Chapter 1

Modularity and Neuropsychology: Modules and Central Processes in Attention and Memory

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Fodor's 1983 monograph, The Modularity of Mind, *rekindled the centuries-old debate on the nature of mental faculties and how they are represented in the brain. Cognitivists and neuroscientists continue to argue the merits of Fodor's twofold distinction between modules and central systems. Here Moscovitch and Umilta examine the distinction from the perspective of cognitive neuropsychology and find it wanting. Their reformulation expands the possibilities for modular organization and suggests that if we want to carve up central systems at the joints, so to speak, we should begin by looking at the functions they serve as integrators and modulators of modular systems.*

Central systems are clearly important in the functional characterization of dementing disorders. Indeed the term dementia is reserved for conditions that heavily affect memory, generic knowledge, and attention, prototypically non-modular systems in Fodor's typology or anyone else's. Moscovitch and Umilta provide detailed models of the neuropsychology of attention and memory to show how the functions we attribute to each, and the deficits associated with them, are based on the delicate interplay of modular and central processes, involving neural networks that are widely distributed in the brain. Degenerative diseases, which have a tendency to disturb cortico-cortical and cortico-limbic connectivity (see Damasio, Van Hoesen, and Hyman, chapter 3), naturally have a disruptive effect on integrative systems of this type.

But the impact of degenerative dementia is not limited to these central systems. The disorders detailed in subsequent chapters of this book implicate more specialized mental faculties, such as those that locate visual arrays in space or interpret words and pictures as semantic tokens. How are these faculties to be understood within the taxonomy of modular systems? Moscovitch and Umilta's answer draws on the notion of assembled modules, whose domain is broad but circumscribed and in whose operation central systems play a definite, albeit constrained, role.

These and other aspects of Moscovitch and Umilta's thesis come up again in following chapters and chapter notes. One point worth emphasizing here is their contention that negative evidence from dementia in some cases constitutes the most compelling evidence for encapsulation of modular systems.

Thus in patients who have serious limitations in retrieving and manipulating information in memory, particularly semantic- or generic-type memory, the continued preservation of functioning within a domain like object identification limits the role that can reasonably be attributed to top-down influences. Considerations of this type figure prominently in other chapters in this book (especially chapters 7 and 8).

M.F.S.

In computer science there are two ways of solving computational problems: One is to design a general purpose processor that can deal with a wide range of data. The second way is to design a stupid, but highly efficient, specific processor that can operate only in a very restricted domain. For example, to read postal codes or bank account numbers, you can build a machine that can read anything or a device that can decode only digits and letters written in a certain format in a particular location.

Nature is faced with similar problems in designing nervous systems. In lower organisms there are many examples of the second type of solution. In humans it has generally been accepted that such rigid "stupid" systems have given way to general purpose processors that can handle any stimulus that is within its sensory capacities and that can use that information to guide a virtually unlimited range of behaviors (Rozin 1976, Sherry and Schacter 1987). A few people, however, have claimed that domain-specific processors continue to exist for higher processes, even in humans. The most obvious examples are those for speech (Liberman and Mattingly 1985) and possibly for face perception (de Schonen, Gil de Diaz, and Mathivet 1986, de Schonen and Mathivet 1989, Yin 1970). Except for Rozin (1976) no one had proposed that these mechanisms may be fundamentally similar to the stupid and "devoted" processors found in lower organisms. It remained for Fodor (1983), however, to make the strongest case that much of what passes for higher-order cognitive processing in humans is of this stupid sort (in Fodor's words it is *modular*) and that it coexists with more central processes on which intelligent behavior depends.

In the first section of this chapter, we briefly sketch what we believe is the current neuropsychological approach to modularity and evaluate Fodor's criteria for modularity from a neuropsychological perspective. To do so, we first make some specific proposals as to what would constitute neuropsychological evidence for Fodor's criteria for modularity. Based on this critique, we offer an alternative view of

modularity and apply it to some neuropsychological data. The third section presents a view of central processes and their functions that is consistent with the neuropsychological literature. In the fourth section we apply the framework we have developed of a coordinated system of modular and central processes to problems of memory and attention.

Current Neuropsychological Approach to Modularity

Double Dissociation

In neuropsychology the typical approach used to relate structure to function is *double dissociation* (Teuber 1955). In this paradigm lesions to area A are associated with deficits to function A, but not function B (or C or D . . .), whereas lesions to area B are associated with damage to function B but not A. (Often it is implicitly assumed that double dissociation exists even in a single case study because one knows that other single cases with different lesions will spare the function in question, but affect another function.) This type of evidence has been used to argue that function A or B is modular (for example, Shallice 1981, 1988). However, double dissociation, or lesion evidence in general, is neutral with regard to modularity as defined by Fodor. It could just as easily be two central processes that are doubly dissociated as two modular ones.

Showing a double dissociation is just the first in a series of steps that the investigator would have to follow to use neuropsychological evidence in favor (or against) modularity. What is necessary is to examine the evidence to see if it conforms to accepted criteria of modularity. As we argue, it is not always possible to determine from the data whether these criteria apply, and sometimes it is impossible in principle to do so.

Characteristics of Modules and Central Processes

Modules are computational devices that receive input, transform it as a result of the computations performed on it, and emit an output. These are common features of all computational devices that are accepted by information processing theories and models and are not unique to modules. What distinguishes one type of computational device from another is the type of input that each accepts, the type of computations it performs, and the nature of the output it emits. Because the criteria for distinguishing modules from other computational devices were set down most explicitly by Fodor, we refer to his criteria in our discussion. According to Fodor, "modular cognitive

systems are domain specific, innately specified, hard-wired, autonomous, and not assembled" (1983, p. 37). They are informationally encapsulated, their processes are mandatory and rapid, and their output is shallow. They follow a characteristic pattern of development and deterioration. These modules are not mere computational devices, but have informational or propositional content. Many of these properties are necessary if perceptual modules are to fulfill their function, which is "to represent the world and make it accessible to thought" (p. 40).

Central processes are the antithesis of modular systems. They are "slow, deep, global rather than local, largely under voluntary (or, as one says, "executive") control, typically distributed with diffuse neurological structures, [and] characterized by computations in which information flows in every which way. Above all they are paradigmatically unencapsulated; the higher the cognitive process, the more it turns on the integration of information across superficially dissimilar domains" (Fodor 1985, p. 4). Central cognitive systems are thus concerned with knowledge that can be, and often is, inferential. It is knowledge concerned with, and influenced by, the fixation of belief.

Nonessential Characteristics of Modules

Even a superficial examination of the criteria used to distinguish between modular and central cognitive systems, however, indicates that many of the criteria that distinguish them do not apply. Thus deployment of attention, which can be mediated by a central process, can be both mandatory and rapid (Jonides 1981, Umilta 1988b). To take an obvious example, it is probably impossible not to attend immediately to one's name if it is spoken loudly or to a sudden flash of light or movement in the periphery.

Three other features that also seem not to be unique to modules are (1) association with a fixed neural architecture, (2) manifestation of characteristic and specific breakdown patterns, and (3) a characteristic pace and sequencing during development. The neuropsychological literature is replete with cases in which functions mediated by central processes, such as memory, attention, and problem solving, are impaired after focal brain damage, which suggests that these functions are closely linked to the operation of circumscribed neural structures (Shallice 1988, Walsh 1988). Moreover, the idea of a fixed neural architecture does not necessarily imply a focal location, but can also be realized by a distributed neural network whose components are nonetheless fixed. Memory and attention also have a characteristic breakdown pattern (Butters and Miliotis 1985, Heilman, Watson, and

Valenstein 1985) and a regular developmental sequence (Cohen and Salapatek 1975). Both short- and long-term memory are known to alter their capacity and mode of operation respectively during development and to undergo predictable modification after brain damage, fatigue, or intoxication (Case, Kurland, and Goldberg 1982, Frank and Rabinovitch 1974 a, b).

The distinction between central and cognitive systems does not depend on these criteria; other criteria are much more critical. It is to these essential criteria that we now turn. As we shall see, these too have their problems.

We are not the first to note the deficiencies with Fodor's conceptions of modularity. The interested reader is referred to excellent critiques by Schwartz and Schwartz (1984), Marshall (1984), Shallice (1984), and the respondents to Fodor's 1985 article. Instead, we wish to indicate how these criteria can be translated (operationalized) in neuropsychological terms. What, if anything, would constitute neuropsychological evidence in favor of or against each of the criteria and, by implication, in favor of or against a particular view of the modular organization of mind? Because this book is concerned with both modularity and dementia, we wish to emphasize the special place that evidence from dementing patients holds in evaluating these criteria, identifying modules, and distinguishing them from central cognitive systems.

Essential Characteristics of Modules

Domain Specificity Each module operates only in a restricted domain. The information a module receives and processes and the hypotheses it projects are highly circumscribed. It cannot accept or deliver information about anything outside its domain. At the neuropsychological level domain specificity is linked to localization of function. Indeed, one interpretation of the results of a double dissociation experiment might be that damage to each region is associated with deficits in a domain that is specific to that region.

There is a problem at both a conceptual and neuropsychological level in defining the domain and determining exactly how restricted it is. A domain can be specified only after one has a theory about the function a module serves and the kind of input it accepts. In the absence of such a theory, it is often difficult to evaluate the neuropsychological evidence. This point can be illustrated with reference to speech perception, where the domain is clearly specified by theory. According to Liberman and Mattingly (1985, 1989), the domain of a speech module is a speech gesture—not a particular set of acoustic features,

but the intended gesture that the waveform conveys. This means that the same information can be conveyed not only through sound but also through vision, as in lip-reading (McGurk and MacDonald 1976). At the psychological level this is clearly shown by the McGurk effect, in which the phoneme subjects perceive is a combination of the phoneme they hear and the different phoneme that they simultaneously see being mouthed. Neuropsychologically, this theory suggests that the same structure mediates both speech perception and lip-reading of speech, but no other information. Campbell, Landis, and Regard's (1986) recent experiments on lip-reading in aphasics confirms this prediction. Had the theory specified the domain as one restricted to the acoustic waveform of speech, the experiment by Campbell and colleagues would have been interpreted as evidence against either domain specificity or a particular theoretical instantiation of it to speech.

In this context it may be argued that the neuropsychological deficits of patients with brain damage may provide the best estimate of what the domain of particular modules might be. Unfortunately, few of the deficits ever appear in isolation, as a strict criterion of domain specificity would require. Aphasias, for example, are typically accompanied by deficits in domains that ostensibly have little to do with language (Benson 1985, Kimura 1982). One can always argue that the constellation of deficits one observes results from a lesion that is too diffuse and encompasses more than one functional area (that is, more than one module). Patients in whom, by chance, the lesion seems to be extremely specific and produces the highly circumscribed deficit that the module predicts are rare; yet they sustain our belief in domain specificity even where, as in aphasia, there are no published reports, to our knowledge, of patients without other accompanying cognitive deficits.

Even when some well-documented neuropsychological deficits do appear in relative isolation, it is often difficult to specify the domain involved or even if the structure that was damaged was modular. Consider, for example, visual object agnosia on the one hand and amnesia and hemi-inattention on the other. Whereas object recognition is considered by Fodor to be a modular process, memory and attention are prototypical horizontal faculties that are mediated by central processes. Yet from a neuropsychological perspective there is little to distinguish between, say, amnesia, neglect, and agnosia with regard to the issue of domain specificity. All are caused by focal lesions and can occur in relative isolation from other cognitive deficits. Whereas agnosias are typically modality specific, memory disorders can be material specific (verbal or nonverbal, motor or cognitive—

Milner 1974, Squire 1987) in much the same way that deficits in identifying speech gestures are material, rather than modality, specific. Admittedly, the domain of a memory module may be difficult to specify, but, then, without an adequate theory, so is the domain of any complex, cognitive module. What is the domain of a visual object recognition module? All possible objects, even those not yet invented? Some integral feature input? What makes Fodor and others willing to accept that object recognition is modular, yet reluctant to concede that memory is modular, is probably less a concern about domain specificity than about other essential criteria, such as information encapsulation.

Before we discuss information encapsulation, let us anticipate the argument we will make with regard to both memory and attention. The reason that memory and attention appear to behave as vertical faculties under some circumstances and as horizontal ones under others is that they are mediated by both modular and central cognitive systems. Damage to the modular parts produces a deficit that is as domain specific as damage to a speech gesture or object-recognition module. In fact it is precisely because it is a modular process that is damaged, rather than a central one, that the deficit can be so circumscribed. Where memory and attentional impairments are caused by damage to a central processor, they are accompanied by a more general cognitive impairment that affects other functions (or faculties) in the same way.

Information Encapsulation: Evidence from Dementia Information encapsulation refers to the resistance of modular processes to top-down cognitive influences. In other words general knowledge of the world cannot affect the operations and outputs of modules—"They go off largely without regard to the beliefs and utilities of the functioning organism" (Fodor 1985, p. 3). Visual illusions and impossible figures persist despite all attempts to have the image conform with the way we know the world must be organized (Kanizsa 1979).

Whereas the idea of domain specificity has figured in neuropsychological investigation, the concern with informational encapsulation is new. The traditional approach of studying patients with focal lesions is not applicable in this instance. If function A is absent, then it makes no sense to try to determine whether the absent function is informationally encapsulated. To test adequately for informational encapsulation, the function under question would have to be intact.

How could questions related to informational encapsulation be framed in neuropsychologically relevant terms? We propose that this issue resembles one that has been taken into account by neuro-

psychologists and in fact gave rise to the method of double dissociation. The question is related to whether neural damage causes general intellectual impairment or a specific loss of function. By and large, until recently, neuropsychologists interested in localization of function have not been concerned with conditions that lead to a general intellectual deficit. Yet it is precisely this kind of evidence that is critical to the issue of informational encapsulation. If a particular function remains intact despite evidence of gross intellectual loss, one can assume that it is informationally encapsulated because the malfunctioning of a central cognitive system, which is the repository of general knowledge, has no effect on that function. A particularly striking example is the patient reported by Geschwind, Quadfasel, and Segarra (1966) who had her hearing and speech areas isolated from the rest of the cortex. This patient could repeat speech and even correct grammatical errors without any ostensible degree of understanding (though the report of the case is not precise regarding just how much intellectual ability remained). Other examples come from patients with Alzheimer's disease who, despite gross intellectual deficits, can continue to read, to repeat and correct grammatically incorrect sentences (for example, Schwartz, Marin, and Saffran 1979) and, in one rare case, to match different views of particular objects without any idea of what the objects were (Moscovitch, personal observation).

A problem with using residual capacities (sparing of function) in the face of generalized intellectual loss as an index for informational encapsulation is that the conditions that gave rise to the general intellectual deficit are likely also to affect each particular module. Even if some modules are not affected, the patient's inability to follow instructions may make it impossible to evaluate the state of the modules. For these reasons, failure to find that a particular modular function is spared, in the face of general intellectual loss, does not constitute evidence against informational encapsulation. Only positive evidence counts.

Bearing this proviso in mind, we suggest the following: *If domain specificity and information encapsulation are the primary characteristics of modules, then neuropsychological evidence both of double dissociation in patients with focal brain damage and of sparing of function in dementia are necessary for establishing that a cognitive system (or process) is modular.* Taking recognition of noncanonical views of objects as an example, evidence from patients with focal brain damage establishes a domain-specific deficit related to perception of those objects (Warrington and Taylor 1973, 1978), whereas evidence from patients with dementia establishes that the operation of that object-recognition module can be normal despite marked changes in cognition.

Shallow Output The output of a module is shallow if it is not already semantically interpreted, but is instead confined to domain-specific features. That is, the output cannot convey any information about either how that output was derived or the relation of that output to our general knowledge of the world. Massaro's (1986; p. 440) explanation of shallow output of a language module captures its essence: We can all agree on what was said, but we can argue forever about what was meant.

What constitutes neuropsychological evidence of shallow output? We propose that it is evidence from patients whose lesion prevents them from interpreting and commenting semantically on the information that they had processed successfully in a specific domain. Ideally the evidence should be obtained from demented patients about whom there is little doubt that their ability to interpret and comment on their perceptions is severely impaired. In short, the ideal patient is one whose central processes are so impoverished that it is reasonably safe to assume that performance is determined primarily by modular output. If demented patients are not available, evidence can be obtained from patients with circumscribed deficits that satisfy the criteria we have proposed.

The relevant neuropsychological evidence can be found in two classes of studies: those using explicit tests of knowledge and those using implicit tests (Graf and Schacter 1985, Schacter 1987b). An explicit test is one in which relevant information regarding an aspect of the subject's knowledge is gathered directly and with the subject's awareness. On implicit tests the relevant information is inferred indirectly from the subject's performance and often without the subject's awareness that a particular aspect of knowledge is being tested. For evidence from explicit tests, let us return to the demented patient who matched objects for identity even when they were presented from noncanonical viewpoints. This ability was almost perfectly intact despite the patient's having no conscious awareness, irrespective of the methods we used for testing, of what the object was, to which taxonomic category it belonged, or what function it served. Thus although she matched noncanonical views of objects almost perfectly, the patient performed at chance on the much simpler task of matching different examplars of dogs, cats, and fish to prototypes of each. On the basis of her performance, we conclude that the patient retained an intact module for representing objects in 3D (Marr 1982, Ratcliffe and Newcombe 1982) and that the shallow output of the module was "pictorial" nonsemantic and highly specific to that object—toaster A or toaster B, but not a generic toaster (as Fodor (1983) proposed).

Other examples of performance determined only by such shallow output are not difficult to find. Demented (and nondemented) patients are reported who can read with no understanding (Schwartz, Saffran, and Marin 1980); there are patients who can judge the grammaticality or well-formedness of sentences, and even correct faulty grammar when repeating the sentence, but not assign meaning to the syntactic structure (Linebarger, Schwartz, and Saffran 1983, Linebarger, in press).

The evidence from implicit tests of knowledge may speak to the issue of shallow output more directly. Because on such tests the aspect of the subject's knowledge that is of interest is tested without the subject's awareness, there is little reason to suspect that central processes, as defined by Fodor, can influence performance. Many of the studies relevant to this issue are discussed at length by Schacter, McAndrews, and Moscovitch (1988). Here we restrict ourselves to only a few brief examples.

Many patients with Wernicke's aphasia or Alzheimer's disease display semantic deficits when asked to define words or match them with each other or with corresponding objects. These patients, however, show normal semantic priming effects on a lexical decision task where explicit knowledge of the word's meaning is not a prerequisite for successful performance (Millberg and Blumstein 1981, Milberg, Blumstein, and Dworetzky 1987, Chertkow and Bub, chapter 7). An interpretation of these results consistent with the notion of shallow output is that the module contains all the semantic associations necessary for normal priming performance on this task so that the output can be shallow, yet appear to be deceptively "deep."

A related effect has been observed in patients with prosopagnosia, (Damasio, Damasio, and Van Hoesen 1982) who often cannot even identify the faces of close relatives or their own face in a mirror when tested explicity. These same patients respond with a higher Skin-conductance response (SCR) to familiar than to unfamiliar faces (Bauer 1984, Tranel and Damasio 1985) and showed interference and facilitation effects, respectively, in reading names that are accompanied by a picture of a noncorresponding or corresponding face (De-Haan, Young, and Newcombe 1987). Indeed on the very same trial the patient may deny recognizing the face even as the SCR indicates that the subject is familiar with it.

Evidence of shallow output, however, is not restricted to perceptual or input modules. The output of memory processes, which are prototypically central, according to Fodor, can also be shallow. Patients who are amnesic on explicit tests of memory may perform normally on implicit tests. They provide the correct answer from memory

even as they claim that they were guessing and had no recollection of the study episode, let alone of the particular item that they produced correctly on the implicit test. (For review, see Moscovitch 1982a, 1984, Schacter 1987a, Schacter et al. 1988, and Shimamura 1986).

Inaccessibility of Intermediate-Level Representations (Nonassembly) There may be several intermediate steps in computing the final output of a module, resulting in different representations at each step. Because modules are informationally encapsulated, only the final output can gain free and full access to consciousness. Intermediate-level representations should not be available to consciousness at all. At a neuropsychological level this means that brain damage should not reveal intermediate-level representations for processes that have been identified as (or are strongly suspected to be) modular. And if they do, the behavior of those processes themselves should not be modular.

Before proceeding further, we distinguish between two senses of the term *inaccessible*. One sense refers only to the subject's being unaware of the representations. The second sense, however, leaves open the possibility that though the subject is not aware of the interlevel representations, they can nonetheless be shown to influence behavior. Our discussion first deals with the latter sense of the term.

Fodor (1983, 1985) noted, for example, that normal subjects are not aware of subphonetic differences, yet their reaction times (RTs) in a phoneme-matching task are influenced by them (Pisoni and Tash 1974, Hanson 1977). Similarly experiments on size constancy have shown that judgments that two objects are identical in size are faster when their retinal images are also identical than when they are not (Blount 1979). In both examples subjects utilize information at interlevel representations of which they are not aware.[2]

As we have noted, both normal and clinical neuropsychological literatures are replete with evidence that higher-order processes of which the subject is unaware can influence behavior (Bisiach et al. 1983, Behrmann, Moscovitch, Black, and Mozer (in press), Schacter et al. 1988, Marcel 1983, Fowler, Wolford, Slade, and Tassinary 1981, Forster and Davis 1984, Cheesman and Merikle 1985; see Dixon 1971, 1981 for review and criticisms of this area).

When the brain is functioning normally, it is probably true that people are not aware of intermediate-level representations. Neurological damage, however, can make available to consciousness those representations that were previously completely inaccessible. One striking example concerns motion perception. Ordinarily we see ob-

jects moving smoothly from one location to another. Even when the motion may not be smooth, as in the phi-phenomenon, the operation of the module gives as its shallow output a smoothly moving object. After lesions to the visual system, however, some patients report seeing the smoothly moving object as a set of static pictures located at various points in the trajectory, much like superimposed stop-action, stroboscopic pictures of a moving object (Teuber, Battersby, and Bender 1960).

A related phenomenon has been reported in face perception by prosopagnosics. Rather than having the shallow output of a face available to consciousness, the patient instead reports seeing visual patches of dark and light (Hécaen, Angelergues, Bernhard, and Chiarelli 1957). The micropsias and macropsias may also be a case in point (Hécaen and Albert 1978). Objects seem unusually large or small because information is accessed before the computations necessary for size constancy are implemented. In the case of agnosias associated with object perception, some theorists believe that what is available to consciousness after brain damage is a $2\frac{1}{2}$D representation or even a primal sketch of the object (Marr 1982, Marr and Nishihara 1978, Ratcliff and Newcombe 1982), rather than the 3D representation, which is typically the only one available to a normal person. In all these cases it seems that the subject's perception is determined more by direct access to projections of the retinal image than would be the case otherwise, that is, when those projections represent intermediate steps of modular processes.[3]

The latter examples strongly suggest that interlevel representations can be accessible to consciousness and imply that modules can be assembled. If more proof that modules can be assembled were needed, one would only have to examine the neuropsychological literature on reading. Unlike language, with which it is obviously intimately related, learning to read typically requires specific training. Learning is often slow and effortful. Much of the child's knowledge outside the specific domain of reading as well as general strategies are recruited (Miller 1988). Nevertheless a significant proportion of children never acquire adequate reading skills. In short, learning to read seems to involve central processes as much as, if not more than, modular processes. Yet once the skill is mastered, reading appears to be modular. If we accept the evidence, the conclusion is that reading modules must be assembled. In fact we are willing to go further and say that the processes involved in many other learned, automated skills are modular by Fodor's criteria and, by extension, must also have been assembled.

Although there are problems associated with many of the criteria for modularity, it is the proposal that modules cannot be assembled that we consider unacceptable. We use our rejection of this criterion as a point of departure for our own speculation about the functional organization of the mind and brain.

To summarize at this point: *At a neuropsychological level a function is considered to be modular if it can be selectively impaired after focal brain damage (domain specificity) and selectively spared in cases of dementia caused by degenerative brain damage (informationally encapsulated).* The neuropsychological evidence that bears on the four[4] essential characteristics of modules was examined and was found inconsistent with a strict interpretation of modularity. Domain specificity was found wanting for a number of reasons: Except for damage to sensory, cortical areas, the domain effected by focal brain damage was difficult to specify in the absence of a proper theory of the affected function. Often the effect of brain damage was not as circumscribed as one would expect if damage affected a single module. Finally, the selective deficits seen after focal lesions were not only restricted to functions that are identified as modular but also obtained for functions that, according to Fodor, are prototypically nonmodular, such as memory and attention. The same seems to be the case for shallow output. With regard to informational encapsulation, the neuropsychological evidence from patients with dementia and sparing of some (modular?) functions is too sparse to be conclusive. The evidence from patients with brain damage also strongly suggests that functions that are believed to be modular can be assembled.

Can Modules Be Assembled? A Proposal for a Different Modular Organization

Why Modules?

In the previous section we noted that among the various features that are characteristic of modules, some seem to be common to almost any biologically relevant computational device, and others have a variety of problems associated with them. Why then subscribe to the notion of modularity at all? The reason is that we believe, with Fodor, that to present to the central processes veridical information about the world quickly, efficiently, and without distortion from the beliefs, motivations, and expectations of the organism, something like modules that are immune to higher-order influences must exist.

Three Types of Modular Organization

We believe, however, that there are three types of modules, differing in complexity and composition. One type consists of a basic module similar to the one proposed by Fodor. The other two types are modules that are assembled from basic modules to form a collection that usually behaves as an indissoluble unit.

Type I: Basic Modules The basic modules are those that alone carry out a single function. These modules probably evolved to deal only with highly relevant and predictable environmental stimuli. Among these would be modules for the perception of basic sensory features in each modality. In addition there may be basic modules that pick up complex sensory information such as faces and emotional expressions, where the relevant information is configurational rather than restricted to a single feature. Alternatively this type of complex information may be handled by type II modules.

The neuropsychological evidence suggests that perception of colors, acoustic frequency, sound location, visual location, motion, depth, faces, and perhaps emotions can each be impaired in isolation (Benton 1985, De Renzi 1982, 1986, Hécaen and Albert 1978, Bowers, Bauer, Coslett, and Heilman 1985) and may continue to function normally when the patient is severely demented. Because in each of these cases all the criteria that define a module seem to be met, the evidence in favor of considering each of these to be modular functions is quite good. The slight discrepancies that might arise concerning the domain over which damage to a module produces some impairment can typically be accounted for by the size of the lesion.

It is less clear that other functions can be subserved by type I or basic modules. A case in point is language: The neuropsychological evidence that modules exist for even theoretically separable aspects of language, such as phonology and syntax, is inconclusive (Saffran 1982). On the positive side are reports of word-deafness and agrammatism, suggesting that phonological and syntactic operations, respectively, can be impaired in relative isolation. The main negative evidence is the failure of many of the patients, certainly the ones with syntactic impairment, to meet the criterion of domain specificity—we are not aware of reports of even a single patient in which speech output or syntactic processing is impaired and all other functions appear normal. Unlike the case for perceptual modules, it is difficult to argue that the size of the lesion can account for the violation of domain specificity in agrammatic aphasia. The reason is that there have been too many studies, from Jackson's (1878/1932) in the last century to Kimura's (1977) and others' in the past decade, linking

speech output and syntactic deficits to more general cognitive and motor impairments. The main conclusion of these studies is that speech output and syntactic deficits are only one set of symptoms that arise from the impairment of more basic functions such as sequencing and resolution of rapid temporal patterns (Keele, Pokorny, Corcos, and Ivry 1985, Luria 1966). The alternative is either to consider both speech and syntactic processing as dependent on central processes, or, if they are modular, to consider their domain to be neither speech nor syntax but some other basic property whose features have yet to be specified.

Type II: Innately Assembled Modules consist of a collection of modules whose organization is innately given and whose output is integrated or synthesized by a devoted, nonmodular processor. We use the term *devoted* to indicate that this processor can deal with information coming only from a particular group of modules and no other. This organization is similar to one proposed by Turvey for vision (1973, Michaels and Turvey 1979) in which a devoted central processor integrates input from modular feature analyzers. Like basic modules, type II modules are domain specific, though their domain is much broader. They correspond to the units or mechanisms in Luria's secondary zone (1966). The reason for proposing type II modules is that it is inconceivable to us that there can exist a module for every class of objects that one encounters in the same way that a module exists for faces. If recognizable objects and modules stand in 1: 1 correspondence, there would have to be too many modules, conceivably an infinite variety of them. If object recognition is served by a single module or even a small numbers of them, the domain over which each module would have to operate would be so large as to make it indistinguishable from a central cognitive system. Proposals for modules whose single, dedicated function is to provide structural descriptions of objects (Riddoch and Humphreys 1987) would seem to us to be more consistent with type II modules than with the basic modules of Fodor.

Additionally, as we noted, evidence that even primitive modular functions may be fragmented into simpler, modular processes leads us to postulate the existence of type II modules. Deficits in object perception can be due to lesions that affect component modules, damage to the devoted central processor that integrates the output from those modules, or disconnection of that output from a higher-order central processor that makes general knowledge available and that is associated with consciousness (Johnson-Laird 1988, Umilta 1988a). The disconnection from the higher order central processor is

common to all types of modular organization and is not relevant to a discussion dealing only with type II modules (Schacter et al. 1988).

It follows from our account that type II modules are capable of modification or learning. Information about specific exemplars of objects, faces, or words are picked up and stored by these modules. Performance on implicit tests of recognition, identification, and memory supports this view (see especially the following section on implicit memory).

The neuropsychological evidence for the existence of type II modules comes from the more complex types of agnosia, such as visual object agnosia, topographic agnosia, agnosia for body parts, and astereoagnosia (object agnosia in the tactile modality) (Bauer and Rubens 1985, Benton 1985, De Renzi 1982). What is characteristic of these agnosias is that they are domain and modality specific, though in each case the domain is quite large; another of their characteristics is that knowledge about the object does not improve its perceptibility in the impaired domain. In some cases the deficits arise, in part, because the output from basic modules seem to be impaired. Here deficits in perceiving basic features accompany the agnosia. In other instances, basic sensory deficits are minimal, but the agnosia arises instead because the devoted central processor seems incapable of integrating the output from the basic modules. In some cases subjects can even copy a line drawing of the object perfectly, but cannot identify it. In addition subjects often cannot imagine what the object would look like in response to a verbal cue, though they can state its function and use the word denoting it properly. Together the evidence supports the idea that it is a devoted central processor rather than a basic input module or a higher-order central processor that is damaged.

From our point of view language is also, in part, mediated by a collection of modules, each with its own function, which are organized to form an intricately related system. The organization is either innately given or innately specified in the sense that early experience modifies it only along prespecified lines and "fixes" the organization so that later experience has little influence on it.

The evidence that language is mediated by a type II module comes from the various aphasias. We have already noted that there are rare cases of language isolation in which phonological, syntactic, and speech output processes can be intact despite the subject's inability to assign meaning to them. In agrammatic aphasia the subject is unable to make conscious use of grammatical knowledge to assign the proper grammatical roles for the lexical items in a sentence (though the ability to do so implicitly may be preserved) (Linebarger et al. 1983,

Saffran 1982, Schwartz et al. 1979). Lexical access, however, and assignment of meaning outside of syntax may be relatively spared. In some forms of receptive aphasia, the converse is the case. The subject may speak grammatically, but has difficulty in assigning meaning consciously to the lexical items. At a simple level one can interpret these results by saying that agrammatic aphasia results from damage to a syntactic module and receptive aphasia from damage to a semantic module. Some might argue that this is hardly an adequate explanation because one merely substitutes one type of nomenclature with another. We simply wish to call attention, however, to the fact the language cannot be conceived as a unitary module, but may be an assembly of modules, each with its own function (Linebarger, in press).

As we noted previously, it is quite possible that language may not fit a modular organization very well. Both agrammatic and receptive aphasias are accompanied by phonological deficits in perception and often in production as well as in other sequential and analytic tasks that have little ostensible relation to language. Is this the result of a lesion overlapping more than one module, or is each module's domain larger than its theoretically assigned function would require? Judging from neurosurgical, stimulation, and CT (computed tomographic) scan studies, the former interpretation would seem preferable because the affected areas are typically quite large. Nonetheless finding a consistent association between two unrelated deficits undermines the case for modular organization.

This type of modular interpretation of language can accommodate recent evidence that agrammatic aphasics are capable of making grammatical judgments (Linebarger et al. 1983) and that receptive aphasics show semantic priming effects for words they do not understand (Millberg and Blumstein 1981, Millberg et al. 1987). This suggests that aphasias, like some agnosias, can arise from a disconnection from a higher-order central processor. According to this view, the syntactic module is intact in the agrammatic aphasic. What is impaired is either the shallow output of the module to a higher-order central processor that assigns syntactic status or role to the different items or the central processor itself. For similar reasons the receptive aphasic patient may not be able to assign the appropriate meaning to the automatic output of an intact lexical module.

Type III: Experientially Assembled Modules are similar to type II modules except that central processes are involved in assembling the component basic and type II modules which, once integrated, carry out functions that become modular with practice. The difference between

type II and type III modules is illustrated by considering the difference between walking and riding a bicycle, or speaking and reading. In the first two instances the organization of the modules is innate in the sense that it is prespecified, though experience is necessary to allow that organization to unfold. In the latter two instances the organization is guided and formed by experience. In the cognitive literature acquired automatized processes would qualify as examples of type III modules. A number of studies have suggested that attention is necessary for assembling subroutines which are then run off automatically (Logan 1978, 1985, Norman and Shallice 1986, Duncan 1986).

Neuropsychological evidence for type III modules are the acquired dyslexias (Coltheart, Patterson, and Marshall 1980, Patterson, Coltheart, and Marshall 1985, Saffran 1984, Shallice 1988) and ideational apraxias (De Renzi and Lucchelli 1988, Heilman and Gonzales Rothi 1985). Automatized processes in reading and learned motor sequences are clearly acquired.

The dissolution of function in ideational apraxia is particularly informative in this regard. Ideational apraxia is the inability to carry out a learned complex action, such as making spaghetti or brewing coffee or shaving, in the proper sequence despite no loss of understanding and no motor disorder. What is particularly striking, and critical from our point of view, is that each element in the sequence can appear normal. It is merely the order in which the sequence is run off that is impaired. Ideational apraxia may be the motor analog of object agnosia and dyslexia in the sense that the output of basic modules are intact, but their integration by a central processor is impaired (Roy 1983).

One prediction is that recovery of function should be better and different after damage to type III than to type II modules because in the former instance new modules could be assembled that together can perform the lost function, albeit in a different way. For type II modules the central processor is devoted, and another one cannot be substituted to take its place. To take reading as an example, damage to type III modules (or to their components) can lead to different forms of reading disorders. Phonological dyslexia results from damage to a type III module that automatically converts graphemes to phonemes. When this module is damaged, the individual must rely on alternate routes to reading (Patterson and Coltheart 1987). An even more dramatic instance occurs in alexia without agraphia, where difficulty of access to the whole word forces the individual to read letter by letter. (Landis, Regard, and Serrant 1980, Shallice 1988, Shallice and Saffran 1986, Patterson and Kay 1982, Coltheart 1985.)

Summary Three different types of modules are proposed: Type I is a *basic module* that meets all the characteristics ascribed to modules by Fodor. Modules that process and deliver information about sensory features, and perhaps about speech and faces, are of this type. The other two types of modules, though assembled, retain many of the features of basic modules. Type II is *innately assembled* from a collection of basic modules by a devoted central processor. Object recognition modules are of this sort. Type III is an *experientially assembled* module in which a central processor effortfully assembles the component basic and type II modules. With repeated use, these component processes become fully integrated and automated and as a result assume the characteristics of module. Examples of type III modules are those concerned with reading and learned, skilled motor sequences.

Central Processes

Fodor (1983, p. 4) conceived higher cognitive faculties as "slow, deep, global rather than local, largely under voluntary (or, as one now says, "executive") control, typically associated with diffuse neurological structures, neither bottom-to-top nor top-to-bottom in their modes of processing, but characterized by computations which flow every which way. Above all, they are paradigmatically unencapsulated; the higher the cognitive process, the more it turns on the integration of information across superifically dissimilar domains." In short, central processes that mediate higher cognitive functions are everything that modular processes are not.

At the beginning of the chapter, we noted that some of the characteristics mentioned here, such as processing speed, direction of information flow, and localization of function, do not always distinguish central from modular processes. The critical differentiating factor, as Fodor emphasizes, is informational encapsulation (and perhaps also domain specificity). Modules are defined by their informational content, which is used in their computations and which determines and restricts the type of hypotheses or propositions they project to central processes. Never mind that often it is difficult to specify the kind of information that modules receive, contain, and project. The crucial points are that the information is limited to a certain type in modules, whereas the information received by central processes and used in their computations can be infinitely diverse. In contrast to modules there is, in principle, no restriction on the information central systems can bring to bear in projecting hypotheses *and* in the types of hypotheses they can project.[5]

Faced with the prospect of accounting for this potentially infinite diversity of information and the hyptheses that central systems can receive, compute, and project, Fodor, from his perspective, sensibly concluded that one cannot have a scientific psychology of central processes. This conclusion is valid only if psychology's goal is to capture the content of thought. Fodor's concern with informational encapsulation, and the theory of knowledge that it implies, led him to dismiss, or at least belittle, the possibility that in some areas of inquiry it is the processes themselves that should be the focus of scientific psychology (or neuropsychology) rather than the information that the processes compute.

In truth, it is probably impossible to separate the information that is represented from the processes involved in representing it (Anderson 1978). When the information content is obvious and the process that computes that information is not evident, then the proper research strategy is to focus on content; when content is unspecifiable, then a clue can be gained by studying the process. Thus by virtue of their properties, especially of domain specificity and informational encapsulation, modules are typically defined by the kind of information they receive, compute, and project. In contrast, we suggest, central processes are defined by the function they serve.

At the neurological level the distinction between modules and central systems is not necessarily that one is more focal in its structural organization and that the other is more diffuse or distributed, as Fodor suggests. The specificity of the deficit following damage, however, should be equivalent in each case, except that in one case the deficit is typically described in terms of loss of knowledge and in the other in terms of loss of function. The defining characteristics of modules, however, suggest that the major difference between modules and central systems lies in their connections; input pathways to modules should be fewer than those to central systems. Sometimes the input pathways to the central systems are known precisely, so that it is possible to specify with some confidence the type of information that each of the pathways deliver. In those cases a kind of paradoxical effect is found that blurs the distinction between modules and central processes. Small local lesions that damage that part of the central system that receives input from only one pathway can lead to deficits of a particular function that are as domain specific as any observed after damage to modules. When the neuronal architecture is known, the distinction between modules and central systems becomes very fuzzy.

An example will help us illustrate our points. More than any other structure in the brain, the prefrontal cortex is associated with the

higher-order functions that are prototypical of central systems. Damage to sizable portions of the prefrontal cortex in humans produces deficits in attention, problem solving, memory, spatial orientation, as well as changes in social interaction and personality (Damasio 1985, Duncan 1986, Milner 1982, Walsh 1988). The prefrontal cortex, which comprises about 30 percent of the cortex in humans, is not a homogeneous structure. A number of cytoarchitectonically distinct zones have been identified, some of which are believed to comprise functionally distinct subsystems on the basis of double dissociation experiments in humans and in monkeys.

One such region that has been extensively studied in monkeys is area 46, a region on the dorsolateral convexity surrounding the sulcus principalis. Lesions in this region produce deficits on delayed response tests only in the visuospatial domain. Performance on a variety of visual tests that do not require memory for a specific location in space is normal. Recent experiments by Goldman-Rakic (1987) and her colleagues have demonstrated that by making ever smaller lesions, the deficit can be restricted to only that portion of space that projects its input, via the parietal lobes, to the damaged zone of area 46. Thus a lesion restricted to a portion receiving projections from only one quadrant of the visual field produces deficits on delayed response tests only if the stimuli appear in the affected quadrant. Performance is normal in response to stimuli in other quadrants. Moreover, with the exception of delayed response, the monkeys' visual responsiveness to stimuli in the affected quadrant is normal. Despite the extreme domain specificity of the deficit, one should not lose sight of the fact that it is caused by a lesion to a part of the prefrontal system that has been characterized as central rather than modular. Indeed, according to Goldman-Rakic (1987), the general function of sulcus principalis may not be different from that of the prefrontal cortex in general—"it is to use short-term representational memory (i.e., internalized knowledge) to guide behavior in the absence of informative external cues." What distinguishes the sulcus principalis from other regions is the specific domain over which this function is performed. Though damage to large portions of the prefrontal cortex produces the kind of global deficits in cognition (and emotion and personality) that one predicts would occur after damage to a central system, the deficits observed following small circumscribed lesions are reminiscent of those that occur after damage to perceptual and motor modules.[6] *Thus, at the global level the prefrontal cortex behaves as a central system, but at the local level the prefrontal cortex (and other cortical areas) may resemble modules.* As Goldman-Rakic put it, "The principal sulcus is therefore as specialized for performance

based on visuospatial memory as is the dorsal motor cortex for voluntary limb movement and the lateral striate cortex for central vision" (p. 381).

We do not have a solution to the potential difficulties that this illustration causes for neuropsychologists wishing to maintain hard distinctions between central and modular systems. The criterion of domain specificity seems to be met by the preceding example. Whether informational encapsulation and shallow output are also satisfied has not been determined.

Until a solution is found, we will continue to adhere to some of the basic distinctions between central and modular systems because we believe, for the moment at least, that the interplay of central and modular systems provides the most useful explanation of normal and impaired cognition. Having committed ourselves to this enterprise, let us proceed with the discussion of central processes.

The criteria used for identifying central processes are criteria of exclusion. If they do not satisfy the criteria for modular processes, then they are central. Because central processes are not domain specific or informationally encapsulated, the focus in studying them will be on the operations they perform, rather than on their informational content. As we noted previously, the technique of double dissociation can be applied as easily to central processes as to modular ones. Consistent with this point of view, we should be able to find evidence of brain damage that impairs one of these processes yet leaves the others intact. In the following paragraphs we briefly review the neuropsychological evidence to support this prediction. Four different types of central systems, defined according to their function, are identified. They illustrate, but do not exhaust, all the different types of central systems that may exist.

Function 1: Forming Type II Modules

As we have already discussed, this function is performed by a devoted central processor in conjunction with type I modules to form type II modules. In many ways the operation of the devoted processor is similar to that proposed by Treisman and her colleagues (Treisman and Gelade 1980, Treisman and Souther 1986) to account for the conjuntion of separable features into an object. The features are provided by the basic modules and their conjunction by the devoted processor (see also Turvey 1973). Though Treisman and her colleagues assume that the operation of this processor always requires attention, it is possible that noticeable amounts of attention are necessary only under the conditions of tachistoscopic presentation, when the combination of features is artificial, and a demanding search is required.

In more naturalistic settings, the processor may execute its functions relatively effortlessly. Phoneme identification and syntactic parsing may also require the services of a devoted central processor.

We believe that devoted central processors are associated with each modality and that each is located in different cortical regions, usually in close proximity to the sensory region that delivers modular input to them. Thus, though the domain may be large, its boundaries are typically confined to a single modality. They are essentially linked to the modules whose input they receive and in combination with which they form a type II module. In other words they are a kind of bridge between basic modules on the one hand and higher-order central systems on the other. Evidence for this type of devoted central processor was noted previously.

Function 2: Formation and Maintenance of Type III Modules

Damage to the central systems having this function leaves the components of type III modules intact, but impairs their organization into an operational unit. In the case of type III motor modules, the order in which the various components are activated will be disrupted, but each component act will be performed normally. Such disorders can be manifested in two different ways, each of which is associated with damage to different regions of the brain: The best known and the one with the longest history in neuropsychology is *ideational apraxia* (De Renzi and Lucchelli 1988, Heilman and Gonzales Rothi 1985, Roy 1983). This condition is associated with damage to the posterior portion of the left hemisphere and results in the disruption of well-learned motor acts in which each of the elements is intact, but which cannot be ordered properly. For example, when asked to shave, the patient knows how to pick up the razor, lather his face, scrape his face, and so on, but the sequence in which these separate acts are conducted is incorrect. As we already noted, the deficit involved in this type of apraxia and the lesion associated with it bear a strong similarity to object agnosia. In ideational apraxia the central systems are required to maintain and run a particular and limited set of highly organized subroutines, whereas in object agnosia they are devoted to integrating input from a restricted set of modules (see also similar proposals by Goldberg 1989, Goldberg and Costa 1986, Goldberg and Bilder 1987).

The central processes involved in maintaining and running off old action sequences are not the same as those involved in organizing new ones. The processes presumed to be impaired in ideational apraxia normally operate only when the sequence has been well learned or is routinized. Before being routinized, the components forming the

sequence must be learned and then effortfully strung together. The function of establishing, rather than maintaining, the appropriate organization of the components is relegated to other central processes. It follows that damage to different brain regions should disrupt these two related functions. This is indeed the case. Damage to anterior frontal cortex impairs the establishment of new routines, which can range from simple motor sequences to solving problems that have many steps. Patients have difficulty in learning to sequence a set of hand movements, face movements, arm movements (Kimura 1977, Kolb and Milner 1981), and a series of actions performed on a latch box (Kimura 1977), though each of the components can be executed flawlessly (see summary in Kolb and Whishaw 1984).

When it comes to problem solving or memory for temporal order, it is not clear that the deficit can be characterized as an impairment in collating new action sequences, even though it is caused by frontal damage. The deficit may arise from impairments in planning or in relating the outcome of each step to the general goal or in subordinating one act to another or in changing sets (Duncan 1986, Milner 1982, Norman and Shallice 1986, Shallice 1982). These last possibilities are to a large extent subsumed under function 4, and they depend on closely related, and maybe even overlapping, but different cortical regions.

The central processes associated with the frontal lobes operate over a larger domain than those in the posterior region, but there are still some constraints. The systems on each side of the brain seem to confine their operations primarily to input received from the same hemisphere or to output governed by that hemisphere. For example, memory for temporal order is impaired by left frontal lesions if the material is verbal, but by right frontal lesions if the material is nonverbal. As more becomes known about the neuroanatomical connections of different regions of the frontal lobes, and about the effects of small focal lesions to these regions, the more likely it is that their operation will satisfy the criterion of domain specificity.

Function 3: Relating Information to General Knowledge

It is this function that perhaps is most problematic to followers of Fodor. How much knowledge is stored in a module? How much information about an object or a word is contained in the shallow output of an object, face, or lexical module? We have already argued that for objects the information seems to be modality and item specific, but does the output provide information about, say, the object's weight, texture, and function (Chertkow and Bub, chapter 7)? If the item is a word, is information made available about its meaning in

different contexts? or if it is a face, is there information about the person's name, occupation, personality trait (Bruce and Young 1986)? These questions were raised in discussing information encapsulation and shallow output, and suggestions were put forward about the type of evidence that might be used to derive answers. What concerns us here, however, is the role that central systems play in supplying the information that is not made available by modules. Two aspects of the central systems' role can be distinguished: one is receiving informational content from modules and the other is the process that relates semantic knowledge to modular output. By combining both aspects, central processes assign meaning to modular output.[7]

For example, systems having this function will interpret the meaning of a 3D representation of an object delivered to it by a type II module. That is, a name could be assigned to the object, its function and relation to other objects made known, and so on. To do this, contact has to be made with general knowledge.

Deficits in this function can arise either because the knowledge base, the semantic core, is "depleted" or because the process that delivers (some of) the necessary semantic information is impaired. In principle it might be possible to distinguish between these two aspects, but in practice it has proved very difficult. What constitutes evidence that the semantic core is deficient? Presumably evidence from patients with global or restricted semantic memory loss in all domains who nonetheless show evidence of adequate processing at the modular level. Such patients should be able, say, to read, repeat words, and recognize items as identical within a modality, yet not know the meaning of the item regardless of the modality in which it is presented. This type of global semantic loss, reported in some demented patients, is in contrast to the modality specific semantic loss associated with the various agnosias (for further discussion, see Chertkow and Bub, chapter 7, and Schwartz and Chawluk, chapter 8).

Though central, the process used to derive the prototypical semantic knowledge of an item—its accepted, common meaning and function—is an *associative process* that is automatically activated by the item. Confronted with the item *chair*, its prototypical meaning and function is immediately apprehended by associative process in anyone who is not demented. This associative process is in contrast to a *strategic process* that allows one to gain access to all the other things one knows, or can infer, about chairs, their potential functions, and even their symbolic value. It is also this kind of process that seems to be involved in identifying by inference stimuli that are either physi-

cally degraded or are perceptually distorted because the modules that ordinarily code these stimuli are damaged.

This distinction between associative and strategic processes related to item knowledge is borrowed from research in episodic memory. Whether the distinction will prove useful in studies of semantic memory remains to be seen. At the neurological level the evidence suggests that damage to the posterior temporal-parietal cortex, either on the left or bilaterally, leads to *associative* semantic memory deficits, whereas damage to the prefrontal cortex leads to *strategic* semantic memory deficits (Luria 1966, Goldberg, 1989).

We should note that strategic aspects of function 3 are also necessary in planning. Indeed it stands at the junction between those central systems that assign meaning to sensation and those that use central, internal representations of the perceptual world to organize action. To execute or choose the appropriate plan, one must bring to bear one's general knowledge on the problem at hand, and one must evaluate the plan in relation to what one knows about the world. The planning function itself, we think, is separate from the function that is necessary for relating information to general knowledge. Patients with function 3 impairment may have ineffective plans because they are based on poor knowledge, but a patient with function 4 impairment may have all the necessary knowledge without being able to formulate an appropriate course of action.

Function 4: Planning

In planning, a goal has to be set, a strategy adopted, the action sequences selected, the process monitored, and the outcome verified against an internal representation of the goal that is to be achieved. Selecting the goal and strategy also requires function 3, and collating subroutines requires function 2. All these functions need conscious intervention of a central processing device that has been termed the supervisory attentional system by Norman and Shallice (1986), a central monitor by Weiskrantz (1988), a central processor by Umiltà (1988a), the central executive by Baddeley (1986), and the operating system by Johnson-Laird (1983, 1988). The operations of this device are assumed to be effortful, slow, and serial. Among its functions is the inhibition of irrelevant or interfering subroutines. Though we agree that some central device is necessary for coordinating all the functions involved in planning, we would like to reserve judgment on whether all the functions are carried out by a single processor or whether they are relegated to a number of processors that are interrelated, but than can be selectively impaired. From the foregoing discussion it should be clear that we exclude from function 4 the

mere collating of action sequences, whether automatic or otherwise, which is achieved through function 2.

Anecdotal evidence has existed since the last century that patients with frontal damage have difficulty in setting goals, organizing purposeful behavior, and adhering to the plan of action in mundane daily activities (Harlow 1868, Bianchi 1922). These patients are easily distracted from their goals and tend to omit relevant activities and insert inappropriate ones. These deficits are highlighted in control laboratory tests. Shallice (1982, 1988) suggests that frontal patients fail in their attempt to solve puzzles such as the Tower of London (or Hanoi) because they follow the solution of least resistance rather than correcting false starts by evaluating their moves with respect to prescribed goals. In finding their way through visual and tactile mazes, frontal patients cannot adhere to the structure of the task, but break rules even in those parts of the task that are easy for them to solve. Frontal patients may even have difficulty in learning because they cannot monitor or keep track of their own current responses in relation to previous events and future goals (Petrides and Milner 1982, Milner 1982).

Apart from rule breaking, these are all what Jackson (1932) would term negative symptoms associated with frontal lobe damage. The most prevalent of the positive symptoms is perseveration (Milner 1966, Luria 1966). It is important to distinguish between perseveration restricted to a specific domain (Goldberg and Costa 1986, Goldberg and Bilder 1989, Duncan 1986), which can occur following lesions to a variety of structures, from a general perseverative loss, which is typically associated with frontal impairment (and perhaps associated subcortical structures; Freedman and Oscar-Berman 1986, Kimura, Hahn, and Barnett 1989). General perseveration can occur either because the subject does not monitor performance, so that an action is repeated after it has already been executed, or because the subject has difficulty in abandoning an unsuccessful strategy or inhibiting a prepotent response and choosing a new one.

Sometimes subjects' inability to plan may cause them to adopt routinized strategies automatically even though they are unsuccessful. Another type of deficit occurs because subjects are too easily deflected from the plan or strategy they have devised by irrelevant salient stimuli that trigger automatized subroutines. Whether this results from a failure in monitoring performance, from an inability to focus attention, or from a tendency for abandoning long-term strategies for a seemingly more immediate, but inappropriate, goal is currently open to debate. Shallice (1982, 1988) attributes these positive symptoms to the release of *contention scheduling*, which is an

automatic process of activating subroutines, from the control of the supervisory attentional system. To use our terminology, the subjects rely on associative rather than strategic processes to guide their behavior.

Summary

Where central systems are concerned, it is generally neither feasible nor interesting to characterize the system in terms of its input domain or the information that it can potentially encode or transmit. The particular function of a central system, however, may be known or open to discovery. Four such functions, each associated with different central processes, were proposed: (1) forming type II modules, (2) forming type III modules, (3) relating information to general knowledge, and (4) planning. At the neurological level, structures mediating central processes can be as localized as those associated with modules, though the former should have more varied and extensive connections to other structures. The deficits resulting from damage to central systems are often as specific as those following damage to modules. Indeed, small, highly circumscribed lesions in a single pathway (or in the central system structure itself) can produce deficits that satisfy all the neuropsychological criteria of modularity. That some systems behave as modules at a local level, but as central processes at a global level, is a paradox that makes it difficult to maintain hard distinctions between modules and central processes, but that may point to a fundamental organizing principle of the brain.

The following section applies the conceptual framework we have developed to memory and attention.

Attention and Memory

Memory and attention figure in almost every cognitive act. Fodor (1983), following Gall, confers special status on memory and attention by making them, and not other horizontal faculties, part of every module. Fodor treats memory as merely stored information, an engram, specific to the module to which it belongs. Attention for him is simply a resource that activates the module of which it is a part and modulates the efficiency of its operation. We believe that Fodor takes too narrow a view of memory and attention. For us, as for others, memory is a complex function, akin to problem solving. Attention is no less complex primarily because its selectional aspect makes use of general knowledge and links it to the executive or supervisory functions of central systems. For these reasons, both memory and atten-

tion are best understood as reflections of the operations of modular *and* central systems. The following discussion elaborates these points.

Attention and Working Memory

Because attention and working memory (WM) are interrelated, to understand one, one must also understand the other. There are two major aspects to attention: selection and resource allocation or expenditure. Resources refer to the mental energy needed to execute cognitive operations (Wickens 1984). We will assume that resources are limited.[8] Selective attention is the process by which cognitive operations and internal representations enter WM, where resources are allocated to them under the supervisory guidance of information already in WM. Information may also enter WM automatically (Navon 1984, Schneider, Dumais, and Shiffrin 1984, Umilta 1988a, b; Underwood 1982).

According to Baddeley (1986, p. 34), WM refers to "the temporary storage of information that is being processed in any range of cognitive tasks." The processing components of the tasks being executed, as well as their informational content, also occupy WM. The cognitive operations and representations in working memory are under conscious control. The capacity of WM is limited. Although not universally accepted, we will assume that the limits of WM are not defined by a fixed number of items or bits of information but rather are set by the cognitive resources necessary for maintaining information and operating on it. The greater the demands of the operations on cognitive resources, the fewer items can be held and attentively processed in WM (Daneman and Green 1986, Baddeley 1986).

One can conceive of selective attention as a process whereby information is conveyed into WM and maintained there or as a spotlight that illuminates whatever cognitive process-representation it focuses on (Baddeley 1986, Klatzky 1984, Posner 1980). Consciousness is the quality we ascribe to experiencing the contents and processes of WM. Put another way, it is only when cognitive information and processes are in WM that we can be conscious of them. Conversely only information that is in WM is consciously apprehended.

For modules, only the output can be available to WM, whereas for most central processes the intermediate steps or outcomes, as well as some aspects of the computations involved, may also be available. This statement needs to be qualified in considering type II and III modules. In the case of type II modules, because a central process is needed to integrate modular output, we may become aware of the output of both modules and the devoted central processor, though not of the way either operates. Such awareness may be evident when

type II modular processes break down after brain damage. It is not known, however, whether conscious awareness of the separate components is also possible when type II modules are functioning normally. With respect to type III modules, such as reading or executing a complex motor skill, like a stroke in golf or tennis, there is conscious awareness of all the intermediate steps during compilation of the modules, but typically only of the final output once the process has been assembled and automatized. For example, in learning to read a nonalphabetic script like Hebrew after one has mastered an alpabetic script, one is acutely aware of the separate graphemes, their sound correspondence, the blending of these, the dawning recognition that it sounds like a familiar word, and the search for what the word might mean. With practice the entire process seems to become as automatic as reading the alphabetic script. However, even once it has become automatized, it may still be possible to make available to WM some of the intermediate steps at the expense of slowing the activity down or breaking it up into its separate components. Consider what happens when one tries to bring under conscious control the intermediate steps of such activities as hitting a ball, driving, or reading.

For central processes, as for modular ones, we are never conscious of the internal working of the algorithms that act on the representations. As we have noted, in this way central processes resemble modular ones: The working of the devices themselves are cognitively impenetrable and computationally autonomous.

Within this framework, neuropsychological evidence of attentional deficits can be listed under the following headings: (1) the capacity of WM is reduced or appears to be reduced, (2) the mechanism for allocating attention is damaged, and (3) entry of information into WM is impaired or blocked.

Attentional Deficits

Reduction in WM Capacity: General Versus Specific Deficits in Demented and Nondemented Patients If WM is assumed to be a single system and its capacity is reduced, there should be a comparable reduction in capacity across all processes in which WM is involved. That appears to happen in conditions, such as dementia or intoxication, that affect the general operation of the nervous system (Baddeley 1986, Morris and Kopelman 1986). However, the idea of a single WM with a fixed capacity runs afoul of data from both the normal and clinical literature that show that WM capacity can vary depending on the task the subject is asked to perform. WM memory capacity as measured by reading span is different from that measured by listening span or digit

span, which in turn differs from that measured by spatial span (Daneman and Tardif 1987, Baddeley 1986). Similarly, verbal auditory WM can be selectively impaired in patients with conduction aphasia, with visual and spatial WM left relatively intact (Shallice and Warrington 1970, Warrington and Shallice 1969, 1972, Vallar and Baddeley 1984a, b). The converse syndrome can also occur (De Renzi 1982). One solution for interpreting these data is to posit multiple WM systems, each of which is allocated to different functions. Put in Fodor's terms, WM is an integral part of each module.

An alternate more parsimonious, and psychologically and neurologically more plausible solution is that there is neither a single WM in Baddeley's sense nor are there multiple WM systems. Instead WM is that entity that reflects or represents whatever processes are currently active and whose outcomes or operations are consciously apprehended. If a processor has a reduced capacity or is damaged, it will appear as if WM capacity is also reduced whenever that processor is operating. In other words, specific deficits in WM are related to specific deficits in the processes to which attention is allocated. To repeat, the capacity of WM will vary depending on which central (and sometimes modular) process is currently active, not because there are multiple WM systems but because multiple processes and their output are capable of capturing and occupying our attention (and conscious awareness).

This proposal makes it possible to distinguish between the effects on WM produced by a general reduction in resources that is caused by neuronal loss or by diffuse neuronal inactivation and those produced by a specific impairment to a particular process, whether modular or central, that is caused by focal damage. If the loss is general, WM capcity will appear to be reduced in many domains and across many cognitive functions, whereas if it is specific, WM capacity will be impaired only within a specific domain or for a particular cognitive function. Moreover, in the general case, the type of cognitive opertions performed by a central system or by a module will not change if resource loss does not exceed a certain threshold, though the efficiency with which those operations are performed may suffer. If a particular central system or module is damaged, however, its operation will be abnormal or, in the extreme case, lost entirely.

The latter points are illustrated clearly in contrasting WM deficits in patients with Alzheimer's dementia and in those with conduction aphasia. Certain patients with conduction aphasia appear to have an impairment restricted to the phonological store (Vallar and Baddeley 1984a, b). These patients have a highly circumscribed deficit in verbal memory span that expresses itself as a difficulty in verbatim repeti-

tion. The case for a phonological storage deficit rests primarily on demonstrations that factors normally thought to interfere with phonological storage capacity, such as word length, or articulatory suppression and phonemic similarity when the words are presented visually, have little or no effect on patients' performance (Vallar and Baddeley 1984a,b, Baddeley, Lewis, and Vallar 1984). For the most part language comprehension and production, as well as nonverbal abilities, are normal. In contrast patients with Alzheimer's disease and a reduced verbal memory span have a phonological store that operates normally, but inefficiently, as evidenced by their sensitivity to factors whose influences depend on a functioning phonological store (Morris 1984). As a consequence of the generalized loss of cognitive resources, the verbal memory deficits of Alzheimer's patients, as measured by their performance on the Brown-Peterson test, were exacerbated when they had to perform even simple concurrent tasks that drew on their already-reduced resources (Morris 1986).

If we assume that as a rule central processes demand more cognitive resources than modular processes, then deficits in Alzheimer patients should be greatest for central as opposed to modular processes. One set of findings seems ideal to illustrate our point: Item search—a slow, serial, effortful, attention-demanding process (Treisman and Gelade 1980) that is almost prototypically central—is impaired in some patients with Alzheimer's disease (Saffran et al., chapter 9). On the other hand feature search—a parallel, automatic process in which target detection appears to be mandatory and modular—is relatively spared in the same patients.

Not merely searching for a particular item but the very process of integration that we believe is accomplished by devoted central processors (type II modules) is also impaired in Alzheimer's patients. Schlotterer, Moscovitch, and Crapper-McLachlan (1983) asked patients to attempt to identify a single target letter that was followed by a visual mask. They measued the interstimulus interval between the target and mask at which the mask no longer affected perception of the target. A homogeneous mask, such as a bright flash of light, impairs target identification by interfering with the pickup of basic features (the operation of a type I module), whereas a pattern mask interrupts a central processor that integrates these basic features (type II module, Kolers 1968, Turvey 1973). Consistent with our prediction Alzheimer patients were impaired only on the pattern-masking task.

A critical component of WM is a central executive (Baddeley 1986), whose function is to summon, coordinate, and maintain processes and information that are active in WM, to allocate resources to them, and to delete them when they are no longer needed. The central ex-

ecutive itself requires resources to perform its function. This central executive is probably part of the set of central systems that we mentioned in connection with function 4.

A malfunctioning central executive is typically an early symptom of dementia. As we noted, central processes are distinguished from modules by the number of pathways and by the cognitive resources they require. The widespread neural degeneration that causes dementia is very likely to disrupt the broad associative and integrative communication pathways that the central executive requires to perform its function, but not necessarily the more limited and restricted modular pathways. A reduction in cognitive resources is also expected to accompany neuronal loss. Last, neural degeneration is often accentuated in the prefrontal cortex and its related structures, which are considered among the critical areas mediating central executive functions. As a result of one or a combination of these causes, performance on tests of WM that depend critically on the central executive are expected to be impaired in dementia.

Baddeley (1986) and Morris (1986) cite the drop in retention on the Brown-Peterson test as evidence of a WM deficit in patients with Alzheimer's disease. That deficit may be caused as much by the central executive's reduced ability to coordinate rehearsal of the items in the face of even limited interference as by the lower level of functioning of the other mechanisms that Baddeley (1986) postulates comprise WM. Similar deficits are observed in patients with closed head injuries (Levin 1990). It is important to note, however, that patients with unilateral left or right frontal lobe lesions perform normally on the Brown-Peterson test (Corsi 1971, cited in Milner 1974) and on Daneman and Carpenter's (1980) reading span test (Frisk 1988), which is also considered a prototypical test of WM. Although it is still possible that the prefrontal cortex mediates some other functions of the central executive, it is unlikely that the WM deficits in dementia that are inferred from these two tests are caused by frontal degeneration alone.

Deficits in Allocation of Attention: Hemineglect and Related Syndromes Although it is not always possible, it is important to distinguish between deficits in attention caused by a malfunctioning central executive and those caused by an impaired processor that is summoned by the central executive. As a rule of thumb the former type of deficit should be broader, encompassing all domains, whereas the latter should be more restricted. The former entails a deficiency in the voluntary allocation of attention that is influenced by current and past knowledge and by motivation; the latter seems to be best described as

the loss of automatic attentional processes that can be overcome, or compensated for, by the recruitment of a more central, voluntary deployment of attention. The former is prototypically central; the latter, we suggest, shares characteristics with modules. Finally, the former is associated, as we noted, with prefrontal lesions, whereas the latter may be affected more by parietal lesions.

Some studies on allocation of attention in space help highlight the differences between these two types of deficits in attention. Damage to the frontal eye fields might initially produce a deficit in allocating attention automatically. However, damage to wider areas of the frontal lobe produces deficits in the voluntary deployment of attention. Guitton, Buchtel, and Douglas (1985) found that patients with frontal lesions cannot reorient attention voluntarily to a side of space contralateral to the lesion when attention is captured automatically on the opposite side. Also Alivisatos and Milner (1989) found that frontal patients do not benefit from a foveally presented stimulus that provides information about the probable location of targets in space. In short they seem unable to make use of expectancies to direct their attention voluntarily in space.

Patients with hemineglect that arises from parietal damage (Heilman, Watson, and Valenstein 1985), however, seem to suffer from a deficit in the automatic deployment of attention. In contrast to the patients with frontal lesions, those with parietal lesions can often benefit from information about the probable location of a target, even if it appears in the neglected field, and can direct their attention toward the target voluntarily in response to a cue (Posner, Cohen, and Rafal 1982, Posner, Walker, Friedrich, and Rafal 1984). They fail, however, to pick up targets that occur unexpectedly in the neglected field when their attention is engaged elsewhere. According to Posner, Inhoff, Friedrich, and Cohen (1987), orienting of attention in space, even when it is unaccompanied by eye, head, or other body movements, can be subdivided into three phases: disengagement of attention from the attended location, movement toward a new position, and engagement of attention there. Parietal lobe lesions impair the disengagement of attention, a process we propose is initiated automatically by the appearance of the stimulus. As will be seen in the next section, we conceive of parietal neglect as a failure of access to WM (and consciousness).

Failure of Access to WM: Deficits in the Automatic Deployment of Attention

Detecting a salient stimulus change, we propose, is a modular process that delivers its output to WM. The domain is not a particular physical feature—it is a change in background stimulation (Jonides

and Yantis 1988). The output is shallow; it provides information that a change occurred, not about what that change was. Once noticed, voluntary attentional mechanisms are deployed to examine the change and interpret it. Whether detection of change is informationally encapsulated is a matter of controversy. Our belief is that expectancies operate postmodularly, after detection has occurred. Indeed one would not want expectancies to play a large role in this process, otherwise one would detect only the expected. In patients with parietal neglect the output of this process does not gain access to WM if the change occurs on the neglected side of space.

If such a detection mechanism exists, one would have to posit that there also exist modules in each sensory modality that are allocated to a particular region of space as it is internally represented and also a higher-order module (type II) in which these sensory-specific spatial maps are integrated into a common one. Salient changes in the information picked up by these modules are automatically delivered into WM. Deficits in deployment of attention arise when the output of these modules fail to gain access to WM. The phenomenon of neglect is not restricted only to the left or right side of space, but can occur even within various regions in front of the subject, as has been shown in monkeys (Rizzolatti and Camarda 1987, Rizzolatti and Gallese 1988). Such specific attentional deficits are exactly what is predicted if separate modular processes are involved. As with other modular processes, deficits in the automatic deployment of attention is rarely seen in the early stages of dementia, though focal lesions can produce a specific impairment (Heilman et al. 1985, Mesulam 1981).

Neglect of the left side of space is not restricted to situations involving stimulus change, but in extreme cases involves the left side of any stimulus or internal representation of it, whether or not it is changing (Bisiach and Vallar 1988). To be consistent, we propose that the subject has information available about the complete extent of the external world and of the stored imagined representation of a scene or object or word from a particular point of view. As with detection the output of the spatial modules of the left side of those representations cannot gain access automatically into WM. Because attentional mechanisms are thus allocated only to the right half of space, what is available to WM and consciousness is the right half representation of the object that is the focus of attention. This interpretation is similar to one offered by Bisiach and colleagues (1983, 1985). If attentional mechanisms are then deployed voluntarily to the neglected space, information there is picked up and consciously apprehended.

One aspect of this proposal that was provocative when we wrote the first draft in 1986, but has become more acceptable since then, is

that the information on the left, though neglected, is available to the subject at a preconscious level. As we discussed in relation to some cases of aphasia, prosopagnosia, and dyslexia, the output of modules, though not in WM and therefore unavailable to consciousness, are nonetheless available to other action or procedural systems. Their influence is detectable on implicit or tacit tests of knowledge. Evidence in favor of this view with regard to neglect is slowly accumulating. The neglect patient's midpoint of a line is influenced by the length of the line in the neglected field (Bisiach, Bulgarelli, Sterzi, and Vallar 1983). This suggests that neglect patients are using information in the neglected field, of which they are not consciously aware, to guide their behavior in bisecting lines. Similarly reading in patients who often ignore the left half of words is determined by the information on the neglected left side. Failure to read the left side correctly is greatest in nonwords (for example, *gencil*), less in compound words (*snowman*), and least in noncompound words (*pencil*) (Behrmann, Moscovitch, Black, and Mozer in press). Judging whether tachistoscopically presented stimuli on the neglected side are identical or different from other stimuli can also be done accurately, although subjects are unaware of the stimulus and believe they are guessing (Volpe, LeDoux, and Gazzaniga 1979). In a recent clever demonstration Marshall and Halligan (1988) showed two pictures of identical houses to a woman with severe left-neglect. One picture showed the house in flames on the left side and the other did not. Although on repeated tests the woman did not detect the flames, and believed the houses were identical, she reliably chose the nonburning house as the one in which she would prefer to live.

The similarity of these neglect phenomena with those in other domains reinforces our belief that both modular as well as central processes are involved in the deployment of attention. The same seems to be true of episodic memory, a topic to which we now turn.

Episodic Memory: Explicit and Implicit Tests

Episodic memory is memory for autobiographic episodes or events that retain a spatiotemporal context. Semantic memory on the other hand is concerned with general knowledge and is usually independent of the spatiotemporal context in which that knowledge was acquired (Tulving 1972). As with attention the key elements of episodic memory are input modules, WM (conscious awareness), and the central executive that regulates memory. To these another key element is added that is unique to episodic memory, namely, a process involved in encoding and consolidating information into long-term memory

and retrieving it from there. Mediated by the hippocampus and its related limbic structures, this process, we argue, is modular. Because our neuropsychological model of episodic memory has been presented in some detail elsewhere (Moscovitch 1989), we only sketch it briefly here.

First, though, we distinguish between two types of memory tests: Explicit tests of memory depend on the subject's conscious recollection of the experienced event. Implicit tests do not rely on the subject's conscious awareness of the remembered event, but merely investigate whether the subject's behavior was modified by it (Graf and Schacter 1985, Moscovitch 1984, Schacter 1987b). Because the mnemonic processes involved in these two types of tests are different, we examine each separately.

Explicit Tests of Memory Because conscious awareness of a previous episode is a necessary property of explicit tests, if follows that WM must be involved. In the sense that strategies and general knowledge are involved in encoding and retrieving what is to be remembered, remembering a previous event is no different in principle from attending to a stimulus or solving a problem. As in attention the entire process may be automatized and appear to be modular so that only the outputs are delivered to WM, in which case the memory simply pops into mind, much as some stimuli seem to pop out perceptually and grab our attention. We refer to this memory process as *associative*. Alternatively WM may be extensively involved at all stages so that the subject is aware not only of the remembered event but also of the strategies and knowledge used to bring that event to mind. We refer to this process as *strategic*.

We agree with Fodor that there is no limit to the type of knowledge that a person can use to try to remember a past event or the type of strategies he recruits to aid in the process. But these concerns apply primarily to memory processes we called strategic. Associative memory processes, as we shall see, are different. Besides, there are regularities in the processes that are used in remembering and in the specific breakdown patterns associated with brain damage. These suggest that there are subcomponents of memory processes that can be identified functionally and that are associated with particular neural structures.

According to our model (figure 1.1), the hippocampus facilitates the formation and retrieval of a cross-modular associative structure. An event, that is, the stimulus information that comprises it, is picked up by perceptual modules. They are modified by the process of decoding the information, thereby creating an engram of it. The output

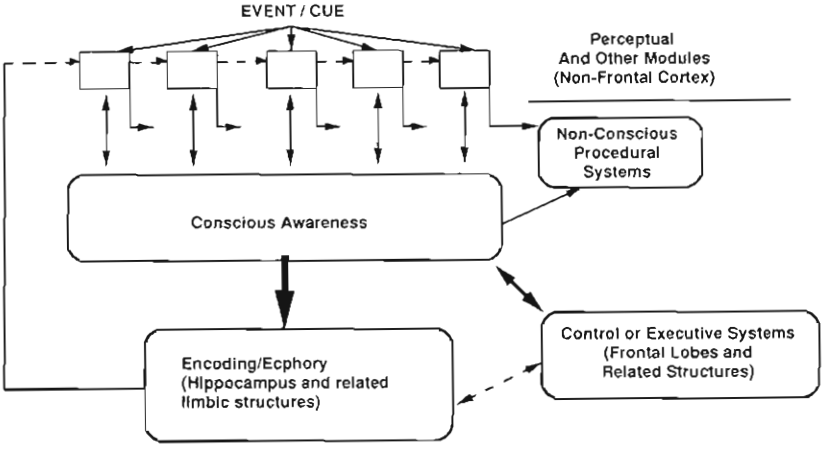

Figure 1.1
A neuropsychological model of memory. The dashed lines indicate that the interaction is optional. The cortical modules that interact with the hippocampal system will vary depending on the information about the event that is available to consciousness when the event is initially experienced and when it is being remembered. (See text for details. From Moscovitch 1989.)

of the perceptual modules are delivered to WM and to central systems that are involved in interpreting them. This consciously experienced, semantically interpreted event, or signals related to it, are then relayed automatically to the hippocampus and its related limbic structures. The hippocampus in turn mandatorily binds the information it receives with the engrams in the various modules and central processes that recorded the experience. Simultaneously the hippocampus encodes all the bound information as a file entry. We propose that consolidation is the process of establishing a long-lasting record or file entry of the encoded event information.

At retrieval an externally presented or internally generated cue enters WM and automatically interacts via the file entry in the hippocampal system with the stored engram in the modules and central systems. The product of that interaction yields an output that enters WM and forms the basis of our conscious experience of remembering. This automatic, mandatory "process by which retrieval information is brought into interaction with stored information" is called *ecphory* by Tulving (1983, p. 169), a term along with *engram* that was first coined by Semon (1921, cited in Schacter, Eich, and Tulving 1978). The information thus retrieved is a product of the cue-engram interaction and may or may not be veridical. Because our colleagues found the term *ecphory* abstruse, the term *automatic retrieval* can be substituted

for it. The processes of consolidation and automatic retrieval are all subsumed under associative memory processes.

This associative system, with the hippocampus at its core, we suggest is modular. The specific domain of this module is the information available in WM. Because that information is usually semantically interpreted, semantic information is typically more useful in remembering a past experience explicitly than is information about the sensory attributes of the stimulus. The shallow output of the associative system is ecphoric, or automatically retrieved, information usually about semantic attributes of the stimulus, but also about sensory attributes if they received attention at encoding. Once initiated, the hippocampally based process is rapid, obligatory, informationally encapsulated, and cognitively impenetrable, that is, once the cue is presented, memory retrieval is automatic and "stupid."

The hippocampal-associative memory system also meets the neuropsychological criteria of modularity. Bilateral damage to the hippocampus produces highly circumscribed deficits restricted to the specific domain of the system—explicit memory for consciously apprehended information. Other cognitive functions, and even performance on other types of memory tests, are relatively unaffected. Conversely episodic memory can be relatively preserved in individuals with semantic memory loss, if the disorder spares the hippocampal formation (Warrington 1982, De Renzi, Liotti, and Nichelli 1987).

Yet, as we noted at the outset, memory often is more akin to intelligent behavior such as problem solving than to the stupid, reflexive behaviors one associates with modules. What confers "intelligence" on memory are the *strategic processes* associated with the prefrontal executive system and other central systems. These strategic processes coordinate, interpret, and elaborate the information in WM to provide the hippocampal-associative-memory system with the appropriate encoding information and retrieval cues that it takes as its input. Comparable processes are involved in evaluating the hippocampal system's shallow output and placing those retrieved memories in a proper spatiotemporal context. What makes us conscious of the various processes involved in memory search are not the workings of the hippocampal-associative system but the operations of the strategic system that occupy WM. We are aware of the questions we deliver to the hippocampus, the answers we get from it, and the evaluation of the answers, but we are not aware of the ecphoric operations of the hippocampus itself (Moscovitch 1989).

As might be predicted, memory impairments associated with prefrontal damage do not occur in isolation but invariably are accompa-

nied by related cognitive deficits in other domains. In patients with recovery of frontal functions, the memory impairments resolved along with the other frontal deficits (Kapur and Coughlan 1980, Stuss et al. 1978).

The type of memory deficits seen after damage to the frontal areas of the brain do not involve item information but rather information about temporal or spatial context related to a particular item (Schacter 1987a, Moscovitch 1982a, Milner 1974, 1982). Memory for temporal order (Milner 1974), list differentiation (Cermak, Butters, and Moreines 1974, Moscovitch 1982a, Huppert and Piercy 1976), monitoring productions (Petrides and Milner 1982), and frequency of occurrence (Smith and Milner 1984) are all impaired following frontal lobe damage. It is not surprising that this is the case because we have already noted that central processes associated with the frontal lobes are important in learning sequences of movements and in keeping plans in proper sequence. Presumably similar or identical processes are involved in remembering contextual information about experienced events.

Perhaps the most striking illustration of an associative memory system operating without the control of the strategic system can be seen in patients who confabulate. These patients typically have large frontal lesions attested by radiological evidence and frontal dysfunction as demonstrated by their performance on psychometric tests. These patients "haphazardly combine information from disparate events, jumble their sequence, and essentially accept as veridical whatever the ecphoric process delivers to consciousness. The minimal organization that their memories show is dependent on loose rules of plausibility and association rather than on systematic strategies aimed at recovering additional ecphoric information. In cases of fantastic confabulation (for example, Berlyne 1972, Stuss et al. 1978), retrieval information interacts with whatever information is currently active in the perceptual and semantic modules to deliver ecphoric information that reflects recent thoughts, perceptions, of fantasies rather than relevant past experiences" (Moscovitch 1989, p. 155).

Significantly recall is much more severely affected in many confabulating patients than recognition, suggesting that recall depends more on central strategic process than does recognition. Recognition may provide a more veridical measure of the operation of the hippocampal-associative-system. In patients with bilateral hippocampal damage, both recall and recognition are severely impaired.

The effects of loss of cognitive resources are consistent with the interpretation of the involvement of associative and strategic processes in memory. Aging, intoxication, fatigue, depression, and some

forms of dementia that spare the hippocampal formation have their initial and greatest effects on those tasks in which encoding and retrieval are highly dependent on strategic processes. Thus list differentiation and source attribution, two tests that require resolution of temporal context, are affected early in aging (Craik 1977, McIntyre and Craik 1987). The high susceptibility to interference seen in the elderly may have a similar source (Winocur 1982a,b). Similarly, free recall is almost always more impaired than recognition. Providing subjects with appropriate encoding and retrieval strategies and cues to compensate for their lack of resources improves their performance (Craik 1977, Craik and Byrd 1982). Similar effects are observed in patients with Huntington's disease, whose memory impairment is associated with caudate-frontal dysfunction (Butters, Salmon, Heindel, and Granholm, in press). Again, as expected, manipulations such as those that involve strategic processes have little lasting effect on amnesics with bilateral hippocampal damage (Cermak and Reale 1978, Cermak 1982).

The distinction between the effects of damage to frontal strategic systems and hippocampal-associative systems is also observed in performance on tests of recent and remote memory. Temporal ordering of both recent and remote memories is impaired in patients with frontal damage or dysfunction (Milner 1974, Moscovitch 1989, Rubin 1986, Sagar et al. 1988a,b, Shimamura, Janowsky, and Squire 1988). This is consistent with the idea that equivalent strategic processes are operating in all domains. In contrast damage to hippocampal-associative memory typically affects recent memories much more than remote ones (Milner and Scoville 1957, Milner 1966, Rubin 1986, Squire and Cohen 1982, Squire, Cohen, and Nadel 1984, Marslen-Wilson and Teuber 1975), although there is not universal agreement on this point (Warrington and Sanders 1971).

Let us assume that the differential effect on retrograde and anterograde memories is real. How can we account for it? The only solution we envisage is that there is a nonhippocampal route to memory traces that also uses WM. This suggestion implies that the hippocampus is necessary for encoding and automatic retrieval only for a short time after the experience. Subsequently alternate routes to those traces can be established that use central processes or the traces can be revived and their content delivered to WM by newly-established automatic associations for those pieces of information that have been retrieved often (Schacter 1989, Squire, Nadel, and Cohen, 1983, Moscovitch 1989).

It follows from this account that old memory traces are retrieved differently from new ones. For infrequently remembered events their

retrieval may be more laborious because they depend on central processes. (Yet if those events had been retrieved only once or twice, they might still require a hippocampal route guided by WM.) For events that have been experienced or remembered frequently, the process would be relatively rapid and automatic, requiring simply their reactivation through firmly established connections outside the hippocampal system. Those memories, moreover, may be quite stereotyped because they are essentially automated subroutines. It would not be unthinkable to say that semantic memories and automatized episodic memories are equivalent in this way (see Cermak 1984 for a similar suggestion).

This type of analysis allows us to rethink the distinction between semantic and episodic memory. After hippocampal lesions it may be as difficult to recall new semantic memories as it is to recollect new episodic memories. What distinguishes semantic from episodic memory is the autobiographical context in which the latter is embedded and that can be used for retrieval. Newly acquired semantic information is learned and retrieved in probably the same manner as episodic memories. With practice semantic memories may be retrieved via a nonhippocampal route. Old semantic memories may be immune from hippocampal damage for that reason. Old, nonroutinized semantic memories may be as laboriously retrieved after hippocampal lesions as nonroutinized episodic memories (Butters and Cermak 1986, Goldberg, in press, Goldberg and Bilder 1986b, Moscovitch 1989, Tulving, Schacter, McLachlan, and Moscovitch 1988).

Implicit Tests of Memory Unlike explicit tests of memory, like recognition and recall, which require conscious recollection of the past, implicit tests assess memory by measuring the effects of past experience on performance. Even a single presentation of a stimulus item is sufficient to improve one's ability to classify, identify, or generate that item if it is presented again in a complete or degraded form or if it is elicited by perceptual or conceptual information. This improvement can occur though the subject may not explicitly remember having been exposed to that item (Schacter 1987, Shimamura 1986).

If the role of the hippocampus is to make information about recent experiences available to WM, then one possible implication is that without the hippocampus information about these experiences is stored, but unavailable to consciousness. Neither the hippocampus nor the frontal systems are the repository of memory traces, nor are they necessary for storing these memory traces, though hippocampal input might strengthen them once they are laid down. As we indicated in the previous section, we believe that these traces reside in, or

are represented by, the modules and central systems involved in encoding the event when it first occurred. As we noted earlier, type II and type III modules are modifiable by experience. The modules store new information relevant to their own function, but they have no access to other memories; their memories are domain specific. On the other hand the information stored in central systems or accessible to them is generic. Thus there is no unique location where memories are stored. Instead we believe, along with others (Squire 1987, Schacter 1985, Mishkin 1982), that memories are distributed in the brain and associated with structures, be they modular or central, involved in processing different kinds of information.

On implicit tests of memory the information and procedures in those modules and central systems can be reactivated directly by priming, rather than through the hippocampal-associative system. With experience or practice the output of the module and associated central systems are delivered more quickly and efficiently. It is this change that serves as an index of memory on implicit tests. We can become aware of the output of either the modules or the central systems, but have no conscious recollection of the previous experience that makes this altered (or speeded) output possible. It is only when the modules and central systems are activated through the hippocampal-associative system that we experience the retrieved information as a memory, as having a sense of familiarity and pastness. As expected, performance on implicit tests of memory is spared in amnesia caused by damage to the hippocampal-associative memory system (for reviews, see Moscovitch, 1982a, 1984, Schacter 1987a, Squire 1987).

A similar proposal has recently been advanced by Schacter (1990). His analysis suggests that research on the effects of structural/sensory features versus semantic attributes on implicit tests of memory may provide a way of distinguishing between the contribution of modular and central processes. The sensitivity of many implicit tests primarily to sensory or structural features of the stimulus, but not to semantic features, suggests that performance on those tests is driven primarily by the output of modular processes that lack semantic content. This is not unexpected because many implicit tests have a strong perceptual component and are believed to be perceptually, or data, driven. In some cases, however, semantic elaboration during the study phase does influence performance on implicit tests. It is significant that in such cases either new associations are formed during the study phase or the implicit tests are conceptually driven rather than data-driven. Instead of it being an implicit test, say, in which the degraded material must be identified, it is a test in which responses must be gener-

ated to semantic cues (Roediger, Srinivas, and Weldon 1989). Our model would suggest that performance on implicit tests that are sensitive to semantic manipulations is mediated by the reactivation of old or newly formed associations in central systems or perhaps type III modules. A prediction that follows from this suggestion is that performance on conceptual, but not perceptual, tests of implicit memory would be impaired in dementia. Recent findings that Alzheimer patients are more impaired on conceptual- than data-driven tests is consistent with our prediction (Butters 1990, Gabrielli 1989). Similarly, in conditions that produce a more modest loss of cognitive resources, such as aging, performance on implicit tests of memory is spared only insofar as it depends on reactivation of perceptual modules (Moscovitch 1982b, Moscovitch et al. 1986, Light and Singh 1987). If central or strategic processes need to be recruited to perform the test at hand, as in learning to solve the Tower of Hanoi in its various versions, then performance will be impaired and possibly associated with frontal dysfunction (Shallice 1982, Saint-Cyr, Taylor, and Lang 1988).

Though the outcome of central and modular processes are delivered to WM on implicit tests, and we use WM to gauge changes in performance, there is no reason why working memory cannot be bypassed entirely. One can imagine that other testing procedures, such as measuring SCR or event-related potentials (ERPs) or heart rate, might also reflect the acquisition of new information, though the subject might not be aware at all of the input that was used to trigger these responses. Some progress has already been made in that direction (for SCR, see Rees-Nishio 1984, cited in Moscovitch 1985; for ERP, see Bentin and Moscovitch 1990).

The traces that are laid down and accessed by implicit tests of memory are the same traces that are accessed by explicit tests of memory. It is the process by which they are accessed that leads to different phenomenological experiences. Also because on explicit tests one is conscious of having experienced an event and of retrieving information about it, conscious recollection is amenable to strategic influences and other operations associated with central processes, whereas memories that are retrieved implicitly cannot be influenced by mnemonic strategies at retrieval.

Because the hippocampal-associative memory system receives as its input the contents of WM, bilateral damage to the hippocampus produces an amnesia that affects explicit tests of memory in all domains. If our hypotheses concerning the processes that mediate performance on implicit tests are correct, such global deficits should not be observed on implicit tests after damage to any single system.

Deficits on implicit tests of memory should be restricted only to performance on tests that directly implicate the affected module or central processor.

The evidence on this matter is sparse, but consistent with this prediction. Patients with Parkinson's disease are impaired on implicit tests involving motor learning and frontally mediated problem-solving (St.-Cyr et al. 1988), but perform normally on word-stem completion and reading of geometrically transformed script (Butters et al. in press, Heindel et al. 1988, Huberman, Freedman, and Moscovitch 1988). Patients with Alzheimer's disease, who have a semantic memory impairment, perform poorly on word-stem completion of infrequent words (Shimamura, Salmon, Squire, and Butters 1987), but not of frequent words (Huberman et al. 1988), and have no difficulty acquiring motor skills or learning to read transformed script early in their disease (Moscovitch, Winocur, and McLachlan 1986).

An interesting prediction that follows from our analysis concerns patients with agnosia, aphasia, or dyslexia whose deficit can be interpreted as a failure of modular output to gain access to consciousness. In those patients in whom modular processes can be shown to be intact, but dissociated from consciousness on tests that do not involve episodic memory, it should be possible to demonstrate normal performance on implicit tests of memory for information coded by those intact modules. Because the outputs do not gain access to consciousness, performance on explicit tests for that information should be severely impaired. For material not processed by those modules, performance on implicit and explicit tests should be normal. To our knowledge, there are no published studies with evidence that can be used to determine whether these predictions are correct.

Conclusion

The conceptual framework of modules and central systems that we developed in the first part of this chapter was used to construct neuropsychological models of attention and memory in the second part. In these models it is assumed that more processing goes on in our minds than enters our awareness. Consciousness can be identified with the phenomenal experience of the contents and operation of a limited-capacity central system. This system can also control cognitive processes to some extent by selectively allocating attention to some mental representations and cognitive processes at the expense of others. As a result those receiving attention become conscious, whereas the others remain nonconscious. In other words the notion that emerges is that of consciousness as the experiential equivalent of

a central processor that selectively receives and operates on the input of the multitude of nonconscious, modular processes (Umilta 1988a).

In recent years this notion has been supported by neuropsychological studies that have provided evidence of a dissociation between implicit and explicit forms of knowledge. The dissociation takes the following form: A brain-damaged patient is asked to perform a task that requires the explicit use of his or her impaired function. Not surprisingly a severe deficit is observed. When, however, the same patient is asked to perform another task that also taps the impaired function, but in an implicit manner, performance may be quite good, sometimes even normal. Apparently the patient does not have conscious access to the necessary knowledge, even though that knowledge can be accessed unconsciously. Variants of this striking dissociation between normal or near-normal performance on tests requiring implicit knowledge and severely impaired performance on tests requiring explicit knowledge have been observed in many neuropsychological syndromes, some of which have been mentioned in the preceding sections of this chapter. In our view, and in accordance with a proposal by Schacter and coworkers (1988), the implicit/explicit dissociation shows that (1) conscious or explicit experiences depend on the activity of a common mechanism, the central processor, (2) this mechanism accepts domain-specific input from modules, and (3) neuropsychological impairments can originate because the output from the modules are disconnected from the central processor. Of course the central processor itself is not completely damaged, and thus a global disruption of consciousness does not occur.

There are alternative explanations of explicit/implicit dissociations that are compatible with the type of neuropsychological models we have proposed. Multiple conscious mechanisms may exist, in which case the dissociation results from a disconnection between the module and its specific central processor (Schacter 1990). It is also conceivable that modules project degraded outputs that are sufficient for performance on implicit, but not explicit, tests of knowledge. This possibility is not likely, given that performance of patients resembles that of normal people on the same implicit tests (see Schacter et al. 1988). Alternatively, if damage occurs to a module needed for demonstrating explicit knowledge, performance may then depend on another module that does not have access to the central processor.

Dissociations between performance on implicit and explicit tests constitute only one source of evidence on which our models are based. Neuropsychological deficits can arise from damage to the modules themselves and from damage to central systems that mediate the control processes or functions of the central processor. In this

chapter we also tried to indicate how the specific disorders that can arise from these causes can be distinguished from those involving disconnections between modules and the central processor.

According to the models we developed, attention and memory are composed of both central systems and modules. By specifying the functions of central systems and modules and how they interact, as well as by indicating the critical role played by consciousness (and nonconscious processes), the models were able to explain a variety of phenomena in attention and memory of both normal and brain-damaged people. The models also predicted the existence, or presaged the importance, of other phenomena that had been previously overlooked, but that had attracted attention since we first began work on these models in 1986. Among the latter phenomena are the dissociations between implicit and explicit knowledge in neuropsychological syndromes, such as neglect, that had not figured prominently in the literature (Behrmann et al., in press, Marshall and Halligan 1988, Schacter et al. 1989); the distinction between memory disorders that are primarily associative and those that are strategic (Moscovitch 1989, Shallice 1988); and the separation of implicit memory tests, at both the psychological and neurological level, into those that are conceptually driven and those that are perceptually or data driven (Schacter 1990, Roediger, Srinivas, and Weldon 1989). It must be admitted that both the discovery of the phenomena and their coming into prominence owed far less to our models than to the research of other investigators who were working independently of us. Nonetheless it is good to know that we are on the right track. This knowledge encourages us and, we hope, others to develop such models more fully and to use them to guide future research.

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Notes

1. The patient's performance can be interpreted as disconfirming Fodor's proposal that the output of visual perceptual modules are basic perceptual categories, that is, that if the input is the visual image of a poodle, the output is "dog" and not poodle or animal.
2. One cannot invariably conclude that interlevel representations are being tapped whenever performance is influenced by information of which the subject is unaware. It is equally possible that the output of a module or the outcome of computations performed by the central processes can also affect behavior without gaining access to consciousness.
3. Sometimes dissolution of function only makes it *seem* as if interlevel representations are available to consciousness, but in fact what occurs is a reorganization of function or change in information processing consequent to brain damage. For example, one type of acquired dyslexia, letter-by-letter reading, may be interpreted as making available to consciousness an intermediate-level representation in reading—namely, individual letters—that ordinarily seem to be identified automatically and without conscious access. A more likely possibility, however, is that the damage has forced the subject to process information in a way not ordinarily used in normal reading (Patterson and Albert 1982). In short we are seeing the emergence not of intermediate-level representations in the operation of a module but of a new strategy that probably depends on central processes that bypasses the modular route entirely. Indeed the same subjects who read letter-by-letter consciously seem able to pick up a great deal of lexical, even semantic, information about the word, at a preconscious level. This kind of recruitment of central processes to take over cognitive operations that may have been modular is not uncommon after brain damage. Processes that were once effortless and automatic now require conscious involvement and painstaking effort. Luria's (1972) book, *The Man with a Shattered World*, gives a good feel for what it means to depend on central processes to make perceptual sense of a world with distorted input from defective modules. Similar examples can be found in Brodal 1973 with regard to movement and in Moss 1972 with regard to speech. Yet it is intriguing to think that in many of these patients, the perceptual, input modules still operate normally—only their output is prevented from reaching consciousness.
4. One additional criterion of modularity, computational autonomy, was not listed among the critical ones because no one, even Fodor, mentions it explicitly. Computational autonomy implies that computations that a processor carries out cannot be altered. Thus if a processor applies a particular algorithm to its input, that algorithm will not change in response to external influences.
5. Under some circumstances central systems may be as computationally autonomous and informationally encapsulated as modules. This is particularly noticeable when one falls into a set way of solving problems and making stereotypic social attributions. Because it is possible to alter performance or social perception by knowledge outside the specific domains involved, the processes are deemed to be central.
6. Such highly specific deficits have yet to be reported in humans, but there is no reason to believe that they cannot be found if a person with a highly restricted lesion is properly tested.
7. These processes may also be involved in setting up expectancies and as such may affect perception at a postmodular level.
8. We are aware that all phenomena that have been explained by invoking resource limitations can be explained equally well by alternative, sometimes simpler accounts

(Navon 1984). Nor is it clear how the concept of resources is translated in neurological terms. Ideally one would want to specify the cognitive operations and neural structures that are typically affected by conditions such as depression, fatigue, heightened motivation, aging, and dementia and that are believed to alter cognitive resources. This goal has not yet been attained. In the meantime we find it useful to retain the concept of cognitive resources for its heuristic value.

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