury results in generalized cerebral atrophy. In cognitive neuroscience, survivors of cardiac arrest have previously been used as models of selective hippocampal damage, on the assumption that hypoxic insult was confined to the hippocampal complex neurons. The results of the present study do not support that premise and imply that aspects of cognitive function other than memory are also likely to be affected by cardiac arrest. (p. 1513)

Similarly, in the most comprehensive review of the literature to date, Caine and Watson (2000) write,

> It would be quite incorrect for us to argue that an isolated amnesic syndrome with accompanying pathology solely affecting the hippocampus does not occasionally occur following an anoxic event . . . However, the mechanisms and distribution of neuronal damage in cerebral anoxia are

such as to render lesions specific to the hippocampus possible, but unlikely. (p. 87)

This conclusion accords with our own experience that etiology alone does not guarantee the locus and extent of neuropathology. When Zola-Morgan, Squire, Rempel, Clower, and Amaral (1992), for example, studied the effects of ischemia in monkeys, they found that damage can be limited to the hippocampus, but that damage also occurs to extrahippocampal areas as the duration of ischemia is extended. For this reason, individual patients with an etiology of hypoxia must be screened neuropsychologically to demonstrate the selective nature of brain impairment (i.e., memory is impaired but other intellectual functions are intact). In addition, patients must be screened neuroradiologically to demonstrate damage to the hippocampus without detectable damage in the adjacent parahippocampal gyrus or in other areas of neocortex (e.g., the frontal lobes). In the absence of independent measures of lesion locus and extent (and no such measures were obtained by Yonelinas et al., 2002), the assumption that damage was restricted to the hippocampus is simply not justified. Even if some of the model-based results reported by Yonelinas et al. (2002) are taken to support the claim that recollection was more impaired than familiarity (e.g., the structural equation modeling results or the remember/know results), such results would not bear on the hypothesis that the hippocampus subserves only recollection. Such a result might mean that, in some of the patients whom they tested, damage extended beyond the hippocampus to regions that are known to be differentially important to recollection (e.g., the frontal lobes; Janowsky, Shimamura, Kritchevsky, & Squire, 1989; Wheeler, Stuss, & Tulving, 1995).

Conclusion

Yonelinas et al. (2004) argue that whereas any one method can be criticized, the use of a multimethod approach that yields a consistent picture is nevertheless compelling. We agree with this assertion. What we disagree with is the idea that the data reported by Yonelinas et al. (2002) yield a consistent picture across the methods they employed. For the test involving the most subjects (by far) and the fewest controversial theoretical assumptions, recollection and familiarity were found to be similarly impaired once a clear outlier was removed from the analysis. For the tests involving many fewer subjects and more controversial assumptions, recollection was found to be more impaired than familiarity. Thus, overall, a consistent picture has not emerged. Because the patients studied by Manns et al. (2003) had radiological evidence of damage limited to the hippocampal region, whereas the locus and extent of putative lesions in the patients studied by Yonelinas et al. (2002) are unknown, the results of the study by Manns et al. bear most directly on the question of whether recall and recognition (and recollection and familiarity) are subserved by the hippocampus. The results of that study suggest that hippocampal damage impairs recollection and familiarity to a similar degree.

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