

Recall and recognition in mild hypoxia: using covariance structural modeling to test competing theories of explicit memory

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Abstract

To test theories of explicit memory in amnesia, we examined the effect of hypoxia on memory performance in a group of 56 survivors of sudden cardiac arrest. Structural equation modeling revealed that a single-factor explanation of recall and recognition was insufficient to account for performance, thus contradicting single-process models of explicit memory. A dual-process model of recall in which two processes (e.g., declarative memory and controlled search) contribute to recall performance, whereas only one process (e.g., declarative memory) underlies recognition performance, also failed to explain the results adequately. In contrast, a dual-process model of recognition provided an acceptable account of the data. In this model, two processes—recollection and familiarity—underlie recognition memory, whereas only the recollection process contributes to free recall. The best-fitting model was one in which hypoxia and aging led to deficits in recollection, but left familiarity unaffected. Moreover, a controlled search process was correlated with recollection, but was not associated with familiarity or the severity of hypoxia. The results support models of explicit memory in which recollection depends on the hippocampus and frontal lobes, whereas familiarity-based recognition relies on other brain regions.

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1. Introduction

Examinations of memory-impaired patients have played a critical role in shaping theories of explicit memory. Extensive empirical and theoretical work has been done to characterize the relationship between recall and recognition. Early theories assumed that a single underlying form of memory was responsible for both recall and recognition. More recently, neuropsychological dissociations between recall and recognition resulting from different types of brain injury have contributed to the rise of two competing classes of theory proposing at least two processes underlie recall and recognition. One class assumes that *declarative memory*, a general form of memory, contributes to recall and recognition whereas additional processes related to executive control contribute to recall but not recognition. The other class assumes that two memory processes, *recollection* and *familiarity*, contribute to recognition, whereas only

recollection contributes to recall. A large body of empirical evidence has contradicted single-process models, but differentiating between the latter two classes of models has proven difficult. In the current study, structural equation modeling was used to directly test the ability of these competing theories to explain observed relationships among recall performance, recognition performance and amnesic severity. The models of interest are first described in more detail, and previous attempts to contrast the models are briefly reviewed. Then, structural modeling methods are described and applied to a study that examined the amnesic effects of mild hypoxia on recall and recognition memory.

1.1. Single-process theories of explicit memory

Single-process theories assume that performance on tests of explicit memory, such as recall and recognition, rely on a single memory process or system (e.g., memory *strength*). Early single-process theorists noted that individuals performed consistently better on recognition than on recall and proposed that greater memory strength was

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required for recall than for recognition (e.g. Postman, 1963). Single-process strength theories remain attractive because of their simplicity. However, a large body of empirical literature now exists that is problematic for those theories. For example, simple strength-based accounts of recall and recognition do not explain behavioral dissociations between recall and recognition observed with manipulations such as word-frequency and list length (for a review of problems associated with simple single-process theories, see Gillund & Shiffrin, 1984). Moreover, neuropsychological studies have indicated that brain damage to frontal regions including prefrontal cortex can severely disrupt recall performance while leaving recognition relatively intact. Such results indicate that frontal lobe regions play a critical role in recall but are less important for recognition. These latter studies are described in more detail below.

1.2. *Dual-process theories of recall*

A number of multi-stage and multi-process models were designed in part to overcome the difficulties associated with the single-process models. One general class of theories assumes that a common form of memory underlies both recall and recognition, but that one or more additional processes or systems contribute to recall. These theories assume that additional controlled memory search and response monitoring processes contribute to recall. A classic example of this view is the generate-recognize model of recall (Kintsch, 1970). In this model, recall tests first require a generation process to produce items that may have been studied. The initial generation process is followed by a recognition decision process that is used to separate studied from non-studied items. In contrast, recognition tests require only the recognition process because the test items are provided to the subject and a generation process is therefore unnecessary. More recent models, however, do not limit the recall-specific process to generation of potential response items, but include a number of strategic control functions such as organizing a memory search (Squire & Zola, 1998), monitoring of retrieval contents (Moscovitch, 1994) and inhibition of irrelevant or inappropriate responses (Shimamura, 1995). For example, Squire and colleagues have advocated a dual-process view of recall in which *declarative memory*, a unified system involving the hippocampus and the surrounding medial temporal lobe, is required for both recall and recognition, whereas a frontal lobe system supports controlled memory search processes involved in free recall, but not recognition (e.g. Haist & Squire, 1992; Squire & Knowlton, 1995; Squire & Zola, 1998). Moscovitch has proposed a component-process model (Moscovitch, 1990, 1994) that is similar in some ways to that of Squire and colleagues. In this model, both recognition and recall rely on information computed by 'associative memory,' a modular, reflexive retrieval process localized in the hippocampus and surrounding neocortical structures. Additionally, depending on the processing requirements by the specific task, either recognition or recall

may reflect the contribution of control processes in the prefrontal cortex involved in directing and monitoring the products of associative memory.

Dual-process recall models have received support from studies showing the importance of the frontal lobes for tests of recall and tests of executive control. For example, lesions to prefrontal cortex can lead to disproportionately large deficits in recall compared to recognition (Janowsky, Shimamura, Kritchevsky, & Squire, 1989; Jetter, Poser, Freeman, & Markowitsch, 1986), suggesting that the frontal lobes contribute to recall to a greater extent than to recognition. Moreover, the recall deficits in patients with frontal lobe damage are often accompanied by impairments in measures of executive function that are thought to tap similar controlled search and monitoring processes, such as verbal fluency (Baldo, Shimamura, Delis, Kramer, & Kaplan, 2001; Bentler & Bonnett, 1980; Janowsky, Shimamura, & Squire, 1989a) and Wisconsin Card Sorting (Milner, Petrides, & Smith, 1985; Parkin, Walter, & Hunkin, 1995). Similarly, healthy aging also leads to reductions in recall and performance on some tests of executive function, but leaves recognition relatively unaffected (Parkin & Lawrence, 1994). Histological and neuroimaging examinations have shown that normal aging is associated with cell loss and reduced blood flow in the frontal lobes (Woodruff-Pak, 1997), providing further evidence for the role of the frontal lobes in supporting the control processes involved in recall but not recognition.

1.3. *Dual-process theories of recognition*

A separate class of dual-process theories assumes that two memory processes contribute to recognition judgments and that one of these also supports recall performance. That is, recognition can be based on the assessment of stimulus *familiarity* or on a *recollection* process whereby qualitative information about the study event is retrieved, such as when or where the event occurred (e.g. Atkinson & Juola, 1974; Jacoby, 1991; Mandler, 1980; Tulving, 1985). Performance on tests of free recall is expected to rely on recollection because the retrieval cues are not complete enough for a familiarity assessment. Evidence for the contribution of two processes to recognition comes from behavioral studies employing numerous behavioral measurement techniques, studies of patient populations, as well as event related potential and neuroimaging studies (for a review see Yonelinas, 2002).

In general, recollection is thought to be more severely disrupted in medial temporal lobe amnesia than is familiarity (Aggleton & Shaw, 1996; Yonelinas, Kroll, Dobbins, Lazzara, & Knight, 1998; Yonelinas et al., 2002). Moreover, several models further propose that different medial temporal lobe regions are involved in recollection and familiarity (Aggleton & Brown, 1999; Eichenbaum, Otto, & Cohen, 1994). According to these models, the hippocampus supports recollection, whereas the temporal lobe regions surrounding the hippocampus, such as the parahippocampal

gyrus, are involved in familiarity-based recognition. Evidence in support of these claims has been reviewed elsewhere (Aggleton & Brown, 1999; Eichenbaum et al., 1994; Yonelinas, 2002).

Although the latter models have focused on regions within the temporal lobe, the frontal lobes are also thought to be involved in these processes, and some models have proposed that the frontal lobes play a particularly important role in recollection (Dywan, Segalowitz, Henderson, & Jacoby, 1993; Schacter & Tulving, 1994; Wheeler, Stuss, & Tulving, 1997). In support of the latter claim, patients with frontal lobe damage exhibit severe deficits on recognition tasks requiring recollection, such as associative recognition and source recognition, but exhibit relatively mild deficits in simple item recognition tasks that could be based to a greater extent on assessments of familiarity (e.g. Janowsky, Shimamura, & Squire, 1989b; Shimamura, Janowsky, & Squire, 1990). Moreover, healthy aged subjects often exhibit selective deficits in recollection (for review see Yonelinas, 2002), and these deficits have been associated with deficits on tests of executive function related to frontal lobe functioning (e.g. Glisky, Polster, & Routhieaux, 1995; Janowsky, Shimamura, Kritchevsky, et al., 1989; Parkin & Walter, 1992).

1.4. Empirical tests of the dual-process theories

The two classes of dual-process theories are similar in assuming that two memory processes contribute to explicit memory tests. However, they represent conflicting views of how the two processes contribute to recall and recognition, and about the neuroanatomical substrates of those processes. Thus, the two classes of theory make different predictions about when recall and recognition will dissociate. A critical difference between the theories regards how hippocampal damage should influence recall and recognition performance. According to the dual-process recall theories, the same declarative or associative memory system plays a necessary role in both recall and recognition, and thus damage to the declarative system should lead to comparable deficits in these two types of tasks. Importantly, the medial temporal lobes in these models are assumed to reflect a relatively unified system such that damage to different regions within the MTL can lead to different degrees of memory impairment, but not to different types of memory impairment (Squire & Zola, 1998). By contrast, the dual-process recognition theories predict that amnesic patients with damage restricted to the hippocampus should generally exhibit more profound recall than recognition deficits, because the preserved familiarity process can be used to support performance on recognition, but not recall tests (Aggleton & Brown, 1999; Eichenbaum et al., 1994).

The results from studies examining amnesic patients with damage outside the hippocampus are less useful in differentiating among these models because both types of theory make similar predictions. For example, in patients

with damage including the hippocampus and the surrounding temporal lobe, several of the dual-process recognition models predict that both recollection and familiarity should be disrupted. Thus, severe deficits should be observed in recall as well as recognition for organic amnesia with etiologies such as stroke and temporal lobectomy (Yonelinas et al., 1998). Moreover, in patients with frontal lobe damage, both classes of theory predict that recall should be disproportionately disrupted compared to recognition.

Only a limited number of studies have directly contrasted recall and recognition in patients with selective hippocampal lesions. However, the bulk of existing evidence suggests that recall is disproportionately disrupted compared to recognition in these patients (but see Manns, Hopkins, Reed, Kitchener, & Squire, 2003 for an exception). For example, in a review of the published recall and recognition studies of amnesia, Aggleton and Shaw (1996) found that patients with relatively selective damage to the hippocampus or its efferents (e.g., hypoxic patients and patients with fornix or mammillary body damage) tested significantly below normal on recall tests, but were within the normal range on recognition tests. Additionally, Baddeley, Vargha-Khadem, and Mishkin (2001) and Vargha-Khadem, Gaidan, Watkins, Connelly, and Van Paesschen (1997) reported impaired recall but normal or nearly-normal recognition in individuals who suffered early bilateral hippocampal injury as a result of hypoxia. Although these latter results might reflect some form of developmental compensation in these young patients (see Manns & Squire, 1999), such an explanation does not easily explain the similar patterns seen in patients who suffered hypoxic events in adulthood. For example, Mayes et al. (2001) found impaired recall and normal or nearly-normal recognition in an individual with selective bilateral hippocampal damage presumably caused by hypoxia. Moreover, Yonelinas et al. (2002) examined a large group of cardiac-arrest patients with mild hypoxia and found that recall was significantly more impaired than recognition.

By contrast, patients with damage extending into temporal lobe regions surrounding the hippocampus exhibited pronounced impairments in both recall and recognition (e.g. Haist & Squire, 1992). Note that Reed and Squire (1997) reported that, across a wide variety of recognition tests, 6 patients with damage limited to the hippocampal formation (data from three of these were included in Aggleton & Shaw's review), all showed significant recognition deficits. Recall was not compared to recognition in this latter study, but Manns and Squire (1999) did so with three of the six patients. Unfortunately, the patients scored below the range for which normative recall data were available, so comparing the proportional deficits on recall and recognition was impossible. Additionally, the degree to which recollection was necessary to perform these tasks is unknown. This is important because both types of model predict that recall and recognition deficits may be highly similar if individuals must use recollection often to recognize test items.

1.5. The current study

Previous findings showing that hippocampal damage led to more pronounced recall than recognition deficits provide support for dual-process theories of recognition while posing difficulties for dual-process theories of recall (but see Manns & Squire, 1999). In the current study we aimed to provide a more rigorous test of these conflicting theories by examining the ability of the models to account for variations in performance observed across individuals. Prior studies have asked whether the theories could account for overall patterns of mean recall and recognition deficits seen in hippocampal patients. In the current study, we asked whether the models could account for individual differences in memory performance among these patients. Structural equation modeling was used to test the ability of the models to account for patterns of variation and covariation among recall and recognition across a relatively large group of patients.

In structural equation modeling, theoretical constructs are specified mathematically as factors underlying patterns of shared variance among observed measures. Models are specified from theory and are fit directly to the variance–covariance matrix of a set of observed variables. The reason structural equation modeling is relevant in this case is that the different theories of explicit memory make

different assumptions about which hypothetical processes contribute to more than one task and which processes are unique to a single type of task. For example, dual-process theories of recall assume that the MTL constitutes a single source of variance underlying performance on recall and recognition tests. In contrast, dual-process theories of recognition predict that the MTL contributes two sources of variance to recognition. Structural equation modeling has the advantage of providing direct statistical tests of the models, allowing one to determine whether rival models can be rejected on the basis of the data. More importantly, structural equation modeling allows direct statistical comparisons among alternative or competing models. Individual assumptions of the models can also be tested by relaxing or restricting parameter values and statistically evaluating the resulting change in fit. This strategy can be particularly useful in resolving debates about the model assumptions that are otherwise difficult to assess.

The primary models that were assessed in the current study are outlined in Fig. 1. By fit indices of these models and examining modifications of these models, a wide range of theoretical proposals can be assessed and compared to one another.

A *single-process model* of recall and recognition is presented in Fig. 1A. Observed variables *recall* and *recognition*

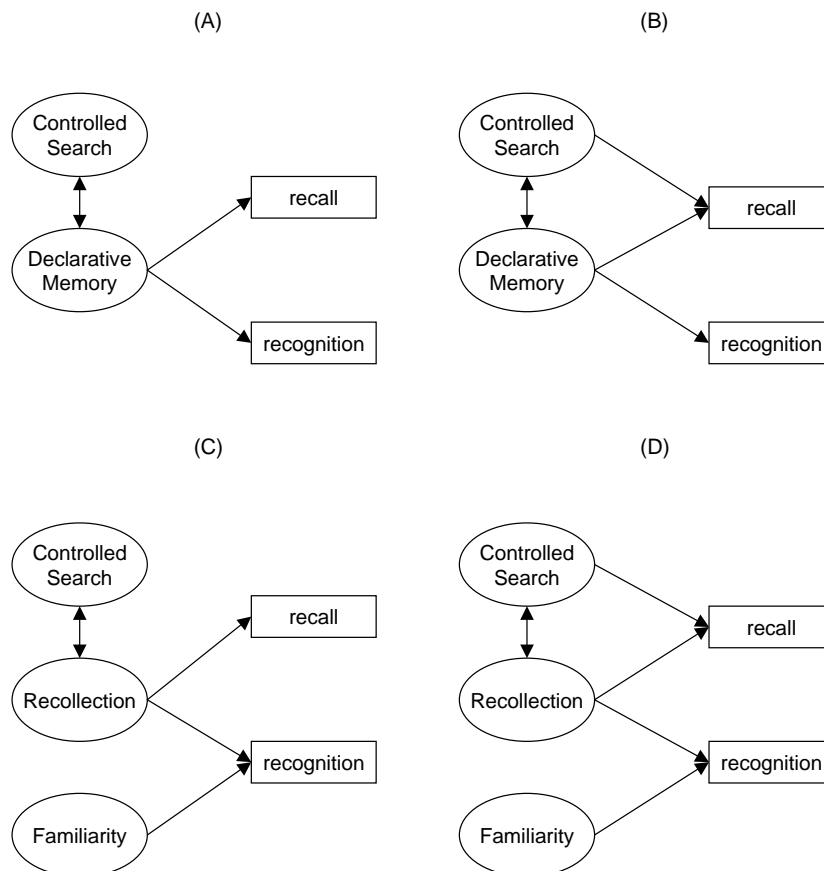


Fig. 1. Four latent factor models of processes underlying recall and recognition: a single-process model (A), a dual-process recall model (B), a dual-process recognition model (C), and a hybrid three-process model (D).

are represented as rectangles. Theoretical constructs or *latent factors* are represented using ellipses. In this model, *declarative memory* is a latent factor that contributes variance to recall and recognition. The connections between variables represent expected covariation and are each associated with a correlation (bi-directional arrow between factors), a regression weight (uni-directional arrow between factors) or a loading (an arrow from a factor to a variable). In the single-process model and all subsequent models, a *controlled search* component was included to account for the finding of a relationship between performance on declarative memory tests and performance on non-memory tests involving strategic and general search abilities. For example, performance on tests of verbal fluency, in which subjects are required to generate words from specified categories, is found to correlate with free recall performance (e.g. Janowsky et al., 1989a; Milner et al., 1985; Parkin & Lawrence, 1994; Parkin et al., 1995). The controlled search process indexed by this task is not specific to declarative memory tests, and thus reflects a component that is at least partially distinct from declarative memory. Thus, despite the inclusion of the controlled search component, the model in Fig. 1A is still considered a single-process memory model because only one memory factor contributes directly to memory performance. That is, because there is a single memory component, anything affecting declarative memory will influence recall and recognition equivalently.

Note that directional arrowheads were included to indicate the likely causal direction underlying the covariance between the factors. For example, variability in declarative memory ability across individuals is assumed to cause variability in overt recognition performance. Uni-directional paths are always shown from latent factors to observed variables and indicate factor loadings of the variable on the factor. However, for connections among factors, the causal direction was ambiguous in one case, so bi-directional arrows were used. A bi-directional arrow was included between controlled search and declarative memory because causality was expected to go both directions. For example, some controlled search processes may influence declarative memory retrieval, whereas other processes may be involved in post-retrieval stages, such as response monitoring. Model fits, loadings and weights can change depending on the assumed causal direction. However, assuming a uni-directional causal relationship from controlled search to declarative memory, and vice versa, did not noticeably alter the loadings and weights in any of the four models of interest.

As discussed earlier, complex dissociations observed between recall and recognition are inconsistent with a simple single-process memory model. Additionally, Nyberg (1994) found that a single-factor model did not provide an adequate account of recall and recognition performance of normal healthy individuals. However, we included this model for two reasons. First, a single-factor model has never been assessed directly for the patient population of interest. Second, it provides a theoretical baseline from which to compare

more complex models. If more complex models are superior to a single-process model, then they should not only provide a statistically acceptable account of the data, but should provide a significant improvement over the simpler model.

A *dual-process model of recall* is presented in Fig. 1B. In accord with several dual-process theories of recall (e.g. Moscovitch, 1994; Shimamura, 1995; Squire & Zola, 1998), declarative memory is assumed to contribute variance to recall and recognition, whereas additional controlled search processes contribute directly to recall. The model is identical to the single-process model except that the controlled search factor directly influences recall. This allows the model to account for additional search and response monitoring demands of recall beyond what is shared by recognition. By contrasting the fit of this model to the single-process model, one can test whether the addition of a selective influence of controlled search in recall statistically improves the fit of the model to the empirical data.

A *dual-process recognition model* is presented in Fig. 1C. In accord with dual-process theories of recognition (Atkinson & Juola, 1974; Jacoby & Dallas, 1981; Mandler, 1980; Yonelinas, 1994), two memory factors, *recollection* and *familiarity*, are assumed to contribute to recognition, whereas only recollection contributes to recall. Note that no path is drawn between familiarity and recollection because they are assumed to be uncorrelated. This convention reflects the assumption of most dual-process models of recognition that the two processes make independent contributions to recognition (e.g. Jacoby & Dallas, 1981; Mandler, 1980; Yonelinas, 1994). Importantly, this model is identical to the single-process model except that an additional factor contributes to recognition. Thus, a statistical comparison of the fits of these two models will test whether the addition of the familiarity factor to the model results in a significant improvement in fit. A version of this model was reported previously by Yonelinas et al. (2002), but no comparison was made in that paper to the alternative models presented here.

The two dual-process models are equally complex because both contain one more free parameter (i.e., connection) than the single-process model. Unfortunately, it is difficult to directly compare the fits of the two models because they are not nested. To overcome this problem we included a hybrid model (Fig. 1D). This model, which assumes that two processes contribute to recall and two processes that contribute to recognition, is the most complex of the four and contains all free parameters of the two dual-process models. Importantly, the hybrid can be compared statistically to the other three by fixing different parameters to zero (i.e., by removing connections). Comparing the fits of the hybrid and dual-process recall models tests whether adding a familiarity process to the dual-process recall model would improve fit. Similarly, comparing the fits of the hybrid and the dual-process recognition model tests whether by adding a direct contribution of controlled search to recall improves the dual-process recognition model. Finally, the hybrid model itself is of interest because dual-process assumptions

for recall and recognition are not mutually exclusive, and an improvement over both dual-process models would indicate both types of assumptions are warranted.

The models were assessed by fitting competing structural equation models to the data from a previous study of recall and recognition in a group of 56 cardiac-arrest patients (Sauvé, Doolittle, Walker, Paul, & Scheinmann, 1996). These patients suffered variable degrees of explicit memory impairment caused by hypoxia, but were otherwise cognitively intact. Because the patients had defibrillators, they could not be scanned. However, they were expected to have relatively selective hippocampal damage. Although hypoxia may lead to severe hippocampal atrophy, other brain regions including the thalamus and the watershed regions in the cerebral cortex and cerebellum can also be influenced (Caine & Watson, 2000; Markowitsch, Weber-Luxemburger, Ewald, Kessler, & Heiss, 1997; Schmidt-Kastner & Freund, 1991). However, in cases in which the hypoxic event is brief and the cognitive deficits are restricted primarily to memory, volumetric neuroimaging (Kartsounis, Rudge, & Stevens, 1995; Vargha-Khadem et al., 1997) and post-mortem neuropathological analysis (Cummings, Tomiyasu, Read, & Benson, 1984; Rempel-Clower, Zola, Squire, & Amaral, 1996) indicate that the hippocampus is the primary region influenced by hypoxia and is the most likely cause of the memory impairments. In the current study, the duration of cardiac arrest in the hypoxic group was on average 2.1 min and the average coma duration was 16 h. Both figures are much lower than those of hypoxics in studies where damage was extensive and multiple deficits were found (e.g. Markowitsch et al., 1997; Parkin, Miller, & Vincent, 1987; Volpe & Petito, 1985).

The hypoxic group in the present study was deemed particularly useful for testing theories of explicit memory because age and coma duration were available for each patient as predictors of memory components. Previous studies have indicated that coma duration directly correlates with the severity of subsequent memory impairments (Sauvé & Walker, 1997). Thus, using coma duration, we could evaluate the effects of hypoxic severity on the underlying memory components of the different models. The age of each patient was also available, allowing us to assess the effects of this variable on the different memory components. Moreover, verbal fluency scores were also available and were used as indicators of general controlled search processes that may influence declarative memory. Finally, repeated measures of the recall, recognition, and verbal fluency were available, allowing us to impose additional statistical constraints on the models.

2. Method

2.1. Participants

Data from 56 survivors of sudden cardiac arrest (30 males) were examined (for earlier reports on subsets of the

current data set see Sauvé & Walker, 1997; Sauvé et al., 1996; Yonelinas et al., 2002). Patients were recruited from hospitals in the San Francisco Bay area and were assessed four times during the first 6 months following successful resuscitation. Patients who were in comas for more 72 h after cardiac arrest were eliminated from study at the initial referral because of a low expected survival rate. The patients were between 35 and 78 years old (mean = 61.80; S.D. = 11.77) and estimates of WAIS IQ were within the normal range (IQ range 80–145; mean = 106.91; S.D. = 14.89).

Individuals were not selected based on the severity of a memory deficit (i.e., the dependent variable), as is usually the case in the study of memory in brain-injured patients. Rather, individuals were selected for participation because they had suffered a hypoxic event (i.e., selected according to the causal variable). The data were originally collected in an effort to determine the recovery of cognitive functions after a cardiac arrest, so presence or absence of a memory deficit was not considered prior to inclusion in the study. As a consequence, not all patients exhibited deficits relative to the sample of healthy controls. However, selecting only those individuals with severe memory deficits would exclude portions of the hypoxic population who may have mild brain injury or relatively high pre-morbid functioning. Moreover, it would have limited the range of observed memory impairments and thus reduced our ability to assess the effects of hypoxic severity on memory.

To confirm that the hypoxic group did have a memory impairment despite not having been selected for this reason, recall and recognition scores of 89 control subjects from Sauvé et al. were also used (Sauvé & Walker, 1997). Controls from this data set were selected if they had complete repeated recall and recognition scores on the same tests as the hypoxics and if their ages fell within the same range as that of the hypoxic sample were used. The average age of the control group did not differ from that of the hypoxic group, $t(143) < 1$. The controls had no history of cardiac or neuropsychological disease.

2.2. Procedure

A battery of behavioral tests was administered during four testing sessions over a 6-month period following successful CPR. However, only test scores from the third (12–15 weeks) and fourth (22–25 weeks) testing sessions were included in the present analysis, because a high degree of variation was observed in the impairment of performance on measures of orientation, attention, reasoning, and motor function during the first two testing sessions (through 9 weeks). Including measurements from the third and fourth testing sessions in the present analysis allowed us to assess the ability of the structural equation models to explain variance in verbal memory performance at a point where cognitive performance had approximately stabilized. For ease of reference, we will use the terms time one and time

Table 1
Descriptive statistics for each of the observed variables

Variable	Mean	S.D.	Range
Coma	14.91	17.06	0.0–65.0
Age	61.80	11.77	35–78
Fluency 1	40.84	12.50	19–67
Fluency 2	41.55	12.33	20–67
Recall 1	6.59	3.79	0–15
Recall 2	7.79	3.71	0–15
Recognition 1	11.59	3.48	2–15
H	12.89	2.20	5–15
FA	1.30	2.11	0–9
Recognition 2	12.45	3.16	2–15
H	13.39	2.22	5–15
FA	0.95	1.92	0–9

Note: Coma: coma duration; age: age at cardiac arrest; fluency 1: FAS verbal fluency at 3 months; fluency 2: FAS verbal fluency at 6 months; recall 1: RAVLT delayed recall at 3 months; recall 2: RAVLT delayed recall at 6 months; recognition 1: RAVLT delayed recognition at 3 months; recognition 2: RAVLT delayed recognition at 6 months; H: recognition hits; FA: recognition false alarms.

two for testing sessions three and four, respectively, in the remainder of this paper.

Eight memory-related variables from the Sauvé et al. data set were examined in the current study. Descriptive statistics for all eight measures are shown in Table 1. The first two variables were coma duration and the age of the subject measured at the time of cardiac arrest. Coma duration was used to represent hypoxic severity as an indirect measure of brain injury. Coma duration, operationalized as the number of hours from collapse to comprehensible speech or ability to follow verbal commands (Sauvé et al., 1996), is a major correlate of post-morbid level of cognitive function, and particularly, of post-morbid memory ability (Levy et al., 1985; Roine, Kajaste, & Kaste, 1993). These data were taken from emergency and paramedic reports, when available, or from the referring physician.

The Rey Adult Verbal Learning Test (RAVLT) was used to assess delayed recall and recognition (Sauvé et al., 1996). The RAVLT measures recall and recognition memory for the same materials after comparable delay periods. The RAVLT also provides a number of immediate recall measures as well, but delay performance was selected as the variable of interest because it is generally more sensitive to hippocampal damage than immediate recall (Manns et al., 2003; Squire & Zola-Morgan, 1991), and is the most consistently observed of all cognitive sequelae among cardiac-arrest survivors (Roine et al., 1993; Sauvé & Walker, 1997).

The RAVLT consists of five consecutive study-recall blocks with a 15-word list (immediate recall), followed by one additional study-recall session with a different 15-word list (distractor recall). After the recall test for the second list, a sixth recall trial was given for the original list (early recall). After 20 min, delayed retention of the original list was assessed first by a final recall test (delayed recall), and then by a recognition test which included all 15 original words and 15 distractors. The RAVLT was administered four times

over a 6-month period. To reduce practice effects, different lists of words were used for each of the four administrations. All recall measures were scored as the total number of original words recalled on the test, so scores had a potential range from 0 to 15. Scores for recognition were computed by subtracting the number of false alarms (incorrect “yes” responses) from the number of hits, resulting in scores with a possible range from –15 to 15, with scores of 0 or less indicating performance at or below chance. This was done so that recall and recognition scores would have the same values for maximum (15) and chance (0) levels of performance, thus enabling an easier interpretation of path coefficients.

Verbal fluency was measured using the Controlled Oral Word Association test (Bentler & Bonnett, 1980). In this test, participants are given single letters and are required to generate as many words as possible starting with each letter. This test is often referred to as the FAS test (e.g. Baldo et al., 2001; Parkin & Walter, 1992) because two trials each are given with the letters F, A, and S. Performance is scored as the number of words generated in 60 s and averaged across the six trials. The FAS task is particularly useful in the current study because it measures general controlled search abilities using a paradigm similar to standard recall in that it involves the organization of a memory search, generation of appropriate verbal responses, and inhibition of irrelevant and redundant responses. Moreover, it is sensitive to frontal lobe damage (Baldo et al., 2001; Bentler & Bonnett, 1980; Shimamura, 1995) and is moderately correlated with declarative memory (Parkin & Walter, 1992).

2.3. Model fitting methods

Structural equation models were fit to the 36 elements of the variance–covariance matrix of the eight observed variables (age, coma duration, recall 1, recall 2, recognition 1, recognition 2, fluency 1, fluency 2). The correlations among the eight observed variables are presented in Table 2, and the standard deviations for each variable are presented in Table 1. Model fitting was performed using maximum likelihood estimation in LISREL 8.0 (Jöreskog & Sörbom, 1999) to reproduce the observed covariance matrix as closely as possible given the constraints of each model. The statistical goodness-of-fit for each model was assessed using χ^2 with $36 - k$ degrees of freedom, where k was the number of parameters estimated in the model. A significant χ^2 for any model indicated that it did not provide an adequate account of the covariance matrix.

Because the χ^2 index of statistical fit is dependent on sample size, four practical fit indices that are relatively independent of sample size were also used to assess model fit. These four indices included the root mean square error of approximation (RMSEA), the comparative fit index (CFI), the non-normed fit index (NNFI), and the consistent version of Akaike’s (1987) information criterion (CAIC; Bozdogan, 1987, 2000). All fit indices are reported for all tested models.

Table 2
Correlation matrix of observed variables

	Coma	Age	Recall 1	Recall 2	Recognition 1	Recognition 2	Fluency 1	Fluency 2
Coma	–							
Age	–0.18	–						
Recall 1	–0.25	–0.32*	–					
Recall 2	–0.45**	–0.25	0.81**	–				
Recognition 1	–0.20	–0.17	0.71**	0.61**	–			
Recognition 2	–0.30*	–0.29*	0.65**	0.71**	0.77**	–		
Fluency 1	–0.08	–0.22	0.32*	0.42**	0.21	0.38**	–	
Fluency 2	–0.08	–0.13	0.27*	0.37**	0.27*	0.40**	0.83**	–

Note: Coma: coma duration; age: age at cardiac arrest; recall 1: RAVLT delayed recall at 3 months; recall 2: RAVLT delayed recall at 6 months; recognition 1: RAVLT delayed recognition at 3 months; recognition 3: RAVLT delayed recognition at 6 months; fluency 1: FAS verbal fluency at 3 months; fluency 2: FAS verbal fluency at 6 months.

* $P < 0.05$.

** $P < 0.01$.

The RMSEA and corresponding probability assess how well the model accounts for the data given that the sample covariance matrix is only an approximation of the population covariance matrix (Browne & Cudeck, 1992). Whereas the χ^2 is a test of exact model fit, the RMSEA enables a test of close fit. Models associated with significant RMSEAs (i.e., $P_{\text{close}} < 0.05$) can be rejected because they do not provide a sufficiently close fit to the unknown population covariance matrix even when error of approximation is accounted for. RMSEA values of desirable models should fall no higher than 0.10, and preferably no higher than 0.05, regardless of the corresponding P_{close} (MacCallum, Browne, & Sugawara, 1996).

The CFI (Bentler, 1990; Tucker & Lewis, 1973) and NNFI (Bentler & Bonnett, 1980; McDonald & Marsh, 1990) assess the fit of the model relative to an independence model in which all covariances among observed variables are zero. Both the CFI and NNFI are fit indices that, generally, range from 0 to 1, with an acceptable fit indicated by a value above 0.90 and preferably above 0.95. The CFI represents the proportion of explainable covariance among observed variables explained by a model. The NNFI has a similar interpretation, but contains a correction for the complexity of the model (i.e., the number of estimated parameters). CFI values are constrained to range from 0 to 1. The NNFI also generally ranges between 0 and 1, but can assume values greater than 1.0 if the χ^2 for a model is less than its degrees of freedom. NNFI values above 1.0 indicate some overfitting, but are not serious if values do not range far above 1.0.

Finally, the consistent version of Akaike's (1987) information criterion (CAIC) is a fit index that corrects for the number of parameters in a model and is a consistent index regardless of sample size (Bozdogan, 1987). The CAIC is useful for comparing non-nested models and penalizes model fits for relative lack of parsimony and for relatively small sample sizes. For any two models with the same degrees of freedom, the one associated with the lower CAIC fits best. For any two models with comparable fit indices (e.g., RMSEA, χ^2 , CFI), the one with the lower CAIC is the more preferred.

Statistical comparisons between nested models were performed by testing the significance of the difference in χ^2 , or $\Delta\chi^2$, using degrees of freedom equal to difference in the number of free parameters in the two models. In addition to fitting the a priori models in Fig. 1, a number of nested submodels were also tested to determine whether critical assumptions about model parameters were justified. For example, if the fit of an a priori model could be improved by freeing a parameter inconsistent with the theoretical basis for the model, then the parameter constraint in the a priori model is unreasonable. Likewise, if an a priori model does not fit better than a simpler model in which a key parameter is fixed, the parameter is unnecessary.

3. Results

3.1. Preliminary analysis: recall and recognition performance of hypoxics

To confirm that the hypoxic group had a memory deficit, the average immediate, distractor, early and delayed recall scores from the RAVLT are presented in Table 3 for time 1

Table 3
RAVLT memory scores for hypoxics and healthy controls

Test	Controls ($N = 89$) Mean (S.D.)	Hypoxics ($N = 56$) Mean (S.D.)
Immediate recall 1	9.87 (2.09)	7.69 (2.27)**
Distractor recall 1	5.85 (2.55)	4.64 (1.84)**
Early recall 1	10.00 (2.93)	7.29 (3.31)**
Delayed recall 1	10.13 (3.26)	6.59 (3.79)**
Delayed recognition 1	13.94 (1.55)	11.59 (3.48)**
Immediate recall 2	9.88 (2.12)	8.72 (2.49)**
Distractor recall 2	5.87 (2.27)	4.98 (2.14)*
Early recall 2	10.42 (3.06)	8.39 (3.47)**
Delayed recall 2	10.16 (3.24)	7.79 (3.71)**
Delayed recognition 2	13.43 (2.34)	12.45 (3.16)*

* Hypoxic mean reliably different than control mean, $P < 0.05$.

** Hypoxic mean reliably different than control mean, $P < 0.01$.

and time 2 along with scores for healthy controls. On recall, the hypoxic scores ranged from +1.49S.D. to -3.11S.D. relative to the control mean for time 1 ($M = -1.09$) and from +1.49S.D. to -3.14S.D. relative to the control mean for time 2 ($M = -0.73$). On recognition, the hypoxic scores ranged from +0.68S.D. to -7.70S.D. relative to the control mean for time 1 ($M = -1.51$) and from +0.67S.D. to -4.88S.D. relative to the control mean for time 2 ($M = -0.42$).

t-tests for independent groups assuming unequal variances were used to determine whether the hypoxic group was significantly impaired relative to the control group on each of the tests. As can be seen in Table 3, the hypoxic group was significantly impaired, at both testing times, on all measures of recall and recognition relative to the control scores. Thus, the preliminary analysis confirmed that the hypoxic group did, on average, show reliably poorer memory performance than the controls, despite not having been selected for study because they had memory deficits.

For the hypoxic group, the immediate recall scores correlated highly with the delayed scores ($r = 0.82$, $P < 0.001$ for both time 1 and time 2). Relative to the delayed recall correlations in Table 3, immediate recall correlated somewhat more strongly with age ($r = -0.49$, $P < 0.001$ for time 1 and $r = -0.38$, $P = 0.004$ time 2) and somewhat less strongly with coma duration scores ($r = -0.23$, $P = 0.08$ for time 1 and $r = -0.35$, $P = 0.008$ for time 2). Although we considered delayed recall to be of primary importance on an a priori basis, this analysis suggests that the choice to use delayed recall in the structural equation models over the more frequently reported immediate recall makes little difference. In fact, when immediate scores were substituted for delayed scores in the proceeding models, the results were highly similar.

3.2. The single-process model

The path diagram of the single-process model is shown in Fig. 2. The model reflects the hypothesis that one latent factor (i.e., declarative memory), underlies performance on recall and recognition. Declarative memory is in turn influenced by the age of the individual and by coma duration. Note that coma duration and age were treated as perfect indicators of latent factors with the same name. This was done only to allow observed variables coma duration and age to predict latent factors in the structural equation model, and is not meant to indicate that these variables are measured perfectly. A controlled search factor is assumed to underlie performance on tests of verbal fluency, and is correlated with age and declarative memory.

Constraints were imposed so that members of each pair of observed tests (recall, recognition, and fluency) loaded equally on their respective factors and had equal residual variances. For example, recall 1 and recall 2 had identical loadings on declarative memory. The inclusion of this constraint requires the pairs of tests be essentially parallel tests (see Jöreskog, 1974; Lord & Novick, 1986) meaning that the tests must be reliable and consistent indicators of their underlying factors.

An additional factor (i.e., recovery) was assumed to add variance to all three tests at time one. This factor was included in the model, and all subsequent models, because test scores at time one were more variable and exhibited somewhat higher covariance than at time two. This may not be readily apparent from the correlations in Table 2, but the covariance of any two variables can be derived by multiplying the correlation by the product of the two variables' standard deviations in Table 1. This shows that recall and recognition

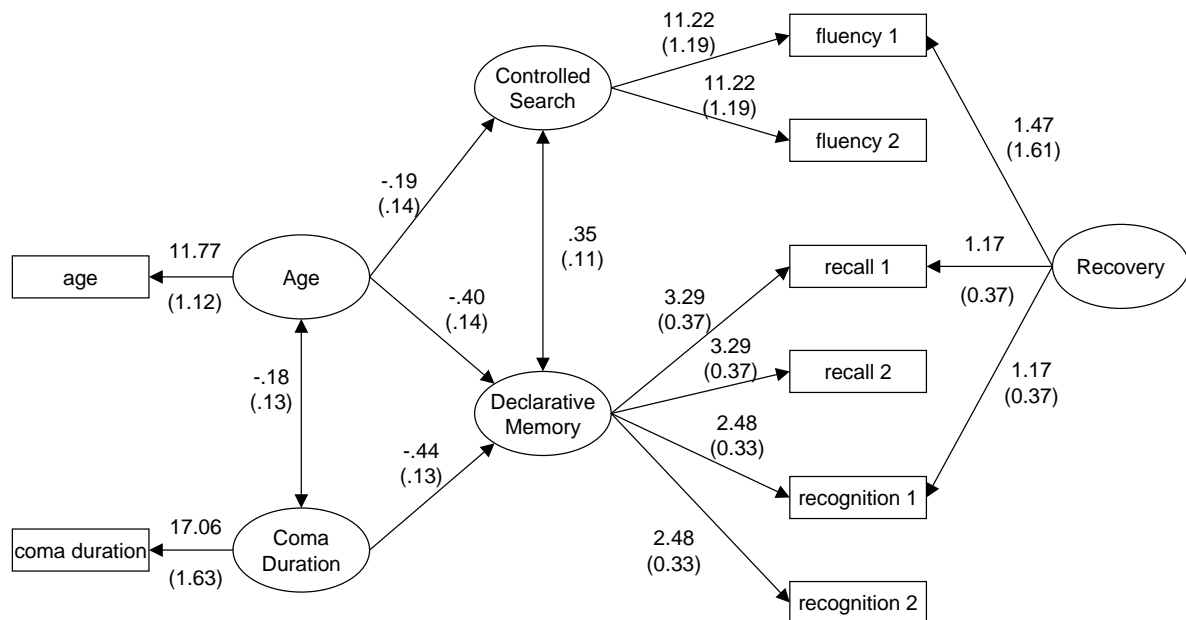


Fig. 2. Path diagram of the single-process model in which recall and recognition load on a single declarative memory factor. Parameter estimates are shown for the standardized solution with standard errors in parentheses.

share more raw variance at time one (9.36) than at time two (8.32). This may be because at the initial test (i.e., 3 months after the injury), general cognitive abilities had not yet completely recovered in all patients. Both recall and recognition were improved significantly at time 2. Removing this factor does not change the overall pattern of conclusions, but it does decrease the goodness-of-fit for all the models. Because recall and recognition were measured on roughly the same scale, and in keeping with the parallel tests assumption noted above, they were allowed to load equally on recovery, whereas a separate loading was required for fluency 1 variable. Allowing different recovery weights for each test did not alter the outcome of the models. The current model also assumes that recovery was not influenced by age or coma duration (i.e., there are no direct connections between recovery and either of these other factors).

The total variance of each latent factor in the model was fixed at 1.00 for convenience in interpreting the loadings and regression weights. Thus, paths denoting regression weights (denoted by β in text) and correlations between factors (ϕ in text) are presented in a standardized (i.e., correlation) metric. The variances of observed variables were analyzed in the same metric presented in Table 1. Thus, loadings of observed variables on factors (λ in text) represent the increase in raw-score performance associated with a 1S.D. increase in the factor score.

The best-fitting standardized parameter estimates for the single-process model are shown in Fig. 2, and Table 4 shows the fit statistics for the model along with those for three modifications to the model. The fit of the single-process model was rejectable statistically, $\chi^2(21, N = 56) = 38.51$, $P = 0.011$, and the measures of practical fit were marginal, with CFI = 0.928 and NNFI = 0.905. Thus, the single-process model did not provide an adequate account of performance.

Three modifications were assessed to determine whether the fit of the model would improve if some of the parameter restrictions were removed. First, to determine whether the equality constraints were justified (i.e., that the behavioral tests at times one and two were parallel tests), the equality constraints on factor loadings and on residual variances were relaxed (modification I). This change did not improve the fit of the model, $\Delta\chi^2(7, N = 56) = 4.07$, $P = 0.77$, indicating that the equality constraints had not compromised the model fit. A second modification was assessed in which both age and coma duration were allowed to influence recovery (modification II). These changes did not improve the fit of the model, $\Delta\chi^2(2, N = 56) = 4.10$, $P = 0.13$. A third modification was tested to determine if coma duration predicted Controlled Search (modification III). Adding a path from coma duration to controlled search did not improve the fit of the model, $\Delta\chi^2 < 1$. In summary, the single-process model was insufficient to account for the covariance matrix. Additional tests of specific parameter restrictions indicated lack of fit was not a result of the potentially restrictive assumptions that (1) behavioral tests at time one and two were parallel, (2) that recovery rate was independent of age and coma duration, or (3) that coma duration did not affect controlled search.

3.3. The dual-process recall model

To derive the dual-process recall model one change was made to the single-process model. The controlled search factor was allowed to have a direct effect on recall that was separate from any connection to recognition. The path model along with the best-fitting parameter estimates for the model are shown in Fig. 3, and Table 4 shows the fit statistics for the model along with those for two modifications. The fit of the dual-process recall model was statistically rejectable, $\chi^2(20, N = 56) = 38.18$, $P = 0.008$, and the practical fit

Table 4
Summary of fit indices for the single-process, dual-process recall, dual-process recognition, and hybrid models

Model	Statistical fit indices					Practical fit indices		
	χ^2	d.f.	P_{exact}	RMSEA	P_{close}	CAIC	NNFI	CFI
Single-process model	38.51	21	0.011	0.111	0.071	110.67	0.905	0.928
Modification I	34.44	14	0.002	0.144	0.023	140.51	0.833	0.916
Modification II	34.41	19	0.017	0.105	0.106	116.01	0.907	0.937
Modification III	37.82	20	0.009	0.114	0.067	114.62	0.898	0.927
Dual-process recall model	38.18	20	0.008	0.115	0.061	115.07	0.896	0.926
Modification I	45.57	21	0.001	0.133	0.020	116.66	0.866	0.900
Modification II	37.50	19	0.007	0.118	0.058	118.96	0.888	0.924
Dual-process recognition model	16.22	20	0.703	0.000	0.827	96.46	1.022	1.000
Modification I	16.08	19	0.652	0.000	0.783	101.42	1.018	1.000
Modification II	16.08	19	0.652	0.000	0.783	101.42	1.018	1.000
Modification III	16.18	18	0.550	0.000	0.722	106.55	1.012	1.000
Hybrid three-process model	15.60	19	0.684	0.000	0.806	100.94	1.021	1.000

Note: d.f.: degrees of freedom for χ^2 -test; P_{exact} : probability of exact fit (χ^2 at d.f.); RMSEA: root mean square error of approximation; P_{close} : probability of close fit (RMSEA); CAIC: consistent Akaike's (1987) information criterion; NNFI: Bentler and Bonett's (Bentler & Bonnett, 1980) non-normed fit index; CFI: Bentler's (1990) comparative fit index. A priori models are shown in bold.

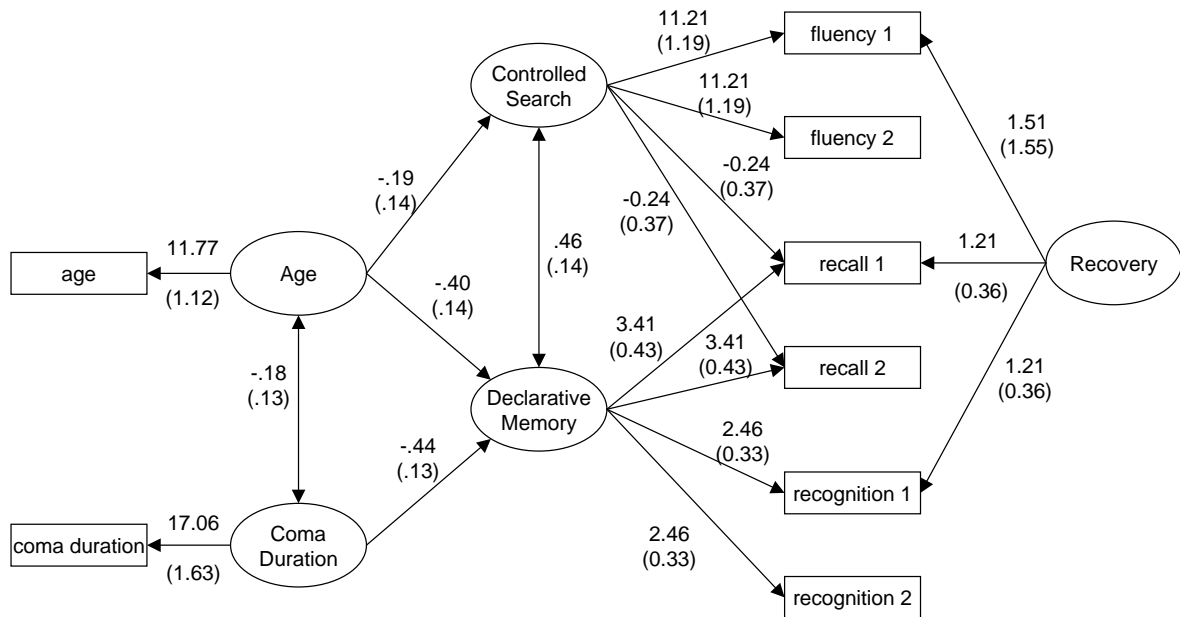


Fig. 3. Path diagram of the dual-process recall model in which a recall loads on declarative memory and controlled search factors whereas recognition loads only on the declarative memory factor. Parameter estimates are shown for the standardized solution with standard errors in parentheses.

indices were of borderline acceptability, with CFI = 0.896, NNFI = 0.926. Moreover, the model did not represent a significant improvement over the single-process model, $\Delta\chi^2(1, N = 56) = 0.33, P = 0.57$. Thus, the model did not provide an adequate account of the data, nor did it reflect an improvement over the simpler model.

Additionally, the contribution of the controlled search factor to recall was not significant ($\lambda = -0.24, z = -0.64, P = 0.52$),¹ even though the correlation between controlled search and declarative memory was significantly positive ($\phi = 0.46, z = 3.33, P = 0.002$). This suggests that recall does not reflect an additional contribution of controlled search processes that are not also shared by recognition, inasmuch as verbal fluency provides a measure of these processes.

A modification of the model was assessed in which the correlation between controlled search and declarative memory was removed but the contribution of controlled search to recall was retained (modification I). This modification led to a significant worsening of fit, $\Delta\chi^2(1, N = 56) = 7.39, P = 0.007$, and the direct effect of controlled search on recall still did not deviate significantly from zero ($\lambda = 0.33, z = 1.21, P = 0.23$). This verifies that any variance shared by verbal fluency and recall was also shared by recognition.

A second modification was tested to determine whether it was reasonable to assume that coma duration did not predict

controlled search (modification II). Adding a path from coma duration to controlled search did not significantly improve the fit of the model, $\Delta\chi^2(1, N = 56) = 0.68, P = 0.41$, and the regression weight itself was not significant ($\beta = -0.14, z = -0.94, P = 0.34$), indicating that coma duration did not predict controlled search.

In summary, a dual-process model of recall did not provide a significant improvement in fit over the single-process model, and the model was statistically rejectable, indicating that the dual-process recall model failed to account for the covariance matrix.

3.4. The dual-process recognition model

To derive the dual-process recognition model one change was made to the single-process model. A familiarity factor was added that influenced only the recognition tests (see Fig. 4). Note that to meet the terminological convention of dual-process theories of recognition, the memory factor underlying both recall and recognition (i.e., declarative memory in the single-process model) was renamed recollection. As was the case with the declarative memory factor in the two previous models, recollection is assumed to be influenced by age and coma duration and, in turn, was assumed to affect recall and recognition. However, in agreement with the dual-process recognition theories discussed earlier, age and coma duration are not expected to affect Familiarity, and there is no direct connection between recollection and familiarity.

The best-fitting standardized parameter estimates for the model are shown in Fig. 4, and Table 4 shows the fit statistics for the model along with those for three modifications. The fit of the model was not rejectable, $\chi^2(20, N = 56)$

¹ Two-tailed z -tests are used to determine whether individual parameter values deviate significantly from zero. Like t -tests used in standard regression, z -values are obtained by dividing the parameter value by its standard error. Thus, standard errors can be obtained for any model parameter discussed in the text where a z -test is reported, and z -tests can be performed for any parameter in model figures where the standard error is reported.

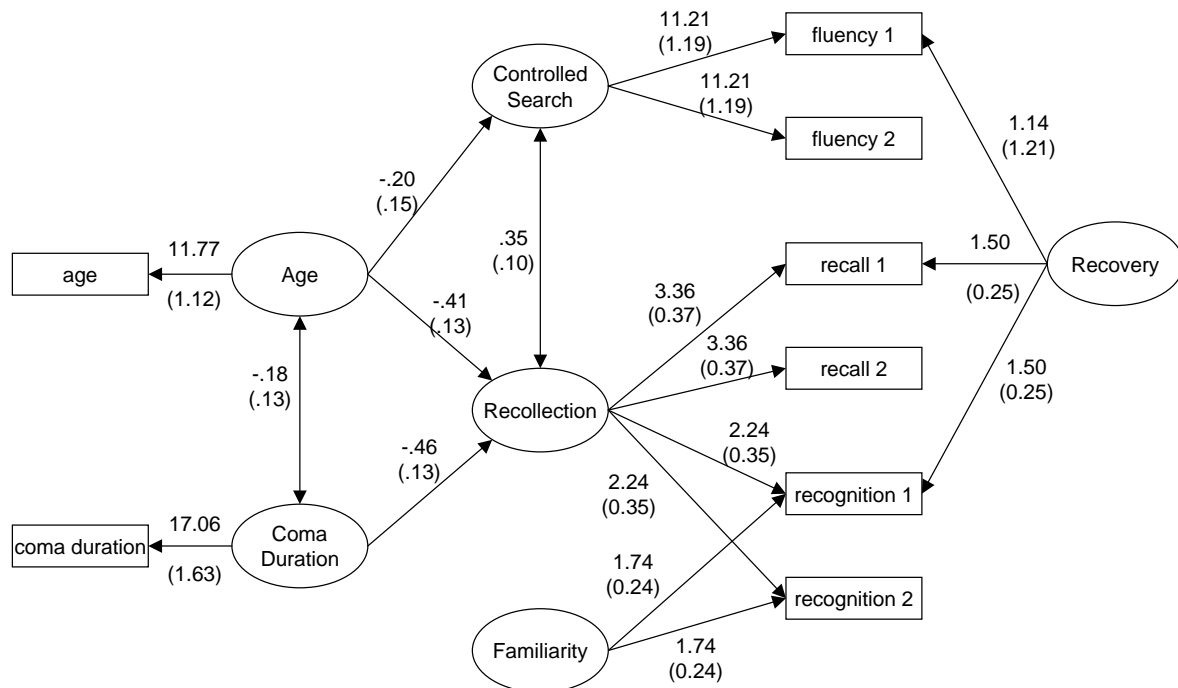


Fig. 4. Path diagram of the dual-process recognition model in which recognition loads on recollection and familiarity factors whereas recall loads only the recollection factor. Parameter estimates are shown for the standardized solution with standard errors in parentheses.

= 16.22, $P = 0.70$, and the practical fit indices were quite good, with CFI = 1.0 and NNFI = 1.022. Importantly, the model represented a significant improvement in statistical fit over that of the single-process model, $\Delta\chi^2(1, N = 56) = 22.29$, $P < 0.001$, and levels of all practical fit indices for the dual-process recognition model were also uniformly better. Thus, freeing one parameter to allow influence of an independent familiarity process to recognition tests substantially improved the fit of the single-process model.

Modifications of the dual-process recognition model were examined to assess three critical assumptions. First, recollection and familiarity were assumed to be independent in the present model (see Jacoby, Yonelinas, & Jennings, 1995, for the rationale of the independence assumption). However, others have argued that these processes are better described as exclusive (e.g. Gardiner & Java, 1991) or redundant (e.g. Joordens & Merikle, 1993). To test the related assumption that these processes are independent across subjects, a path was added between recollection and familiarity and the model was refit (modification I). This modification, however, did not lead to a significant improvement in fit, $\chi^2(1, N = 56) = 0.14$, $P = 0.71$, and the fitted factor correlation ($\phi = -0.07$) did not differ significantly from zero, $z = -0.37$, $P = 0.71$. Thus, recollection and familiarity appear to be best described as independent factors across individuals.

The second critical assumption of the dual-process recognition model in Fig. 4 is that Familiarity contributes only to recognition, whereas Recollection contributes to both recall and recognition. The absence of a familiarity contribution to free recall has been often assumed (e.g. Mandler, 1980),

but never demonstrated directly. To evaluate the tenability of this assumption, a path was added from familiarity to recall (modification II). Thus, both recollection and familiarity were allowed to affect both recall and recognition. No significant improvement in fit was associated with this modification, $\Delta\chi^2(1, N = 56) < 1$, indicating that familiarity did not contribute significantly to recall performance. Moreover, based on the best-fitting parameters for the model, Recollection significantly influenced recognition ($\lambda = 2.43$; $z = 5.27$, $P < 0.001$) and recall ($\lambda = 3.35$; $z = 9.13$, $P < 0.001$). In contrast, familiarity influenced recognition ($\lambda = 1.58$, $z = 3.16$, $P < 0.001$), but not recall ($\lambda = -0.24$, $z = -0.37$, $P = 0.71$). These effects are illustrated in Fig. 5, which presents the unstandardized factor loadings of recall and recognition tests on recollection and familiarity presented as probabilities. The loadings are interpreted as regression slopes reflecting the predicted change in the probability of above-chance recall or recognition for every one standard deviation increase in recollection or familiarity. An examination of Fig. 5 reveals that a one standard deviation increase in recollection predicts an increase in the probability of both recall and recognition. By contrast, the same increase in familiarity predicts an increase in the probability of recognition, but no noticeable change in recall.

The dual-process recognition model in Fig. 4 assumes that age and coma duration influence recollection, but not familiarity. To determine whether these assumptions were reasonable, age and coma duration were allowed to influence both recollection and familiarity (modification III). However, no significant improvement in fit was gained, $\Delta\chi^2(2, N = 56)$

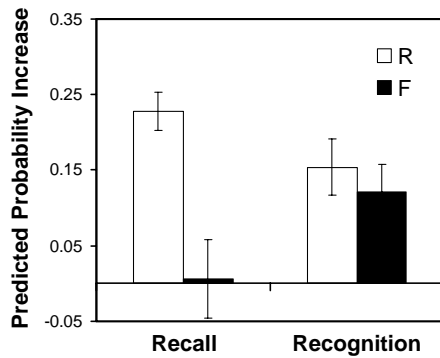


Fig. 5. Estimated contributions of recollection and familiarity to recall and recognition performance. Plot shows model-estimated increase in recall and recognition probabilities with every 1S.D. increase in recollection or familiarity. R: recollection; F: familiarity.

= 0.004, $P = 0.98$, and neither of the path coefficients from age or coma duration to familiarity were significantly greater than zero ($\beta = 0.00$, $z = 0.021$, $P = 0.98$ and $\beta = 0.04$, $z = 0.20$, $P = 0.84$, respectively). In contrast, path coefficients from Age and Coma Duration to Recollection were significantly greater than zero ($\beta = -0.41$, $z = -3.03$, $P = 0.002$ and $\beta = -0.47$, $z = -3.55$, $P < 0.001$, respectively).

These latter findings involving coma duration and age are illustrated in Fig. 6A and B. Fig. 6A presents the estimated effects of hypoxia on recollection and familiarity. Estimates of recollection and familiarity are plotted as z -scores at two levels of hypoxic severity. The z -scores are scaled such that performance at a coma duration of zero hours represents a recollection or familiarity score of zero. Zero hours was just less than 1S.D. below the mean coma duration and is intended to represent the expected level of performance of healthy controls. Recollection and familiarity estimates are plotted for the mean coma duration of 15 h and for 1S.D. above the mean at 32 h. Estimates were derived using the regression coefficients in modification III that linked coma duration to the recollection and familiarity factors. The figure shows that hypoxic patients who were in a coma for 32 h are expected to have a decrease in recollection of 0.90 standard deviations below normal levels, whereas familiarity is essentially unchanged. Fig. 6B presents the estimated effects of aging on recollection and familiarity. Estimates in Fig. 6B are plotted as z -scores at two ages, where levels of recollection and familiarity at the mean age of the sample (62 years) are set to zero. The estimates of recollection and familiarity were derived using the regression coefficients in modification III that linked age to the recollection and familiarity factors. recollection and familiarity are plotted for 1S.D. below the mean age (50 years) and 1S.D. above the mean age (74 years). The figure shows that relative to the mean age, younger hypoxic patients (i.e., 50 years old) had a level of recollection that was 0.41S.D. greater, and older hypoxics (i.e., 74 years old) had a level of recollection that was 0.41S.D. lower. By contrast, familiarity is essentially unchanged for younger and older hypoxics (Fig. 7).

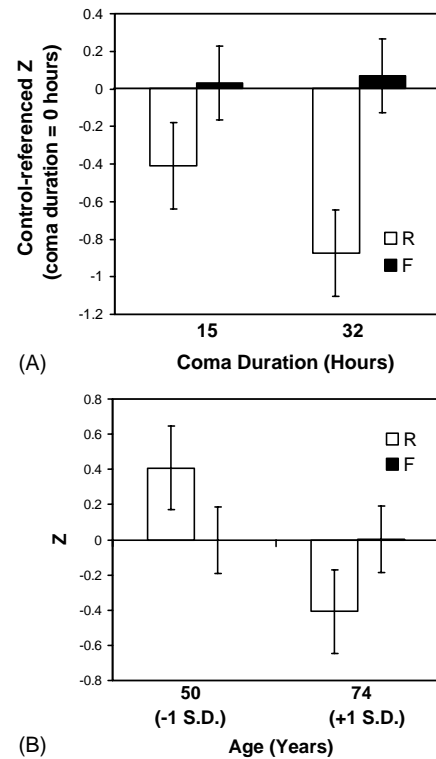


Fig. 6. Estimated effects of (A) coma duration on recollection and familiarity and (B) age on recollection and familiarity. Panel (A) shows model-estimated z -score differences in recollection and familiarity factor scores relative to scores expected at a coma duration of zero. Panel (B) shows model-estimated z -score differences in recollection and familiarity factor scores relative to scores expected for the mean age of the sample. R: recollection; F: familiarity.

In summary, the dual-process recognition model provided a statistically acceptable account of the data, and a significant improvement in the fit over the single-factor model. Moreover, several of the assumptions underlying the model were assessed and found to be consistent with the data. That is, the assumption that recollection and familiarity were independent across individuals was supported, and the assumption that hypoxia and aging influenced recollection but not familiarity was also supported.

3.5. The hybrid three-process model

A hybrid model combining the dual-process account of recognition with the dual-process account of recall is presented in Fig. 7. The model assumes that recall is directly influenced by controlled search and recollection, whereas recognition is influenced by recollection and familiarity. The best-fitting parameter estimates for the model are shown in Fig. 7, and the fit statistics for the model are presented in Table 4. The fit of the model was not rejectable statistically, $\chi^2(19, N = 56) = 15.60$, $P = 0.68$, and the practical fit indices were quite good, with CFI = 1.00 and NNFI = 1.02. However, there was no improvement in fit over the dual-process recognition model, $\chi^2(1, N = 56) = 0.62$,

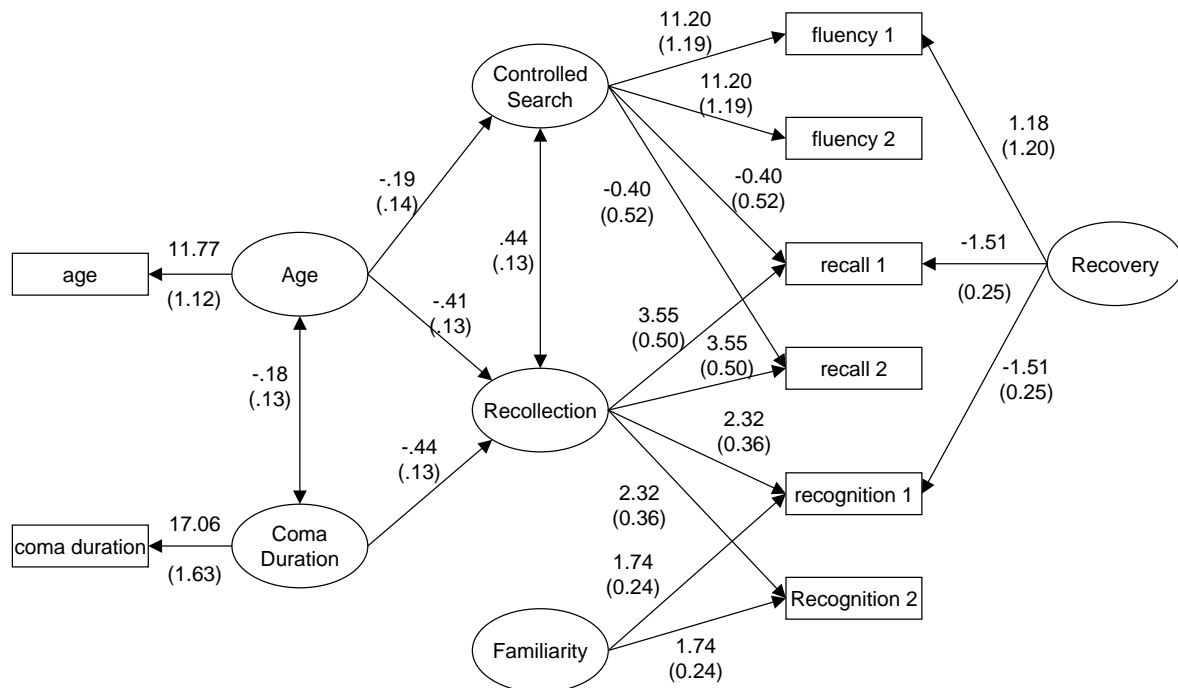


Fig. 7. Path diagram of the hybrid model in which recall loads on recollection and controlled search factors and recognition loads on recollection and familiarity factors. Parameter estimates are shown for the standardized solution with standard errors in parentheses.

$P = 0.43$, indicating that allowing strategic processes to directly affect recall did not lead to an improvement in the dual-process recognition model. There was, however, an improvement in fit over the dual-process recall model, $\chi^2(1, N = 56) = 17.58, P < 0.001$, indicating that adding a familiarity process to the dual-process recall model did lead to a significant improvement in the model.

In summary, the hybrid model, which made dual-process assumptions for both recall and recognition, accounted for the covariance matrix better than the dual-process recall model, but did not improve on the dual-process recognition model. The results suggest that assumptions underlying the dual-process model of recognition are critical in accounting for recall and recognition memory.

3.6. General comparison of all model solutions

An examination of the fit indices for the four models (see Table 4) indicates that all models including a contribution of familiarity to recognition (i.e., the dual-process recognition and hybrid models) were associated with acceptable χ^2 ($P > 0.05$) and RMSEA values (< 0.10), whereas all other model solution were rejectable. This indicates that, relative to the effects of other parameters we tested, the addition of familiarity to recognition was the most critical for improving the fit of a single-process model of recall and recognition.

Non-nested models cannot be compared using the χ^2 -statistical index. However, the CAICs can be compared for models across different nested hierarchies. For any given set of models, the one with the lowest CAIC value fits the

data best given the number of free parameters. The model with the best CAIC was the dual-process recognition model, followed by the hybrid model, and then by different versions of the single-process and dual-process-recall models. The CAIC results indicated that the dual-process recognition model was the most preferred of the various models.

One final issue is whether the preferred model for the hypoxics also provides a reasonable account of the control data. To address this, the dual-process recognition model was modified slightly and fit to the 89 healthy control subjects. The model was similar to that in Fig. 4 with the exception that coma duration and fluency scores were not included in the covariance matrix (the latter was not measured in this group), and recovery, coma, and controlled search factors were removed from the model. Additionally, because there were variance differences across test sessions in the controls, the error variances of time 1 and time 2 measures were allowed to differ. This modified dual-process recognition model for controls was not rejectable, $\chi^2(6, N = 89) = 11.41, P = 0.08$ and other fit indices were acceptable (RMSEA = 0.095, $P_{\text{close}} = 0.181$; NNFI = 0.94, CFI = 0.96). Thus, the preferred model for the hypoxics is also a reasonable model for the controls.

4. Discussion

Structural equation modeling was used to assess the ability of three general classes of explicit memory theories to account for covariance of coma duration, age, verbal

fluency, recall and recognition memory in a group of sudden cardiac-arrest patients. The results revealed that there were two separate sources of memory-related variance underlying recognition performance, one of which also contributed to recall. A single-process model with only one memory component underlying recognition and recall failed to provide an adequate account of the data. A dual-process recall model, in which recall loaded on a declarative memory factor and a controlled search factor, also failed to account for the data, and it did not provide a significant improvement over the single-process model. Moreover, several modifications of that model also failed to account for the data. In contrast, a dual-process recognition model where recognition loaded on both a recollection factor and a familiarity factor, but recall loaded only on recollection, provided an acceptable account of the data, and was a significant improvement over the single-process model. Moreover, further analyses indicated that several of the model's underlying assumptions were supported, including the assumptions that recollection and familiarity were independent, that coma duration and aging influenced recollection but not familiarity, and that recall was influenced by recollection but not by familiarity. Finally, a hybrid model incorporating the assumptions of the two dual-process models provided a significant improvement over the dual-process recall model, but not over the dual-process recognition model, thus giving further support to the dual-process recognition model.

4.1. The relationship of the current results to previous studies

The current results converge with previous studies examining the overall effects of mild hypoxia on recognition and recall. For example, in a previous analysis of the recall and recognition deficits in the same group of cardiac-arrest patients examined in the current study, recall was disproportionately disrupted compared to recognition (Yonelinas et al., 2002). To the extent that hypoxia disrupted recollection, but not familiarity, recall should be more disrupted than recognition. Other studies examining patients with relatively restricted hippocampal damage or damage to the fornix have found similar deficits (Aggleton & Shaw, 1996; Baddeley, Vargha-Khadem, & Mishkin, 2001; Mayes et al., 2001; Vargha-Khadem et al., 1997).

Unlike the previous studies, however, the current analysis was not aimed at testing a single prediction about the overall level of performance, but rather quantitatively assessed different models' abilities to account for the covariance of several different variables related to explicit memory. The results indicate the dual-process recognition model provided a statistically acceptable account of the data and was in closer agreement with data than either the single-process or the dual-process recall models. However, the results also suggest that minor modifications would bring rejected models in line with the data. For instance, allowing a subdivision within the medial temporal lobe to account for

independent familiarity and recollection effects would appear to improve the models that incorporate a dual-process view of recall (e.g., Moscovitch, 1994; Shimamura, 1995; Squire & Knowlton, 1995).

The current results converge with results from other methods that have been used to examine the effect of hypoxia on recollection and familiarity. For example, several hypoxics from the present sample of cardiac-arrest patients have been studied using a remember/know procedure in which subjects indicated whether each test item was recognized on the basis of recollection or familiarity, and using a receiver operating characteristic (ROC) procedure in which subjects rated the confidence of their recognition judgments (Yonelinas et al., 2002). Estimates derived from both the remember/know and ROC procedures converge with those of the present structural equation modeling results in showing that the hypoxics exhibited deficits in recollection, but not familiarity. In that study, a simplified structural model was also used that did not consider controlled search processes or alternative models, and it too led to comparable conclusions.

The present results also converge with those from other methods indicating that healthy aging is related to a decrease in recollection but not familiarity. For example, the remember/know and ROC procedures, as well as the process dissociation procedure (Jacoby, 1991) in which recollection is measured as the ability to remember where or when an item was earlier studied, all converge in showing that aging leads to a deficit in recollection that leaves familiarity intact (for reviews see Light, Prull, La Voie, & Healy, 2000; Yonelinas et al., 2002). Moreover, several previous studies have indicated that recollection deficits in aging are related to deficits in executive function as measured by tasks like verbal fluency and card sorting (e.g. Glisky et al., 1995; Parkin & Walter, 1992).

The similarity of the results derived from the dual-process recognition model with those from the existing literature using other methodologies increases our confidence that the structural modeling method provided an accurate account of the processes underlying explicit tests of memory. Moreover, this convergence verifies that the components identified in the models reflected the processes we aimed to examine. That is, the two sources of variance that contributed directly to recognition in the dual-process recognition model were labeled recollection and familiarity, but there is nothing inherent in the structural models themselves that necessitate what these factors correspond to. The only way to do this is to demonstrate convergence between measures of these factors and other measures of these processes. The fact that the two factors behaved in accord with previous studies of recollection and familiarity suggests that we correctly identified the model components.

4.2. Limitations of the current study

When evaluating the results of the present study, it is important to consider some limitations of the structural

equation modeling approach (Cliff, 1983). For example, the models tested here are not confirmed by the data; models can only be disconfirmed. There may be any number of additional models we did not test that account for the covariance matrix as well as, or better than, the models presented here. The model testing effort presented here was intended to identify which aspects of existing theoretical proposals are consistent with the observed relationships between recall and recognition, and where the models need revision.

Although the models contain causal relationships (e.g., it was assumed coma duration causes changes in recollection), the models themselves do not actually confirm the direction of causality for any of the relationships. For relationships involving necessarily antecedent variables such as age and coma duration, it would make no sense to reverse the direction of the causal path in the model (e.g., to allow memory to affect age or coma duration). On the other hand, the direction of the relationship between search processes and memory was somewhat ambiguous. Although it is likely that controlled search processes influence declarative memory or recollection, it is also possible that these processes may depend to some extent on memory. For the models presented here, varying the direction of this path did not affect the model fits nor did it lead to appreciable differences in the path estimates. Thus, the current conclusions hold regardless of the true causal relationship between these two factors. Nonetheless, future studies further examining the nature of the interaction between strategic processes and recollection will be useful in fully characterizing the dual-process model.

It is unknown whether our conclusions about the effects of cardiac-arrest hypoxia generalize to other forms of hypoxia or to patients suffering from more severe hypoxic events. For example, hypoxia can arise from several different causes, including respiratory arrest, carbon monoxide poisoning, and anesthetic accidents, and these may have different neuropathological and behavioral consequences (Caine & Watson, 2000). Moreover, as the severity of the hypoxic event increases, the likelihood increases that regions outside the hippocampus are involved (e.g. Smith, Auer, & Siesjo, 1984), and thus, more severe hypoxic patients may exhibit deficits in both recollection and familiarity. Support for the possibility that severe hypoxic patients may behave differently comes from a recent study by Manns et al. (2003) who reported results from a remember/know experiment on a group of patients that included hypoxic patients. They found that the patients exhibited deficits in both recollection and familiarity, which is in conflict with the remember/know results reported by Yonelinas et al. (2002). Although this discrepancy might reflect that fact that some amnesics have difficulties using the remember/know distinction (Baddeley, Vargha-Khadem, & Mishkin, 2001), the deficits in the former study may be attributed to the heterogeneity of their patients' etiologies and greater severity of the hypoxic episodes suffered by those patients. The patients studied here and in the Yonelinas et al. (2002) study were all mild cardiac-arrest hypoxics who suffered hypoxic

events of a few minutes or less and had coma durations that were less than 72 h. Our patients' memory impairments were also mild relative to Manns et al.'s (2003) group. For example, the average delayed recall and recognition deficits in our patient group were less than two standard deviations below the control mean for time 1 and less than one standard deviation below the control mean for time 2. Although the severity of the hypoxic event suffered by the patients in Manns et al. (2003) was not assessed quantitatively, they appear to be more amnesic: all but one WMS-R delayed memory score was below 50. Although WMS-R scores were not available for all of the present sample, the immediate RAVLT recall scores in our Table 2 are higher than the immediate RAVLT scores reported for the amnesics in the Manns et al. (2003) study. Future studies examining recollection and familiarity in more severely hypoxic patients using measurement methods other than the remember/know procedure will be important in resolving this issue.

Another question regards the extent to which there may be search processes selectively related to recall. In the current study, the controlled search factor was related to the recollection factor but not directly to recall. This means that any variance shared by verbal fluency and recall was also shared by recognition. However, it must be emphasized that the current results do not indicate there are no controlled search processes that are differentially involved in recall and recognition, only that in the current patient group, our estimate of these processes had its impact on recall and recognition via the recollection process. In fact, this result is perfectly consistent with component-process models like that of Moscovitch and colleagues (Moscovitch, 1990, 1994) that allow for contributions of common controlled processes to recall and recognition performance.

There are also two critical limitations that must be kept in mind when interpreting the finding that search processes did not directly affect recall. First, this pattern of results may not hold for different groups of patients. For example, as discussed earlier, patients with selective frontal lobe lesions also have greater deficits in recall than recognition. Their deficits may arise for different reasons than those of the hypoxic patients, so it is possible that for frontal lobe patients, there may be evidence for a direct link between search processes and recall that is distinct from that between search processes and recollection. Second, the relationship between search processes and recall may depend on the type of executive function tasks that are measured. For example, if another measure of executive control such as the WCST were used, more evidence for a direct link between executive control and recall may have arisen. There is some evidence for this latter possibility, as discussed below.

One final question about the current results is whether it is ever possible for familiarity to contribute to recall performance. The current results indicated that familiarity did not contribute to recall performance. Given that subjects were not provided with any retrieval cues in the recall test, it may not be surprising that assessments of familiarity did

not contribute to performance. However, in other types of recall tests, familiarity may be quite useful. For example, in cued recall tests subjects are presented with cues and must generate candidate responses, and in this case, assessments of cue familiarity may be useful. Moreover, in study lists where the items are interrelated or form semantic categories, subjects may be able to generate items that might be assessed for familiarity.

4.3. Neuroanatomical substrates of recollection and familiarity

The current results are consistent with models that propose that recollection is subserved by the hippocampus, whereas familiarity relies on surrounding temporal lobe regions (e.g. Aggleton & Brown, 1999; Eichenbaum et al., 1994). That is, to the extent that mild cardiac arrest leads to selective hippocampal damage, the selective effects seen on recollection indicate that the hippocampus is required for recollection but not familiarity.

Two cautionary points regarding this conclusion must, however, be acknowledged. First, selective hippocampal damage could not be verified in the current patient group because high resolution MR scans could not be obtained, so claims about the selectivity of hippocampal damage can only be inferred on the basis of previous studies of similar patients. As discussed in the introduction, hypoxia has been used as a model for selective hippocampal damage, but at least in severe cases of hypoxia, the damage has been documented in surrounding brain regions. Moreover, even if neuroimaging and histological examinations detect no damage outside the hippocampus it is possible that such damage was simply undetected (Caine & Watson, 2000). Although a wealth of lesion studies in non-human primates and rats have indicated that the hippocampus plays a necessary role in explicit memory, the specific deficits seen in hypoxia may be due in part to damage outside the hippocampus. Second, hypoxia frequently does not damage the entire hippocampus, and thus the results may point to functional segregation within the hippocampus rather than a segregation between the hippocampus and parahippocampal gyrus. That is, it may be the case that there is a portion of the hippocampus damaged in mild hypoxia that is necessary for recollection, but not familiarity. Conversely, some other region in the hippocampus not damaged by mild hypoxia may be necessary for familiarity. Although this possibility is highly speculative, it is consistent with the finding that hypoxia tends to influence some regions within the hippocampus, such as the pyramidal cells in CA1, much more than other hippocampal regions, such as the dentate gyrus (e.g. Zola-Morgan, Squire, & Amaral, 1986), and with recent evidence that different regions within the rat hippocampus may support different mnemonic functions (e.g. Bachevalier & Meunier, 1996; Gilbert, Kesner, & Lee, 2001; Kesner, Gilbert, & Lee, 2002).

In addition to the contribution of the MTLs, the current results are also consistent with the claim that the frontal lobes

play a particularly important role for recollection. That is, the factor intended to represent controlled search processes related to frontal lobe function was correlated with recollection, but not correlated with familiarity. Moreover, aging, which is known to lead to pronounced frontal lobe atrophy (Woodruff-Pak, 1997), was found to influence recollection, but not familiarity. Thus, the current results are in agreement with those of previous studies indicating that recollection is dependent in part on the frontal lobes (e.g. Glisky et al., 1995; Janowsky et al., 1989a; Knowlton, 1998; Parkin & Walter, 1992; Shimamura et al., 1990).

The specific role of frontal lobe processes in explicit memory, however, has not yet been well specified. In the current study, we focused on one type of controlled process (i.e., memory search), and we examined only one measure of this process (i.e., verbal fluency). Although verbal fluency is sensitive to frontal lobe damage (Baldo et al., 2001; Bentler & Bonnett, 1980; Shimamura, 1995) and shares a variance component with recall and other tests of executive function (Salthouse, Fristoe, & Rhee, 1996), there is growing evidence that there may be several partially distinct executive control functions that are differentially involved in strategic uses of explicit memory (e.g. Parkin & Lawrence, 1994; Parkin, Yeomans, & Bindschaedler, 1994). For instance, the verbal fluency task may rely on spontaneous flexibility, or the readiness of a response to a cue (Eslinger & Grattan, 1993), whereas other frontal tasks like the WCST may reflect reactive flexibility, or the facility with which response strategies change when task demands change (Parkin & Lawrence, 1994). While spontaneous flexibility as measured by verbal fluency appears to be related to recollection in both recall and recognition, reactive flexibility as measured on other tasks like WCST may load more exclusively on memory processes supporting recall compared to recognition. In support of this possibility, the number of errors on the WCST correlates highly with the discrepancy between recall and recognition, whereas verbal fluency does not (Parkin & Lawrence, 1994). However, the cause of the former correlation is unclear, and there is evidence that WCST performance relies in part on the explicit memory for previously learned strategies (Corcoran & Upton, 1993). Thus, it cannot be determined whether shared variance between WCST and recall is a result of executive task-switching process, memory retrieval processes, or both. The important point is that the present models do not rule out the possibility of other direct frontal lobe contributions to recall, and future studies are needed to clarify the functional and neuropsychological relationships among the memory processes and different executive control processes.

5. Conclusions

The results demonstrate that the examination of the covariance of recall and recognition on the one hand, and factors known to influence memory such as hypoxic severity

and aging on the other, can be critical in testing conflicting theories of explicit memory. By modelling covariance structures, we found that single-process and dual-process recall models were inadequate to account for performance, whereas a dual-process recognition memory model did provide an acceptable account of performance. Although covariance modeling clearly does not replace the need to examine the effects of different experimental variables and group differences on overall memory performance, the current results indicate that it can be critical in providing quantitative assessments of existing theories in ways not easily addressed by traditional methods.

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