1 Amnesia and Memory Research

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Recent years have witnessed an explosion of research in the analysis of memory and amnesia. For the student of normal memory, this has meant an endless stream of experiments and theory exploring countless phenomena of memory and the appearance of several journals devoted almost exclusively to this research. Although not quite so large in comparison, the ever-increasing flow of articles concerning human amnesia that have appeared in journals such as *Cortex* and *Neuropsychologia* over the past 15 years testifies to the growing vitality of this important sector of psychological research.

Unfortunately, and despite the increasing vigor of their respective fields, students of normal and abnormal memory have not enjoyed active communication with one another; consequently, the developments in the two fields over the past 15 or 20 years have proceeded largely in parallel. In the experimental psychology of intact memory, perhaps the sole point of meaningful contact with amnesia research has taken the form of the inclusion of clinical facts about the amnesic syndrome in theoretical discussions of the distinction between short-term and long-term memory (Atkinson & Shiffrin, 1968; Wickelgren, 1973, 1979). Beyond that, however, it would be difficult to find any systematic account of amnesic phenomena in the numerous recent theories of normal human memory, and utilization of amnesic data by mainstream experimentalists in attacking problems of normal memory is likewise rare. Indeed, several leading researchers have contended that amnesic phenomena currently lie outside the desirable boundaries of memory research. Murdock (1974), for instance, has cautioned that: "It is difficult enough understanding the memory of normal college students; it will be time to consider the abnormal cases after we can cope with the normal cases [p. 5]." And Postman (1975) has cast doubt on the usefulness of amnesic data for

experimental psychologists, warning that: "extrapolations from pathological deficits to the structure of normal memory are of uncertain validity [p. 308]."

There are, however, some positive signs of change. For instance, one can point to the recent appearance of papers concerning various amnesic phenomena in "hard-core" memory journals: Several studies of Korsakoff patients have appeared in the *Journal of Experimental Psychology* (Cermak & Reale, 1978; Oscar-Berman, 1976), experiments concerning drug-induced amnesia have been published in the *Journal of Verbal Learning and Verbal Behavior* (Birnbaum, Parker, Hartley, & Noble, 1978; Eich, Weingartner, Stillman, & Gillen, 1975; Hartley, Birnbaum, & Parker, 1978), and a recent issue of *Memory & Cognition* contained an experimental study of Korsakoff amnesics (McDowell, 1979). Furthermore, the appearance of theories such as the one recently put forward by Wickelgren (1979) constitute a major advance in pulling together the insights of experimental psychology and the observations on amnesia.

It is only fair to point out that students of amnesia have harbored their own doubts concerning the usefulness of experimental psychology in the analysis of memory and its pathology. As long ago as 1901, Pierre Janet, one of the century's most innovative clinical psychiatrists, sarcastically depicted psychology's role in the study of memory as follows: "The psychologists in their descriptions admit of no other elementary phenomena of memory than conservation and reproduction. We think that they are wrong, and that disease decomposes and analyzes memory better than psychology [p. 102]." Despite similar misgivings expressed by other students of pathological memory from time to time, signs of hope are evident here too, as a number of contemporary amnesia researchers have sought to establish an alliance with experimental psychology. Expressed largely in the work of Warrington and Weiskrantz in England, and Cermak and his colleagues in Boston, the methods, findings, and theories of experimental psychology are beginning to find a home in the analysis of amnesia. Studies of amnesia that utilize the insights of experimental psychology have been undertaken by an increasingly diverse group of researchers in the past few years, and there is every reason to believe that this trend will continue in the future.

In short, there is reason to believe that we may be on the verge of a "golden age" in which the interaction between the experimental psychology of normal memory and the investigation of amnesic deficits will be more thorough and meaningful than it has been in the past. Texts like the present one will hasten the arrival of this golden age, inasmuch as one of its important functions is to explore ways to remove the remaining barriers and problems preventing the integration and interaction of the two fields.

The purpose of this chapter is to confront some of these key issues and clarify problems surrounding them. The chapter is offered in a spirit of constructive criticism from two experimental psychologists who firmly believe that both the experimental psychology of memory and the analysis of amnesic deficits stand to benefit from increased understanding and utilization of each other's methods, data, and theories.

The chapter is structured as follows. First, several historical features of the relations between experimental psychology and amnesia research are considered, together with a brief analysis of the losses suffered by each because of its ignorance of the other. Second, some contemporary problems are outlined that have to be confronted before a further integration of the two fields of research can be effected. These problems include subject variability in studies of amnesia, logic of experimental inference, pitfalls in the application of experimental paradigms to amnesic populations, conceptual confusion in experimental study of memory, and difficulties encountered in the laboratory production of analogues of amnesia. In the third and final section of the chapter, a general framework for the study of memory and amnesia is briefly outlined.

HISTORICAL RELATIONS BETWEEN AMNESIA AND MEMORY RESEARCH

It may be indicative of the late 19th-century psychological *Zeitgeist* that Théodule Ribot published his classic treatise on amnesia, *Diseases of Memory* (1882), just three years before Hermann Ebbinghaus thrust his epoch-making experimental study of memory upon the scientific world in 1885. And it is interesting to observe that Ribot foresaw an emerging era in which phenomena of amnesia would be at the center of psychological attention:

> The disorders and maladies of this faculty [memory], when classified and properly understood, are no longer to be regarded as a collection of amusing anecdotes of only passing interest. They will be found to be regulated by certain laws which constitute the very basis of memory, and from which its mechanism is easily laid bare [p. 10].

Alas, the central role that Ribot envisaged for amnesic phenomena in the new psychological science of memory never took shape: The lines of research initiated by his own work and the studies that followed in the tradition of Ebbinghaus occupied virtually nonoverlapping spheres in subsequent years.

The separation between amnesia and memory research becomes clear to anyone who studies the developments in the two fields in the late 19th and early 20th centuries. Investigations of intact memory in the experimental tradition initiated by Ebbinghaus were pursued largely in two countries, America and Germany. The experimental psychology of memory flourished in the 30 or so years following the publication of Ebbinghaus' book. Under the leadership of eminent researchers such as Georg Müller, Ernst Meumann, and Mary Calkins, the young science ambitiously tackled numerous phenomena of memory (see Murray, 1976). But the proliferation of methods, data, and theory that characterized the experimental literature did not include a systematic interest in amnesic phenomena: The mainstream experimental journals were conspicuously free of

articles dealing with memory pathology. There were exceptions, but these were exceedingly rare. For instance, in the first volume of the *Psychological Review*, we find a case study of amnesia resulting from gas poisoning reported by Dana (1894). In what was perhaps the most serious attempt by a mainstream experimentalist to grapple with the problems of amnesia, Burnham (1903) drew on available evidence on processes of organization and perseveration (Müller & Pilzecker, 1900; Müller & Schumann, 1893), to support his thesis that retrograde amnesia is attributable to what we now would call an encoding deficit: "The memory is lost because it was never completely organized [p. 132]." And several years later, Hennig (1910) reported another pathological case study in the *Zeitschrift für Psychologie*. These isolated instances, however, did not succeed in generating any sustained interest in amnesia on the part of experimental psychologists.

A reciprocal lack of interest in experimental psychology can be discerned when one examines the articles appearing in clinical journals that published research on amnesia. These papers were for the most part comprised of extensive case studies or descriptive analyses of small pathological populations (Coriat, 1907; Freund, 1889; Gregor & Roemer, 1907; Konrad, 1907; Korsakoff, 1889/1955; Kutner, 1906; Moll, 1915; Paul, 1889). The authors of these studies either did not know of, or did not care about, the experimental facts generated by mainstream memory researchers; the names of Ebbinghaus, Müller, and Meumann that dominated the experimental literature are not to be found in most clinical reports. Again, the exceptions that can be uncovered were few. Krauss (1904) utilized recently developed tachistoscopic techniques for evaluating memory span in his case study of an amnesic patient. Buckley (1912), citing the experimental work of Kirkpatrick (1894) on modality effects, commented that data obtained from his patient contradicted the previous findings that visual memory exceeds auditory memory: "Words written for him were not remembered as easily as when they were spelled for him audibly [p. 436]." And in a rather thorough analysis, Pick (1915) applied theories of thought processes derived from the tenets of the Würzburg school to his analysis of Korsakoff patients and concluded that these patients exhibited a deficit in goal-directed thinking that could be comfortably explained by the Würzburg theory.

Before we consider some of the consequences that followed from the mutual isolation of amnesia and memory research, we should point out that a brief period of reciprocal interest did seem to take shape starting around 1930. For instance, we find three studies of amnesia appearing within a year of each other in the experimental journal *Archiv für die Gesamte Psychologie*. Ranschburg (1930), a well-known experimental psychologist, applied mainstream theory to disorders of attention and memory; Krauss (1930) attempted to conceptualize amnesia in terms of Kurt Lewin's recently formulated dynamic theories; and Störring (1931) informed experimental psychologists of a dramatic case of pure amnesia that in several respects resembles the famous modern case of H.M. Similarly, three papers on amnesia appeared in close temporal proximity to each other in various

American psychology journals. Both Ray (1937) and Sears (1936) were impressed by the apparent similarities between clinically documented retrograde amnesia and the laboratory phenomenon of retroactive inhibition. Sears cautiously concluded that the resemblances between the two phenomena were "suggestive [p. 237]," whereas Ray argued more boldly that: "these phenomena are manifestations of the same underlying mechanism [p. 341]." And Conkey (1938) used a variety of newly developed psychological tests to explore memory and other intellectual deficits following closed-head injury.

The interest in amnesia demonstrated by the aforementioned psychologists in the 1930s was matched by a new willingness on the part of clinicians to utilize conceptual tools provided by experimental psychology. Bürger (1927), for instance, offered an interpretation of memory pathology cast in terms of the newly popular gestalt theory. Syz (1937) provided an interesting discussion of concussion amnesia that drew heavily on experimental work on interference and on what appeared to be a new and iconoclastic theory of memory proposed by Bartlett (1932). Gillespie (1937) attempted an even more detailed account of amnesia within the framework of Bartlett's theory and explored the possibility that defective operation of schemata and voluntary recall contributed to amnesic pathology.

The manifestation of mutual interest between the two groups of students of memory in the last 10 years or so before the second World War was not exactly overwhelming, and the reasons for the apparent beginning of a rapprochement are not clear—neither are the reasons for its apparently abrupt termination.[1] But it does seem appropriate to suggest that the seeds of several intriguing research possibilities were sown during this period.

For the most part, as we have seen, there existed a gulf that separated the experimental psychology of memory and the clinical study of amnesia in the years following the contributions of Ebbinghaus and Ribot. What were its consequences? We believe that both sides missed out on potentially key insights from the other. On the clinical side, the parade of case studies that dominated the journals remained free of standardized experimental procedures and explored only a few restricted areas of memory function in amnesic patients. It is precisely in these areas that the experimental psychology of memory could have aided students of amnesia most: Mainstream memory researchers had developed an arsenal of research techniques and generated a vast body of data that could have increased substantially the scope of amnesia research and enriched its analytical precision. Early students of amnesia paid scant attention to these possibilities, and their research remained correspondingly narrow.

But the experimentalists also paid a price for ignoring the data generated by clinical observations of amnesic deficit. As we have argued elsewhere (Schacter, Eich, & Tulving, 1978), amidst the impressive amount of experimental evidence

[1] It is worth noting that in Britain, students of intact memory and students of amnesia have demonstrated a much more consistent interest in each other's efforts over the past half-century.

reported by post-Ebbinghausian researchers, one important area of memory function remained virtually unexamined: the process of retrieval and the psychological conditions that affect its success or failure. Memory research at the turn of the century was powerfully influenced by the classical doctrine of associationism, which regarded one variable as particularly crucial to the outcome of memory performance: the strength of associations. In keeping with this idea, early research focused its theoretical attention on elucidating the variables that controlled the strength of associations: repetition, nature of materials, presentation modality, interference, retention interval, and so on. If the strength of association between two elements was strong enough, then recall of one in the presence of the other was assumed to be assured—whatever the conditions of retrieval. This all at the time when students of amnesia knew better. Their data on shrinking retrograde amnesia and so-called functional amnesia, in which successful recall was specific to a given psychological state (Prince, 1916), indicated that retrieval could initially fail and later succeed without any change in the strength of associations; it also showed that retrieval was sensitive to specific psychological conditions. The mainstream experimentalists, however, took little notice of these observations and continued to study memory without reference to conditions and processes of retrieval; their unduly restricted approach could have been considerably enriched had they been willing to ponder seriously the data generated from various amnesic conditions. Robert Sears acknowledged this sorry state of affairs in his 1936 paper: "In general the experimental psychology of memory has been most productive in studying the effects of various conditions of learning on recall; little or nothing has been done with the study of recall itself independent of the learning process. It is essentially in this latter field that amnesia of reproduction lies [pp. 269–270]." Until experimental psychology came to grips with the problems of retrieval, Sears contended, "it will have little of significance to offer in the field of memorial abnormality [p. 270]." His prophetic statement seems to have been vindicated by current trends.

This section of the chapter can be summarized simply: Both the experimental psychology of memory and the study of amnesia have suffered from their failure to take each other seriously. We think that neither field can afford to continue the practice.

INTERACTION OF MEMORY AND AMNESIA RESEARCH: FIVE PROBLEMS

In this section of the chapter we discuss five problems whose clarification, we believe, is central to a more satisfying interaction between students of memory and amnesia. The first three topics concern various aspects of the amnesia literature: (1) problems of subject variability; (2) the interpretation of differences between pathological and normal populations; and (3) the application of

paradigms derived from experimental psychology to amnesic phenomena. We scrutinize these problems critically from our vantage point as experimental psychologists. The fourth section focuses on possible difficulties encountered by the student of amnesia in following theoretical developments in the experimental study of normal memory. In the final section we briefly describe and evaluate the efforts of experimental psychologists to model patterns of amnesic deficit in normal subjects.

Subject Variability

One of the first questions that an experimental psychologist approaching the literature on amnesia must ask himself is almost embarrassingly naive: What is an amnesic? Clearly, one must have some relatively specific notions of what kinds of patients are called "amnesic" before one can make meaningful comparisons between experiments performed with different samples of patients. Yet this seemingly innocent question will more than likely cause considerable confusion for an experimental psychologist who wishes to compare data across experiments with different subjects in the hope of extracting some general conclusions about the nature of amnesic deficits.

The experimental psychologist who starts exploring the amnesia literature will quickly find that there are no strictly defined criteria that describe the boundaries of the amnesic syndrome. Rather, he will find that patients suffering from a considerable variety of neurological deficits—Korsakoff's syndrome, encephalitis, Huntington's chorea, penetrating brain lesions, and closed-head injury, to name just a few—are all loosely described as "amnesic" by various investigators. A fairly typical characterization of the amnesic syndrome is provided by Baddeley (1975):

> The most striking feature of such patients is that they appear to forget incidents in their daily life just as fast as they appear. Typically they are unable to tell you where they are, what year it is, who is Prime Minister, what they had for breakfast or to supply any of the vast number of details of daily existence that a normal person takes for granted [p. 328].

Although such characterizations are no doubt clinically accurate and useful, they are of little help to the experimental psychologist first grappling with the amnesia literature: He is told that amnesics are people who forget a lot, that is, that amnesics are amnesics.

The problem becomes more complex when it is realized that not all amnesics are, in fact, characterized similarly. For example, it has been demonstrated that amnesics suffering from Korsakoff's disease and Huntington's chorea can be distinguished by a number of features of their memory performance. Korsakoff amnesics' performance can be aided by distributed (versus massed) practice and by conditions of low (versus high) proactive interference, whereas the perfor-

mance of Huntington's chorea patients is not significantly affected by these manipulations (Butters, Tarlow, Cermak & Sax, 1976); Korsakoff patients tend to make errors of *intrusion* in the Brown-Peterson short-term memory task, whereas chorea patients make more errors of *omission* (Meudell, Butters, & Montgomery, 1978); and Korsakoff patients benefit from the presentation of category names as retrieval cues after studying a categorized word list (Cermak, Butters, & Gerrein, 1973), whereas chorea patients apparently do not (Caine, Ebert, & Weingartner, 1977).

Experimental psychologists may be able to minimize possible confusion over various controversies in the amnesia literature if they are attuned to these possible patient population differences. For instance, if one traces the various disagreements between the research group headed by Cermak and Butters in Boston, who favor an "encoding-deficit" interpretation of amnesia, and the Warrington and Weiskrantz team in England, who have favored a "retrieval-deficit" explanation, it may be seen that these investigators have based their conclusions on quite different populations of patients. The Boston group has generally restricted itself to populations of alcoholic Korsakoff patients, whereas Warrington and Weiskrantz have studied patients with multiple etiologies. A number of questions then arise: In what sense have these investigators all been studying "amnesics"? Would an amnesic defined by the criteria of one group be considered amnesic by the other? More generally, how valid are theoretical conclusions drawn from a given patient sample without reference to some standardized, "base-line" criteria that would serve to establish the degree to which one sample of patients is comparable with others? And in what sense do we have a theoretical controversy as to the nature of amnesia, if it is possible that different theoretical positions apply to different groups of patients?

The problem of comparability of patients used in experiments is a severe one, and no facile solutions for it exist. One useful change in current practice would be for students of amnesia to agree upon and adopt a screening device, a battery of tests, that would be routinely administered to all subjects in experiments and the outcome reported for each patient in the form of a "memory profile." Such a profile would serve as a base line for investigators attempting to compare studies performed on different samples of patients. Clinical batteries such as the Wechsler test are probably too coarse and theoretically outdated to be of much use; it would make more sense to create a screening device that incorporates the recent knowledge acquired in both memory and amnesia research. Although problems might arise in the construction and adoption of such a test, it seems clear that something like it is urgently required not only to facilitate communication between experimental psychologists and students of amnesia, but also between different groups of students of amnesia. Until such time as the problem of subject variability is directly confronted and solutions sought, we must not be surprised to find many experimental psychologists who share Postman's doubt

concerning the "uncertain validity" of amnesic data in building theories about human memory.[2]

Characteristics of Pathological Memory: Logic of Experimental Inference

One of the principal aims of amnesia research is to selectively identify features and aspects of memory performance that are especially impaired in a given population. It is not enough simply to demonstrate that amnesics perform at a generally lower level than normals; it is also important to know which features of memory are *differentially* affected in an amnesic group. The analysis of differential effects would be informative in at least two different ways. First, if it is observed that a given feature of memory (e.g., semantic encoding, short-term memory, retrieval processes) is more severely impaired than others in a specific constellation of neurological deficits, then it is permissible to infer that deficits in this particular feature of memory are critically implicated in the observed amnesia. Second, the observation that different aspects of memory are especially affected in amnesia may provide investigators with important clues to the organization and functioning of intact memory.

The observation of differential effects is of basic significance in the study of amnesia. We have found it useful to formulate a *rule of differential effects:* The effect of an experimental manipulation must be greater for amnesics than control subjects before deficits in the memory performance of amnesics can be attributed to corresponding underlying processes. It does not matter how dramatically a particular treatment (e.g., cuing, providing additional time for rehearsal) affects the operation of a given memory process in a group of amnesics. One can only entertain the hypothesis that amnesia reflects a deficit in a particular process when it can be shown that the memory performance of an appropriate control group is not affected, or is affected less markedly, by the same treatment. The fundamental importance of the rule of differential effects has been implicitly accepted by students of amnesia for a long time, albeit often overlooked. Recently Weiskrantz (1978, p. 377) explicitly postulated it as a necessary condition to be satisfied before theoretical statements about observed experimental findings can be made.

Whether or not "simple" differential effects (corresponding in statistical terminology to noncrossover interactions) are *sufficient* conditions for inferring selective deficits in underlying processes is a debatable point. Some of the

[2]In this connection we must note that a very promising beginning in the direction discussed in the foregoing has been made by Warrington and Weiskrantz (1978). They provided systematic diagnostic and test data for each of their 11 amnesic patients. It would greatly help matters if other investigators followed suit.

potential pitfalls that are associated with inferring selective deficits from such noncrossover interactions are cogently discussed by Crowder (this volume), and this issue merits careful consideration in future analyses of amnesic populations. It seems most prudent to suggest that "simple" differential effects may be *necessary* conditions for maintaining hypotheses concerning underlying deficits but they may not be *sufficient* conditions for establishing the precise nature of these deficits.

Many readers of this volume are probably wondering why we belabor the obvious: Everyone ought to know that differential effects are an important aspect of amnesia research. The reality of the situation, however, is not so simple: There exist in the literature many cases in which the rule of differential effect is violated. Consider, for example, a recent study that highlights the central issue in a relatively unambiguous fashion. This is Experiment 3 reported by Cermak and Reale (1978). These authors examined the role played by depth of processing in the amnesia of Korsakoff patients. Their experiment was patterned after the studies of Craik and Tulving (1975), which demonstrated that, in normal college students, memory performance for various kinds of verbal materials is better following semantic analyses of these materials at the time of study than following orienting tasks involving acoustic or graphemic analyses. Cermak and Reale compared recognition memory of control and of Korsakoff patients for words about which the subjects had made graphemic, acoustic, or semantic judgments at the time of study. The experiment produced two critical findings. First, Korsakoff patients did show a "levels effect": Recognition performance was highest in the semantic condition, intermediate in the acoustic condition, and poorest in the graphemic condition. This is exactly the finding that has been observed in many experiments with normal subjects. The second critical finding of the experiment was that the control subjects' recognition performance was at ceiling because of the short list needed to show any effect on Korsakoff patients' performance. Cermak and Reale (1978) used the Korsakoffs' data alone to support their contention that under noninstructed conditions: "Korsakoff patients' anterograde amnesia might stem, in part, from a tendency not to perform semantic encodings of to-be-remembered verbal information [p. 173]." Applying the rule of differential effects, we must conclude that these data do not demand such an interpretation. In order to argue that Korsakoffs' amnesia "stems from" a deficit in semantic encoding, it would be necessary to observe that the memory performance of amnesics is differentially improved, when compared with controls, by experimentally manipulated semantic encoding. Because Cermak and Reale's control subjects' recognition performance was "at the ceiling" in all conditions, and because their amnesic subjects' performance was still far below the performance of controls in the semantic condition, there simply is no evidence in this experiment that forced semantic encoding differentially benefits amnesics. The results of the experiment tell us only that amnesics, like normals in other experiments, do show a "levels effect" under certain experimental conditions. The

rule of differential effects dictates that, solely on the basis of Cermak and Reale's data, no inferences can be drawn about the role played by defective semantic encoding in Korsakoff amnesics.

Consider next a recent study reported by Winocur and Kinsbourne (1978). In Experiment 4 of their paper, these investigators evaluated the hypothesis that a major component of Korsakoff patients' memory disorder could be attributed to defective operation of context-retrieval mechanisms. In order to test their hypothesis, Winocur and Kinsbourne compared performance of Korsakoff patients and controls in two conditions. In the "standard" condition, both subject groups received four presentations of 12 semantically related *A–B* pairs of words and were tested for cued recall of *B* members of pairs in response to *A* members 48 hours later. Testing by relearning was terminated after one errorless trial; if this criterion was not attained, then testing went on for nine trials. In the second condition, all the foregoing procedures were carried out in an identical fashion, but in a highly distinctive "contextual" setting: Both learning and test trials were accompanied by red lights and classical music. The idea of this treatment was to render the study and test conditions, as well as the to-be-remembered material, more distinctive.

Winocur and Kinsbourne's data indicate that Korsakoff patients benefitted substantially from the introduction of the distinctive context: Recall was significantly higher in the distinctive condition than in the control condition. Unfortunately, the performance of control subjects was perfect in both conditions after just two test trials. Winocur and Kinsbourne (1978, p. 678) acknowledged the existence of this ceiling effect, but nevertheless concluded that: "The results of Experiment 4 also support the notion that a major component of the amnesic syndrome is a disruption of mechanisms essential for context-retrieval [p. 679]." Thus, the rule of differential effects was again violated. Because the performance of control subjects was perfect, it is not known whether the control subjects would have benefitted equally from the contextual manipulation under conditions in which the ceiling effects were absent. Accordingly, a legitimate conclusion from Winocur and Kinsbourne's data is that the performance of Korsakoff patients can be improved by introducing a salient and distinctive learning and testing context. What is not legitimate is the inference that defective context retrieval is implicated in Korsakoff's amnesia. This latter hypothesis would be permissible only if it were shown that, in comparison with controls, Korsakoff patients differentially benefit from distinctive contextual cues.

We conclude this section by considering one more case in which the rule of differential effects is ignored: Experiment 1 of the paper by Huppert and Piercy (1976). These investigators explored recognition of three types of materials by Korsakoff amnesics and controls: pictures, high-frequency words, and low-frequency words. Recognition was tested at intervals of 10 minutes, 1 week, and 7 weeks; half of the items were tested by a Yes–No procedure and the other half by a two-alternative forced-choice. We focus on one particularly striking finding

reported by Huppert and Piercy: The amnesic subjects made far more false positive errors in Yes–No recognition on high-frequency words than on low-frequency words. Combined with the high percentage of correct recognition on high-frequency words, these data indicated that amnesics almost always say "Yes" when confronted with a high-frequency word on the test. Huppert and Piercy (1976) used these data as a basis for formulating their major hypothesis:

> The finding that Korsakoffs have an overwhelming tendency to make a positive response to familiar items (frequent words) regardless of whether they are stimuli or fillers, leads us to the hypothesis that recognition by context is extremely weak in Korsakoff patients in contrast to their relatively well-preserved ability to identify items simply as either previously seen or not previously seen [p. 12].

This hypothesis may be correct, but no support for it exists in Huppert and Piercy's experiment if their data are viewed according to the rule of differential effects. It is quite true, as Huppert and Piercy contended, that the number of false positive responses committed by amnesics was much greater for high-frequency words than for low-frequency words. At the three retention intervals, the amnesics made roughly 60%, 65%, and 75% false positive errors for high-frequency words, compared to about 30%, 30%, and 40% for low-frequency words. But when the data of control subjects are examined, a very similar pattern of results can be observed, only with a lower base line: Control subjects committed 30%, 40%, and 50% false positive errors for high-frequency words and only 10%, 15%, and 25% for low-frequency words. Thus control subjects, like amnesics, commit far more false positive errors for high-frequency words than for low-frequency words; there is no interaction between type of subjects and type of words in the false positive data. The existence of such an interaction would be required for the inference that a deficit of "context recognition" contributes to the Korsakoffs' memory disorder. All that can be concluded from the pattern of data they obtained is that the amnesics employ a looser criterion for saying "Yes" to both high-frequency and low-frequency test words than do control subjects.

There is an important lesson to be learned from the difficulties encountered in the studies that have been briefly reviewed in this section: The logic relating data to theory is not often enough spelled out and consequently the inferences that have been made on the basis of experimental findings may not always be logically acceptable. Investigators would do well to keep in mind the distinction between the kinds of inferences that are permissible from nondifferential effects and those permissible from differential effects (when amnesics are compared to appropriate controls). It is precisely this distinction that has been blurred in the illustrative cases we considered. Additionally, even the limits to the kinds of inferences that can be made on the basis of simple differential effects should be carefully considered in light of the points raised by Crowder (this volume). Explicit articulation of criteria that specify varieties of permissible inferences in

amnesic studies, and their acceptance by researchers, will not only benefit the research enterprise, but will also improve the quality of interaction between amnesia and memory researchers.

Application of Experimental Paradigms to Amnesia Research: Some Problems and Pitfalls

One of the more encouraging trends in recent amnesia research has been the steady increase in the application of paradigms and theories derived from the experimental psychology of memory to pathological populations. Many students of amnesia have abandoned the atheoretical clinical "test battery" approach of earlier years and have instead approached their problems within theoretical frameworks. The well-known series of experiments by Cermak and his colleagues as well as those of Warrington and Weiskrantz, and the recent work of Fuld (1976), Riege (1977), McDowell (1979), and Caine, Ebert, and Weingartner (1977) are just a few of the attempts to understand problems of amnesia in the light of experimental study of human memory.

Several problems related to this approach, however, exist that merit critical attention. First there is the problem of selection: Which paradigms from experimental psychology will prove most useful in the analysis of memory dysfunction and how do we go about choosing them? The number of experimental paradigms potentially applicable to amnesic phenomena is truly staggering: The Sternberg paradigm, Brown-Peterson paradigm, priming paradigm, dichotic listening paradigm, recognition-failure paradigm, Bransford and Franks paradigm, directed forgetting paradigm, sentence verification paradigm, Wickens' release-from-proactive-interference paradigm, and Posner's letter-matching paradigm are just a few of the many that students of amnesia have been tempted to utilize. Given this kind of embarrassment of riches, there is one particularly serious pitfall that investigators of amnesia may be well advised to guard against: the temptation simply to import an experimental paradigm in order to find out how a given amnesic group performs on it. In the absence of specific hypotheses that dictate the choice of paradigms, such an approach is doomed to tell us little more than that amnesics perform less well than do normals on many different tasks. It is possible, of course, that such an approach might turn up a few illuminating surprises—amnesics might do better or worse than originally expected in a particular situation—but in all likelihood much effort would be expended in pursuit of vanishingly small theoretical dividends. Selection of experimental paradigms should always be guided by their potential usefulness in distinguishing between theoretical alternatives and providing as strong tests as possible of various theoretical positions.

A second and related problem concerns the responsibility that an investigator assumes when he or she decides to use an experimental paradigm in the analysis of an amnesic deficit. Most paradigms in the experimental psychology of mem-

ory quickly give rise to a sizable literature, in which original interpretations of data generated by the paradigm are revised or seriously questioned, important new results appear, and the limitations of the paradigm are critically assessed. If the study of amnesia is to benefit from the utilization of experimental paradigms, investigators should assume responsibility for acquainting themselves with the complex and often unresolved issues that frequently surround the paradigms of their choice.

We present two examples to illustrate the point. First let us consider a study of closed-head injury patients reported by Brooks (1975). This study, which has been examined in detail elsewhere (Schacter & Crovitz, 1977), used various paradigms from experimental psychology to distinguish between disorders of long-term and short-term memory in patients who had emerged from the temporary phase of posttraumatic amnesia. Brooks relied heavily on data obtained from two experimental procedures to support his argument that long-term memory is defective in these patients whereas short-term memory is intact: a comparison of immediate versus delayed recall in the Glanzer and Cunitz (1966) paradigm, and an analysis of semantic versus acoustic confusion errors. Brooks found that immediate recall for closed-head injury patients was not different from that of controls, but that delayed recall was lower in the patients than the controls. He also found that the patients made relatively few semantic confusion errors. Based on the assumption that the Glanzer and Cunitz paradigm can effectively separate the contributions of short-term and long-term memory, and considering the early results showing the tendency for errors from long-term memory to be semantic confusions (Baddeley, 1966) whereas errors from short-term memory tend to be acoustic confusions (Conrad, 1964), Brooks argued for a differential impairment of short-term and long-term memory in closed-head injury patients.

Brooks' data are beyond dispute, but his interpretation can be questioned in light of literature related to the paradigms Brooks used. For instance, Watkins (1974) has presented strong arguments, based on the data reported in the literature, that the Glanzer and Cunitz paradigm entails severe difficulties as a technique for separating short-term and long-term memory. And Craik and Lockhart (1972) have questioned the validity of semantic versus acoustic error analysis as a measure of short-term versus long-term memory, pointing to experiments demonstrating that a variety of codes are available for short-term memory processing. These are issues that must be faced by investigators who employ concepts and techniques related to the short-term versus long-term memory distinction; if such issues are not confronted, the kinds of extrapolations that are made concerning amnesic deficits may be overly simplistic.

Consider next Experiment 1 in a paper by Cermak, Butters, and Moreines (1974). These investigators contrasted performance of Korsakoff patients and controls on the release-from-proactive-interference paradigm (Wickens, 1970). In normal subjects, memory performance becomes increasingly depressed over successive trials on which to-be-remembered items from the same conceptual

category are presented; this effect is attributed to the buildup of proactive interference. Release from proactive interference is defined by a marked increase in performance when the category of to-be-remembered items is changed. Cermak et al. (1974) induced proactive interference by testing patients on four successive trials with consonant letter triads or with number triads and then examined performance on the release trial that followed the four buildup trials. Thus, four trials of letter triads were followed by a number triad, or four trials of number triads were followed by a letter triad. They found that both Korsakoff patients and controls benefited from the shift of category: Recall improved significantly on the critical trial in both subject groups. However, when Cermak et al. induced proactive interference by presenting four trials of word triads from a given *semantic* category (e.g., vegetables, animals, etc.) only the recall performance of control subjects increased on the critical trial on which words from a new category were introduced. Cermak et al. originally interpreted these data to indicate that Korsakoff patients do not properly *encode* the semantic features of stimuli; hence they are not affected when the category shift occurs and fail to show the typical release-from-proactive-interference effect.

Although the differential performance of Korsakoff patients in the alphanumeric and taxonomic release conditions cannot be denied, the "encoding" interpretation of Cermak et al. must be called into question, because of other data relevant to the Wickens paradigm. We refer specifically to an experiment reported by Gardiner, Craik, and Birtwistle (1972), who showed that even when release failed to occur under the standard conditions of the Wickens paradigm, presentation of appropriate retrieval cues at the time of recall could in fact elicit a release effect (see also O'Neill, Sutcliffe, & Tulving, 1976). It is therefore possible that Korsakoff patients may well have *encoded* the to-be-remembered words semantically, but simply failed to show the release effect owing to defective retrieval processes. Further evidence for this possibility has been presented by Cermak (this volume). But the point we wish to make in the present context does not concern "encoding" or "retrieval" explanations of amnesia. Rather, it is to indicate that it is sometimes necessary to temper interpretations of data generated by paradigms derived from experimental psychology in light of relevant literature.

Many students of amnesia, of course, are very much at home with paradigms of experimental psychology and put them to good use. An excellent example of a study that offers a critical and thorough analysis of the issues surrounding an experimental paradigm while using that paradigm to achieve new insights into amnesic dysfunction is found in the work of Kinsbourne and Wood (1975). Indeed, it would not be amiss to recommend that their paper might well serve as a model for future efforts to elucidate the nature of amnesic deficits using paradigms of experimental psychology.

Our critique so far has focused on various difficulties in the neuropsychological literature that we think should be confronted before a fruitful interaction with

experimental psychology of memory can be achieved. But we do not wish to imply that all or even most obstacles to the desired synthesis reside in the amnesia literature. A meaningful interaction between the two fields can only be brought about when difficulties stemming from the experimental psychology of intact memory are taken into account. Accordingly, we consider next one of the severe problems that students of amnesia are likely to encounter as they approach the literature on normal human memory and then turn our attention to some of the ways in which investigators of normal memory can make a positive contribution to the understanding of amnesia.

Confusion and Change in the Experimental Psychology of Memory

We have argued that students of amnesia should be aware of the complex issues surrounding paradigms and theories imported from the experimental psychology of memory. This admonition, however, covers only a small part of the problem, and it is now time to view the issue from the perspective of an investigator concerned with memory pathology. The neuropsychologist who seeks to employ the insights of memory research in an analysis of amnesia is confronted with a somewhat perplexing situation. He or she may be initially hopeful that psychological research can provide some firm, clear perspectives on human memory and so may approach the literature with the idea of using these insights to further his or her understanding of amnesia. But this optimism may quickly sour: The student of amnesia will discover that few theorists agree on the basic principles of memory functioning and that theoretical fashions change faster than the English weather. Techniques that look promising on initial publication are qualified, criticized, and complicated beyond all recognition within a few years, and they no longer appear capable of telling anything about amnesia. In short, a prospective user of theories and principles of experimental psychology will frequently find little that is straightforward and all too much that is ambiguous.

The dilemma has been well illustrated by the problem of short-term versus long-term memory and some of its theoretical offspring. Students of amnesia who began with the literature of the late 1960s concluded that there was much evidence favoring a separation of the two memory stores and justifiably began to contemplate ways in which this insight could apply to analyses of amnesia. But by the mid-1970s, the apparently widespread agreement on a multistore theory was almost entirely eroded, and it was soon replaced by a baffling variety of competing theories. Some neuropsychologists then noted the new interest in levels of processing and seized upon the ideas of Craik and Lockhart (1972) as a framework for exploring amnesia. But only 6 years later the depth of processing position was subject to widespread criticism (Eysenck, 1978; Nelson, 1977) and serious alternatives were made for its replacement (Baddeley, 1978). The issues surrounding the theory have become complex and muddled, and it appears no

longer to be applicable to the investigation of amnesia. This story is likely to be much the same in other important areas, such as the relation between recognition and recall, the nature of "context effects" in memory, and the similarities and differences between verbal and visual memory: The literature is characterized by a bewildering variety of theories, paradigms, and frameworks that do not converge upon any obvious consensual view (Tulving, 1979a).

What are investigators of the amnesic syndrome to do as they confront the spectre of everchanging theoretical and even metatheoretical diversity? How can they choose any one particular approach when there are a host of others that attack the problem in different and sometimes opposing manners? And why should they bother to invest any effort at all in a particular experimental paradigm or theory of memory, if it can be fairly certain that radical changes will soon occur or that the tradewinds of fashion will quickly blow in a new direction? These questions touch on some of the more troublesome problems that currently plague the experimental psychology of memory, and simple answers are not easy to come by.

The situation, however, is not entirely hopeless. There is at least one strategy that could be fruitfully employed by the investigators of amnesia who seek guidance from the experimental psychology of memory: Select a theory, framework, or paradigm that makes sense, that appears to be informative, and that looks potentially enlightening, and then be consistent in using it. Commitment to one definite point of view will make it easier to keep abreast of developments relevant to it and theoretical debates surrounding it. Under these conditions it will be easier to avoid some of the pitfalls described in the previous section of this chapter. By being aware of the issues surrounding a chosen vehicle of exploration of amnesia, and by using it consistently in light of these issues, investigators of memory disorders will be in a better position to make a positive contribution, not only to the understanding of amnesia, but to the more general theory of memory.

It is impossible to predict when the experimental psychology of memory will be sufficiently mature to offer students of amnesia a more stable picture of mnemonic function. In the meanwhile, there are plenty of potentially useful insights to be cultivated, and a consistent application of those ideas—as well as some patience with this occasionally chaotic field—is just about all that can be asked.

Strategies for Studying Amnesia: When are Normals Amnesic?

We close our analysis of some of the problems that seem to stand in the way of a productive integration of research on memory and amnesia by shifting our attention to investigators of intact human memory. What can these researchers do to facilitate communication with students of amnesia?

As we noted, one of the most conspicuous signs of the intellectual distance that still separates researchers of intact and disordered memory is the widespread failure of mainstream experimental psychologists to attempt theoretical explanations of empirical observations of memory dysfunction. Unfortunately, this problem cannot be solved simply by urging experimental psychologists to pay attention to what goes on in the pages of *Cortex* and *Neuropsychologia,* or by admonishing them to include amnesic data in the purview of their theoretical efforts. We are more inclined to believe that data from pathological populations will not receive widespread theoretical attention until difficulties such as the ones we have mentioned in this paper are overcome. As it now stands, many experimental psychologists are simply not sure how to interpret findings from experiments with amnesic subjects. Whatever experimental psychologists can do to help solve some of the difficulties is likely to be of benefit to the integration of the two fields.

A second way in which students of normal memory might contribute to the understanding of amnesia is by exploring the conditions under which normal subjects can be rendered amnesic. For instance, it is known that a number of pharmacological agents can impair the memory performance of normal subjects, including alcohol (Birnbaum & Parker, 1977), marijuana (Darley & Tinklenberg, 1974), and various anesthetics (Adam, 1973). The performance of intoxicated subjects in memory experiments is not usually examined or analyzed with an eye to elucidating clinical amnesias. What is needed are questions, and answers to questions, such as: What are the qualitative similarities and differences between intoxicated normals and clinical amnesics? Are there certain drugs that can mimic specific patterns of amnesic deficit? How far can such parallels be taken and how can we interpret them?

Because most psychological students of memory are not in a position to do pharmacological studies, these questions have to be explored in a logically similar but nominally different context, one in which patterns of amnesic deficit are induced through psychological manipulations in the memory laboratory. Are such inductions possible? If so, how do we interpret these deficits? We approach these questions by considering several relevant studies.

One such experiment has been reported by Tulving (1969) under the title of "Retrograde Amnesia in Free Recall." In this task, subjects were presented with common nouns, one at a time, at a relatively rapid rate, and were then asked for free recall of the list. Some of the lists contained one "high-priority event" defined as the name of a famous person—such as Freud or Columbus. Subjects had been told that whenever such a name occurred in the list they were to be sure to recall it. The results of the experiment showed that high-priority events were recalled almost perfectly. A more interesting finding, however, was that one or two words that had appeared in the list just before the high-priority event were recalled considerably less well than were words from the same serial positions in the control lists, which did not include any high-priority events. It looked as if

the occurrence of an event requiring special attention somehow produced an impairment of memory for immediately preceding events. This demonstration of "retrograde amnesia" in free recall has been explored further by Schulz (1971), Ellis, Detterman, Runcie, McCarver, and Craig (1971), Detterman and Ellis (1972), and Fisk and Wickens (1979), who have replicated the phenomenon, extended its generality, and considered various explanations of it. Detterman and his colleagues have also found evidence for "anterograde amnesia"—that is, depressed retention of items following the critical event.

Our use of quotation marks around the phrases "retrograde amnesia" and "anterograde amnesia" highlights one of the central issues at stake in these studies: To what extent do these laboratory-induced effects parallel the phenomenon of clinical amnesia? Detterman comments on this possibility following Experiment 3 of his 1975 study, in which he attempted to show that laboratory-induced amnesia empirically resembles clinical amnesia in a fairly specific manner. He noted that whereas clinical retrograde amnesia is characterized by recovery of lost memories over time, there is no such recovery in clinical anterograde amnesia. Accordingly, if laboratory-induced amnesia is to provide a reliable analogue of clinical phenomena, this pattern should be demonstrable with normal subjects. Detterman explored this possibility by letting his subjects hear 15-word lists under retention instructions, and induced amnesia with the critical word presented in the eighth position of the list; the critical word was rendered distinctive simply by presenting it at louder volume than the other words. Subjects attempted recall at delays of 0, 30, and 120 seconds in a between-subjects design. Detterman found that recall of words in the four serial positions following the critical word was depressed at all three retention intervals; that is, the anterograde amnesia did not improve with time. The picture was not so clear with the four items preceding the critical word. The magnitude of induced retrograde amnesia increased from the 0 to the 30-second conditions, before disappearing completely at the 120-second delay. Detterman pointed to the similarity between these data and the clinical observations of Yarnell and Lynch (1973). These researchers found that retrograde amnesia following concussion in football players was more profound some 3 to 20 minutes after the trauma than immediately following it, before gradually shrinking with further passage of time.

In a subsequent experiment, Detterman (1976) demonstrated that retrograde amnesia could be eliminated in his laboratory paradigm by presenting intralist retrieval cues, whereas anterograde amnesia was unaffected by cuing procedures. Thus, laboratory-induced retrograde amnesia was reversible and laboratory-induced anterograde amnesia was not—a finding that roughly, though not entirely, parallels clinical observations. Detterman (1976), though generally cautious in advancing claims for an underlying similarity between the two phenomena, did hypothesize that: "This empirical similarity between induced and clinical retrograde amnesia suggests that the same explanation may be appropriate for both [p. 624]."

Detterman's case rests on the observation of two types of similarity between laboratory-induced and clinical retrograde amnesia. First, there is a *formal* similarity between the two phenomena: In both cases, some critical event (e.g., perceiving an outstanding word, or a blow to the head) depresses memory performance involving preceding events. Second, there is the *empirical* similarity demonstrated by experiments. The theoretical problem of interest is to gauge how far these observations of similarity support the assertion that the two phenomena may be explained in the same terms. At an empirical level, it is clear that the analogy breaks down as soon as we examine the memorability of the critical event itself in the two situations: The actual traumatic event in clinical cases is almost never remembered, whereas the critical event in the laboratory situation is remembered best of all. Admittedly a blow on the head may have many consequences other than the depression of memory for the immediately preceding experiences, and it may be some of these other properties that are responsible for this difference between the laboratory analogue and the clinical amnesia. Nevertheless, the question arises as to whether this glaring empirical disparity vitiates any attempts to argue seriously for a genuine similarity between the two phenomena.

We think that the answer to this question depends on the purposes of the experimenter in studying laboratory-induced amnesia. If he wishes to establish some isomorphic laboratory analogue of clinical amnesia that can be used as a device for testing hypotheses about clinical phenomena, then difficulties such as the one previously noted are indeed imposing ones. It makes little sense to speak of the clinical phenomenon and its laboratory analogue as the "same" thing when there are obvious empirical discrepancies between them. There are no extant criteria that can tell us exactly when we have successfully modeled a clinical phenomenon in the laboratory; we do not know how to gauge the significance of the various empirical dissimilarities that would undoubtedly appear in the course of such attempts. A similar problem was acknowledged some years ago by Rapaport (1950) in his critique of the attempts made by Ray (1937) and Sears (1936) to explain retrograde amnesia in terms of laboratory-induced retroactive inhibition: "These attempts at comparison fail to take cognizance of the complexity of amnesia, and were satisfied to state the obvious but superficial analogies of two phenomena which were known to different degrees, described in different terms, and observed in different settings by people of different backgrounds [p. 197]." Until suitable criteria are found, attempts to build laboratory analogues that can serve as testing ground for theories of amnesia seem to be of questionable value. (See the exchange between Weiskrantz and Warrington (1975), and Woods and Piercy (1974), for an example of the serious problems with this approach.)

There exists, however, a second approach to the use of laboratory analogues of amnesia that may be less devastated by the difficulties we have mentioned. Rather than trying to *explain* clinical amnesias by means of laboratory

analogues, it may be more useful to use such analogues as devices for *generating hypotheses* that later can be tested directly in the clinical arena. One of the major advantages of studying normal college-age subjects is that they are in plentiful supply and permit the investigator greater range and flexibility in exploring new ideas than does the study of clinical patients to whom access may be difficult. The experimental psychologist who can construct a laboratory situation that captures the major features of a given clinical amnesia would be in a position to explore a greater range of theoretical possibilities than his counterpart working within the tight restrictions imposed by the practical realities of clinical research. The absence of criteria that could be used for establishing the degree to which laboratory-induced and clinical amnesia are similar could not, of course, be completely avoided. However, because the goal of the research we have suggested here is to generate hypotheses that could be tested in clinical domains—and not to argue that a laboratory phenomenon can "explain" a clinical one, or that the two are somehow the "same thing"—the problem of interpreting differences between laboratory simulations and clinical reality would be a less serious one.

THE AMNESIC SYNDROME: POSSIBLE DIRECTIONS FOR FUTURE RESEARCH

In this last section of the chapter, we speculate briefly about the future of research on amnesia. If there are mutual benefits to be derived from a closer integration of psychological study of normal and pathological memory, what should be the focus of this integration? In earlier sections of the chapter we talked about some of the problems that need to be solved; here we briefly mention two possibilities for psychological theories of amnesia that appear to hold promise.

Interaction between Encoding and Retrieval Processes

The first possibility consists of adopting a general framework for psychological analysis of memory that clearly recognizes the interaction between encoding and retrieval processes as the fundamental determinant of "goodness" of memory. The second possibility takes the form of further exploration of the idea, proposed in slightly different forms by a number of investigators, that the essence of the amnesic syndrome lies in the impairment of episodic rather than semantic memory. We discuss these two matters in turn.

The general framework for psychological analysis of memory that holds interaction between encoding and retrieval operations to be of fundamental importance in determining memory performance has been discussed at greater length elsewhere (Tulving, 1976, 1979b); we present only its summary here. Of particular relevance to amnesia research are two assumptions of this framework. First,

"goodness" of the original experience, encoding, study, or "consolidation," cannot be specified in absolute terms, independently of the conditions that will prevail at retrieval. Numerous experiments have demonstrated that a "strong" memory trace of an item or event need not produce a higher level of memory performance under all conditions. Depending on the conditions of retrieval, a memory trace that appears to be "weaker" in one situation may produce a higher level of retention performance in another. Second, differences in the effectiveness of retrieval cues cannot be specified in absolute terms, independently of the conditions under which the original experience occurred or was registered. Again, many experiments have been reported whose results have shown that the effectiveness of retrieval cues is critically dependent on the manner of encoding of to-be-remembered events at the time of their original occurrence.

To the extent that these two assumptions can be thought of as rather general ones, holding for all instances of remembering, they are incompatible with widely held ideas about the "locus" of the amnesic deficit lying in either the storage (encoding, "consolidation") or the retrieval processes. (For a crisp review of these theories, see Rozin [1976].) If it is true that "goodness" of encoding conditions can only be specified in relation to retrieval conditions, and effectiveness of retrieval cues specified only in reference to specific encoding conditions, then it does not make much sense to think of amnesic deficit as a consequence of only inadequate encoding or consolidation, or only as reflecting the lack of availability of appropriate retrieval cues or retrieval information. The amnesic deficit, like all other phenomena of memory, needs to be understood in terms of the joint effects of encoding and retrieval processes.

The evidence most directly relevant to the proposition that it is the interaction between encoding and retrieval operations and processes that determines memory performance is derived from what we call encoding/retrieval experiments. This is a type of memory experiment in which both study and test conditions are experimentally manipulated in an orthogonal design. The nature of to-be-remembered materials and other well-known determinants of memory performance can either be held constant or varied as additional dimensions. Let us look briefly at two illustrative examples.

In Experiment 2 of the series reported by Fisher and Craik (1977), subjects studied two types of paired associates. In one type, the *A* member of the pair rhymed with the *B* member (e.g., *hat*—CAT), whereas in the other type, the *A* member was semantically associated with the *B* member (e.g., *dog*—CAT). Subjects were instructed to relate the *B* word (the target) to the *A* word (the cue) at the time of study. At the time of the test, half the target words from rhyme pairs were tested with rhyme cues, whereas the other half were tested with semantically associated cues; the same arrangement held for testing of target words from the associatively encoded pairs. Thus the design was one in which two encoding conditions (rhyming or associative encoding) were crossed with two types of retrieval information (rhyming or associatively related words). The

results of the experiment showed a crossover-type interaction between encoding and retrieval conditions: Targets from rhyme pairs were better recalled to rhyme cues (26%) than to associative cues (17%), whereas targets from associative pairs were better recalled to associative cues (44%) than to rhyme cues (17%). These data make clear that the goodness of encoding conditions—are rhyme-encoded or associatively encoded words better remembered?—could be expressed only relative to the type of cue presented at retrieval: In either encoding condition, memory performance was higher when the cues matched encoding than when they did not.

An earlier experiment reported by Barclay, Bransford, Franks, McCarrell, and Nitsch (1974, Exp. 1) makes the same point in an even more dramatic fashion. Subjects in this experiment heard 10 sentences in which a target noun was embedded in a context that emphasized specific properties of that noun. For example, subjects in one group might hear the sentence, "The man tuned the PIANO," whereas the subjects in another group would hear the same target noun in a different sentence, "The man lifted the PIANO." The subjects in both groups subsequently were presented with two types of retrieval cues for target nouns (e.g., PIANO); these cues either matched or did not match the meaning of the target noun as suggested by the intrasentential context. Thus, for the sample sentence, the two cues were "something heavy" and "something with a nice sound." Again, in the design of the experiment two encoding conditions were crossed with two types of retrieval information. The results of the experiment showed a strong crossover interaction between encoding and retrieval: The effectiveness of a cue such as "something heavy" for the target noun PIANO was much greater following the appropriate encoding ("The man lifted the PIANO") than following an inappropriate encoding ("The man tuned the PIANO"); the effectiveness of the cue "something with a nice sound" was exactly the reverse. These data, like those of Fisher and Craik, cannot be understood by thinking of one encoding condition being "better" than the other, or one kind of retrieval information being more "effective" or "useful" than the other: Memory performance in this experiment clearly depends on both the nature of encoding operations and the nature of the available retrieval information.

The experiments described are just two of the many that demonstrate encoding/retrieval interactions; we could also cite the work of Dong (1972), Goldstein, Schmitt, and Scheirer (1978), Lauer (1974), Morris, Bransford, and Franks (1977), Thomson and Tulving (1970), Tulving and Osler (1968), and others.

How does the acceptance of the importance of the interactive relation between encoding and retrieval conditions modify specifications of the amnesic deficit in terms of either storage processes alone or retrieval processes alone? For a concrete illustration, consider the well-known experiment by Warrington and Weiskrantz (1970). In this experiment Warrington and Weiskrantz demonstrated that amnesics can differentially benefit from fragment cues at the time of testing:

Although the performance of the amnesics was much below that of controls on a Yes-No recognition test, it was indistinguishable from that of the controls when fragment cues were made available to both groups of subjects. The original interpretation of these data was recently reaffirmed by Weiskrantz (1978) as follows: "This carries the strong implication that the amnesic deficit lies with mechanisms beyond the initial input of the items into storage, and obviously suggests a difficulty with retrieval [p. 378]." The idea is that the low level of retention performance of the amnesics in the Yes-No recognition test cannot be attributed to inadequate encoding or consolidation: Fragment cues produce a high level of recall based on the same "inadequate" storage conditions. We agree with Weiskrantz's conclusion, but we also assume that the fragment cues were effective, for the amnesics, and copy cues in the Yes-No recognition test ineffective, not only because of differences in the two types of test but also because of the particular storage format of to-be-remembered items. Although the design of the Warrington and Weiskrantz experiment did not include different encoding conditions and therefore does not provide relevant evidence, we assume that their data reflect the same kind of interaction between encoding and retrieval as that revealed in, say, the Barclay et al. (1974) experiment. Thus we think that the difficulty that the amnesics had with retrieval in the Yes-No recognition test should be understood in terms of both what was stored and how it was to be retrieved, and not in terms of the latter factor alone.

The dichotomy between "storage phenomena" and "retrieval phenomena" in research on normal as well as on pathological memory may have served a useful purpose at one time, but it is no longer theoretically defensible. Indeed, a number of students of amnesia have concluded on the basis of their own observations that both storage and retrieval processes are impaired in amnesia (Cermak, this volume; Fuld, 1976; Marslen-Wilson & Teuber, 1975). These conclusions, together with the lessons that can be learned from encoding/retrieval experiments with normal subjects, should encourage all students of amnesia to seek understanding of their object of interest in the interaction of encoding and retrieval conditions.

Episodic and Semantic Memory

Let us turn now to the second "possibility" for a psychological theory of amnesia. This is the idea that amnesia entails impairment of episodic and not of semantic memory. The fact that amnesic patients have particular difficulties remembering their personal experiences, in contrast with their relatively normal language system and intact knowledge of the world, has been noted by various students of amnesia, but it is only in relatively recent times that the idea has gained prominence in theoretical thinking about the nature of the amnesic syndrome.

Although Korsakoff (1889/1955) described the syndrome now named after him as a loss or impairment of the feeling of personal familiarity with recalled

events, he did not concentrate on this characteristic of the disease. Claparède (1911/1951) did. He distinguished between two kinds of mental connections: those established between and among representations, and those established between representations and the "me," the rememberer's personal identity. Korsakoff's syndrome, Claparède said, is a pathological disorder of egocentric connections between representations and the "me." Claparède's Korsakoff patient was capable of learning new things—how to find her way around in the hospital, that nurses are useful sources of information about the hospital routine, or that "sometimes pins are hidden in people's hands"—what she could not do was remember her own experiences after the onset of the illness. The discrepancy between this patient's apparent ability to retain some of the semantic contents of a recently experienced episode and her inability to locate this episode in her personal past was clearly illustrated by Claparède (1911/1951):

> When one told her a little story, read to her various items of a newspaper, three minutes later she remembered nothing, not even the fact that someone had read to her; but with certain questions one could elicit in a reflex fashion some of the details of those items. But when she found these details in her consciousness, she did not recognize them as memories but believed them to be something "that went through her mind" by chance, an idea she had "without knowing why," a product of her imagination of the moment, or even the result of reflection, [p. 69].

This distinction between what we now call episodic and semantic memories was also made by Nielsen (1958), as pointed out by Crovitz (1977). Nielson used the term "temporal amnesia" for a loss of autobiographical memories and the term "categorical amnesia" for a loss of memory for learned facts. He also argued that categorical (semantic) amnesias represented a dysfunction of cerebral hemispheres, whereas temporal (episodic) amnesias were related to lesions of the hippocampus.

More recently, Kinsbourne and Wood (1975) have presented considerable evidence in support of their argument that amnesic forgetting represents a selective episodic memory deficit, with relative sparing of semantic memory. They proposed that: "all instances of amnesic forgetting can fit into the category of episodic memory [p. 281]"; they also speculated about a possible neuroanatomical correlate of the distinction between episodic and semantic memory. Their general theme is further pursued and elaborated by Wood and Ebert in their chapter in the present volume.

Some recent ideas put forward by Gaffan (1976) likewise suggest a special disturbance of episodic memory in the amnesic syndrome. Gaffan has argued that: "the deficit of human amnesics in recall is due to their impairment in familiarity discrimination [p. 240.]" When amnesic patients are confronted with a list-learning task, Gaffan contends, they may be entirely capable of making responses at the time of recall that provide evidence of retention of semantic content from the learning episode. But they may lack the experience of episodic

familiarity with these responses and are consequently unable to discriminate between appropriate list items and inappropriate intrusions. Rozin (1976), too, has proposed that the "most central feature" of all amnesic syndromes is: "the loss of feeling of familiarity on either exposure to or recall of previously experienced events [p. 36]," or, in other words, loss of episodic memories. He listed a number of specific phenomena that are generally accepted as characteristic of the amnesic syndrome and suggested that a "modified retrieval theory," which is centered on amnesia as impairment of episodic memory, gave a better account of the phenomena than other extant psychological theories. Among other things, Rozin points out, the idea helps us to understand repeated failures to find amnesic syndromes in infrahuman subjects, inasmuch as: "it is likely that they do not have a personal reference system in the first place [p. 37]."

The difference between the relative prevalence of the amnesic syndrome in humans and its absence in animal experiments has prompted Weiskrantz (1977, 1978) to suggest that the central feature of amnesia in human beings lies in the "striking dissociation between the subjects' commentaries and their objective performance [1978, p. 385]" regarding their recently learned tasks. Weiskrantz suggests that amnesia represents a state of dissociation between levels of processing—that is, amnesics may lack the experience of remembering even when they show objective evidence of learning—a suggestion akin to those based on the distinction between episodic and semantic memory.

We like the idea that the amnesic syndrome represents primarily a derangement of episodic rather than semantic memory, for at least four reasons. First, because of its sound agreement with clinical observations and empirical facts, it commands almost universal acceptance by students of amnesia, and thereby represents one of the few valid generalizations about the otherwise complex and highly variable syndrome. Second, the idea may provide an important theoretical link between cases of organic amnesia, with which we have been concerned in the present chapter, and the so-called functional amnesias of hypnosis, hysteria, fugue states, and multiple personality. Various early 20th-century clinical investigators of functional amnesia noted that their patients frequently could report the semantic contents of recently experienced events even though they could not identify these events as part of their personal past (Janet, 1901, p. 103; Prince, 1910, p. 265; Sidis, 1914, p. 275). Recent research on hypnosis has uncovered a similar phenomenon known as *source amnesia* (Evans, 1979; Evans & Thorn, 1966). Source amnesia can be produced by asking hypnotized subjects questions about little known facts and then providing them with answers to items that they answer incorrectly. When subjects are later asked these same questions after they have emerged from the hypnotic state, a significant proportion of them can provide the correct answers but cannot remember the hypnotic episodes during which they acquired them. Kihlstrom and Evans (1979) have explicitly characterized source amnesia as: "perhaps the most striking example of the dissociation of episodic and semantic memory in posthypnotic amnesia [p. 142]" Although

the critical variables underlying the phenomenon are not yet understood, and the extent to which source amnesia is characteristic of other functional amnesias has not been systematically explored, the possibility that episodic and semantic memory are differentially impaired in both functional and organic amnesia may provide an important basis for formulating a unified theoretical approach to these two general classes of memory pathology.

Third, the idea may well turn out to be an important stimulant of research on episodic memory done by experimental psychologists. So far there is little in either experimental or theoretical literature on normal episodic memory that has been directly concerned with rememberers' feelings of recollected events as "coming from" their personal past. The behavior of amnesic patients suggests that one cannot make inferences about intactness of such feelings simply on the basis of an experimental subject's ability to make a correct response in a memory task, even if it is known that the origin of the response must have resided in a unique learning episode. When experimental psychologists come to grips with the problem of personal familiarity and feelings of "pastness" of recollected experiences, they will have taken a step closer to the integration of research on amnesia and memory.

Finally, we like the idea, because it makes it possible to think of amnesias that result from generalized brain malfunction, such as concussion and senility, as a special case of a general pattern of neurological and mental development. The pattern is defined by the temporal sequencing in loss and recovery of function that was described by Ribot (1882) for cases of generalized brain disease. Ribot began with the observation that in amnesia, recent memories are more impaired than remote memories; this observation is known today as "Ribot's law." But he went on to place his law in a much wider biological panorama:

> This law, general when applied to memory, is only one phase of a still more general law in biology. It is a well-known fact in organic life that structures last formed are the first to degenerate.... The law which we have formulated is only the psychological expression of a law of life, and pathology shows in its turn that memory is a biological fact [p. 127].

Ribot, then, contended that the probability that a function is impaired or lost in brain trauma is inversely related to its stage of acquisition in the developmental or maturational sequence; in recovery, the sequence is reversed, with functions acquired earlier recovered before those acquired late. Rozin (1976) has recently given us a modern version of these ideas in a Jacksonian (1884) evolutionary framework applied specifically to memory: "I believe that those aspects of memory that appear first in evolution also develop first and usually disappear last in disease [p. 16]."

How, then, does this general pattern of ontogenetic and phylogenetic development—what might be characterized as the principle of "last in-first

out''—relate to the differential impairment of episodic and semantic memory in amnesia? We believe that episodic memory represents a later stage of development than semantic memory; in other words, that episodic memories ''grow out'' of semantic knowledge (for similar notions, see Claparède, 1911/1951, p. 59; Kinsbourne & Wood, 1975, p. 284; MacCurdy, 1928, pp. 126-127; Schachtel, 1947, pp. 14-15). Semantic knowledge, according to this hypothesis, is one of the necessary conditions of episodic remembering. Another necessary condition is the person's awareness of his or her self, what Claparède called ''the continuity and personal character of consciousness [p. 67]'' or the ''me-ness'' to which facts must be associated in order for them to become personal memories. This developmental sequence—semantic memory preceding episodic memory—may hold both phylogenetically as well as ontogenetically. The relatively late phylogenetic development of episodic remembering has been suggested by Baldwin (1906, pp. 302-305), Claparède (1911/1951, p. 59), and more recently by Kinsbourne and Wood (1975, p. 284). The possibility that episodic memory represents a late stage in a human being's mental development permits us to understand its greater vulnerability in generalized brain disease and thereby to place the central phenomenon of the pathological condition known as amnesia in a broader biological perspective.

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