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Attention, Memory, and Concepts in Autism.

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Abstract*

In this paper, it is hypothesized that many of the behavioral abnormalities found in autistic persons result from deficits in fundamental cognitive abilities. Memory and attention are the most likely candidates. The memory deficit may be primarily one of retrieval, possibly exacerbated by an encoding deficit. However, both types of memory deficit are probably the result of a primary deficit in attention. This is supported by the observation that the autistic memory deficit resembles that following frontal lobe, rather than mediotemporal lobe, damage. This and other evidence is used to draw a parallel between autism and frontal lobe syndrome. In light of this analogy, how a primary deficit in the fundamental cognitive ability of attention may be responsible for the more secondary autistic deficits in memory and more advanced forms of cognition, such as language acquisition, symbol manipulation, rule extraction, and social interaction, is explored.

Introduction

Several theories have been posited about how the minds of autistic people differ. Some suggest that autistics have an altered theory of mind (Baron-Cohen, 1989). Hobson and Lee (1989) consider the disorder to be primarily one of affect, and still others emphasize the contribution of fundamental cognitive abilities (Gillberg, 1990). This last is the stance argued for here.

It is hypothesized that underlying deficits in attention give rise to a host of other cognitive deficits in autistic persons, such as those in memory, language, and certain thought processes. Because research on basic cognitive abilities in autistic people is relatively sparse, the evidence to be presented is only suggestive. Nevertheless, the ideas put forth in this paper may serve to guide much needed research into the mechanisms which may underlie the variety of behavioral deficits in autistic persons.

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Many cognitive abilities of autistic people differ from those of the general population. There are some areas in which they seem markedly deficient and some in which they are relatively spared, or even unusually proficient. Their apparent social withdrawal not withstanding, the most striking cognitive deficit is in language. It has been estimated that as many as 19% of autistic children of 8-10 years are mute, and 31% speak only some words, though not in conversation (Ricks and Wing, 1975).

Level of language attainment has been correlated with estimates of intelligence (Ricks and Wing, 1975), suggesting a relationship between language and other cognitive abilities. This may best be understood within the framework of the mind as instantiated within a massively parallel and distributed neural system in which there is extensive feed back between systems, the reality of the neocortex. Within the neocortex it is likely that different systems that interact with each other are likely to mutually modify the information processing within each other. The extensive back projections between areas of neocortex indeed support that the effect of a system A, such as attention, feeding forward onto a system B, such as language, may depend upon the nature of the information processing within system B (language). Thus the nature of the neural machinery may enable language systems to affect how attentional systems affect linguistic processing.

Such effects may be most apparent within the context of cognitive abilities which rely heavily on both language and attention. Specifically, symbol manipulation, inference, or deduction, all of which may have been enabled or facilitated by the development of language, may also depend upon systems subserving attention. Autistic persons experience difficulty with tasks requiring symbol manipulation. If their attentional systems are damaged, assuming the symbol manipulation task requires both attention and language, their deficit may most accurately be described as one of attention that creates a deficit in one, the other, or both, language and/or symbol manipulation. This paper focuses upon how deficits in more fundamental cognitive abilities, such as attention, memory, or emotion, may result in language disabilities.

Another reason to shift the focus of research from language to other basic aspects of cognition is that language is not likely to be one of the most basic cognitive abilities (Bates, 1990). This may be argued at least from an evolutionary standpoint. Evolution usually acts by building upon already existing structures (Killackey, 1987), and language ability is one of the most recent to evolve. Thus, it probably depends upon the existence of other cognitive abilities for its function, such as attention, memory, and perception.

Basic Cognitive Deficits

In this regard, memory and attention are likely candidates. Memory is a ubiquitous property of the brain, and attention is known to modify memory. Indeed, both memory and attention seem to be abnormal in autistics. A course of study in which of the interaction of memory, attention, and then language, and the emergent properties to which they give rise, are examined, is more likely to yield a logical and coherent account of the structure of the cognitive deficits in autism. This is in contrast to the approach of addressing the problem exclusively at a high level of cognitive description without consideration of possible underlying, more basic, deficits. However, high level descriptions are necessary to define the problem so that the neural mechanisms which underly them may be discovered. Such descriptions may even be suggestive of the underlying mechanism. Nevertheless, once a high level description of the problem has been put forth to guide research, it may be more fruitful to concentrate on cognitive abilities that are the cornerstones of more complex cognition and build up from there, likely redefining the problem itself. Such an approach is advantageous also because fundamental cognitive abilities should be simpler, relative to higher order cognition.

Memory

Memory and Autism. Paradoxically perhaps, memory is one of the areas of cognition in which autistics seem to be relatively spared, or even especially able. From as early as Kanner's (1943) original article, autistics have been considered to have prodigious rote memory skills, to such an extent that as many as 9.8% of them are categorized as idiot-savants. Nevertheless, a memory deficit could account for at least the memory-dependent cognitive deficits in autism, including their language problems. For example, autistic children tend to use a holistic approach for language acquisition, as well as for other learning situations. This has been proposed to be the reason for their extended echolalia, in which they just tend to repeat back what has been said. (Prizant, 1983). Prizant (1983) suggested that autistics have an impaired semantic memory system, while their episodic memory system, which enables rote memory, remains intact. For example, autistics have trouble segmenting sentences into the meaningful parts which would be stored in a semantic memory system. While current research on memory would modify these proposals, this example illustrates the role that a memory deficit could play in the development of memory dependent cognitive abilities in autistic people.

Further support for the role of memory deficits in the autistic syndrome comes from a number of researchers who have sought to draw a parallel between autism and the amnestic syndrome (Boucher et al, 1976; Heltzer, et al, 1981). This research avenue is supported by the marked learning disabilities of autistic children. After all, memory is the result of the learning process, though other information processing deficits may also be involved. Those who favor a memory explanation of the autistic disorder prefer to attribute the biological abnormality to the mediotemporal lobes. Damage to these structures has been found both in animals and in humans to result in profound anterograde amnesia, as well as some retrograde. This amnesia, however, is restricted to explicit, or declarative, memories (Squire, 1987), accompanied by marked sparing of implicit or procedural memories.

The seminal paper on memory in autistics was that by Boucher and Warrington (1976). Their main finding was that, over a 30 or 60 second interval, autistics were impaired on free recall and recognition, but performed at essentially normal levels on tests of cued recall. In a further series of studies, Boucher (1981) looked into the nature of the memory deficit and sparing in autistics. In general, it was confirmed that autistics do indeed have good cued recall abilities (Boucher, 1981). However, they are impaired at free recall of recent events, and lack any ability at face recognition (Boucher, 1981). Retrieval from memory by autistic persons requires stronger cueing than in normals. Their memory ability, rote learning, like cued recall, is externally cued or predominantly self-cued. It may be this facet which allows rote memory to exist and flourish in the the autistic person, serving well under some, but not all, behavioral conditions. Whereas cues serve well to evoke remembrance in autistics, such people must rely more heavily upon them. Autistic persons apparently are unable to evoke memories internally and spontaneously without distinctive retrieval cues, and will seem to be impaired on tasks requiring free recall. Thus, abnormal retrieval of stored knowledge may be at least partly responsible for both their memory abilities and deficits. With regard to idiot savants, in the absence

of a normal memory system, autistics may overuse their intact abilities, such as rote memory, permitting some of them to appear to be mnemonists.

In general, these findings argue against mediotemporal lobe involvement. Mediotemporal lobe amnesia is thought to arise from a deficit in encoding. Such brain injured people cannot consolidate new information, but they have no trouble recalling information acquired prior to their lesion. This suggests that people with mediotemporal lobe lesions have intact retrieval mechanisms. Rather, their deficit is primarily one of encoding. Thus, the autistic memory deficit cannot be solely attributed to an abnormality in mediotemporal lobe function analogous to the amnestic syndrome, though there may be some subtle involvement of this brain region. Nevertheless, autistics do have a memory abnormality, but it may have to be attributed to another cause. The alternative to be explored in this paper is the parallel between frontal lobe syndrome and autism. In addition to the behavioral analogies to be presented, Gedye (1991), in an extensive survey of the literature, has suggested that "the variety of etiologies that cause frontal lobe seizures also accounts for the variety of etiologies traced to autism." Thus the neurology of frontal lobe disorders and autism supports the behavioral parallels.

Frontal Lobe Syndrome and Autism. Memory abnormalities are found in people with lesions of the frontal lobes. This form of memory disorder is qualitatively different from that of organic amnesia. In a well controlled series of studies, Janowsky, et al (1989) found that , in contrast to previous studies showing greater deficits, on most types of memory tasks, frontal lobe patients performed near normal levels, particularly relative to amnesiac and Korsakoff's syndrome patients. They therefore concluded that frontal lobe syndrome does not involve the kind of global amnesia present in mediotemporal patients. Nevertheless, frontal patients did tend to perform at lower levels than the control groups, though this finding did not reach statistical significance. For example, frontal lobe patients were somewhat impaired on free recall. This is congruent with similar findings in autistics, though the evidence suggests that autistics may be more impaired (Boucher et al, 1976; Boucher, 1981). A number of Janowsky, et al's (1989) findings are consistent with those of autistics, including recognition (Ameli et al, 1988) on which both perform well and word fluency (Boucher, 1988) on which both groups are impaired.

This last finding is particularly interesting. Impaired word fluency in frontal lobe patients has been interpreted as a reduction in the fluency and spontaneity of complex behavior (Shimamura, et al, 1991). In contrast, while autistics generated fewer miscellaneous words, Boucher (1988) found that they performed near normal when provided with a category. Perhaps autistics find a category name a better cue than do frontal patients. The autistic people's deficit at free generation in the miscellaneous condition, nevertheless, is consistent with their impaired free recall and their known lack of creativity. Both groups may be less apt to spontaneously search their knowledge base to generate responses. However, when a strong contextual cue is provided, active search may be facilitated, or may be less necessary for performance.

These considerations may suggest that neither autistic persons nor frontal lobe patients have problems consolidating memories of an experience, as do organic amnesiacs. Rather, the problem may be of a different nature: retrieval (Shimamura, et al, 1991). Retrieval from memory involves either implicit, nonconscious, or explicit, conscious, use of stored information. Explicit retrieval is an active process that may require the participation of attention, which may be the mechanism whereby the internal structure of knowledge may be actively searched. Thus, so called explicit memory deficits in frontal patients and autistics may be more fundamentally ones with mechanisms of attention rather than with memory, per se. It is also possible that their retrieval difficulties are exacerbated by a deficit in encoding that results from the interaction of attention with the explicit memory system. Thus there may be a parallel between the dynamics of learning and that of retrieval. This parallel may be that both involve attention. Some form of attention may direct what gets encoded, while the same or a different mechanism of attention aids retrieval.

Attention

Frontal Lobe Syndrome and Autism. With this in mind, the majority of the memory deficits in frontal lobe patients are thought to be associated with their inability to plan and organize their behavior (Mayes, 1988). Learning and memory that require the initiation and maintenance of effortful and organized strategies of encoding and/or retrieval, as well as the ability to switch from one strategy to another pose the greatest problems for frontal lobe patients (Mayes, 1988). This has indeed suggested to some that the frontal lobe syndrome may involve a deficit in sustained attention. *Effortful* and *maintenance* suggest that attention is required, as does *switching ability*. Attention provides the organism with a way to orient, maintain, and shift its awareness to different parts of its knowledge base, to different perceptual systems, and to different aspects of them. Frontal lobe patients may have poor planning ability because they cannot maintain attention. Thus, deficits in mechanisms of attention may disrupt the information processing involved in planning which may lead to an inability to

form complex memories, as well as hindering memory retrieval processes.

Attention and Working Memory. These neuropsychological considerations are supported by animal studies of prefrontal working memory, which evolved out of STM research in psychology (Goldman-Rakic, 1989). These studies focus on spatial working memory. Spatial processing involves the parietal lobes, as well as prefrontal, and the parietal lobes have also been implicated in attention.

The relationship between working memory and short term memory (STM) is also important because attention and STM are thought to be closely related. The connection may be that sustained attention is required in order to hold and place information in STM. Frontal patients tend to do poorly on STM tests, such as digit span (Shimamura, 1991). Thus, this may be due to an attentional deficit. It was once commonly thought that memory formation proceeded from STM to long term memory (LTM). Thus, any deficit in STM would also produce a deficit in LTM, or in one's overall knowledge base. Currently memory researchers do not bring STM studies into their theories, nor do most explicitly consider attention. It will therefore be suggested here that frontal lobe patients are abnormal in the way that attention acts on the working memory modules of the frontal lobes. The primary deficit in patients with frontal lobe lesions may be due to the extensive disruption of cortical connectivity necessary for attention to bind cortical modules together both spatially and temporally in a way that is necessary for the performance of complex behaviors, especially those requiring good STM.

Other evidence that supports the frontal lobe parallel are the findings that both populations seem to have a tendency toward perseveration of no longer appropriate responses, as well as in solving problems for which they cannot use a well established routine (Mayes, 1988; Shimamura, et al, 1991). Both of these are strong characteristics of both syndromes. There are several explanations for such behaviors, but the one relevant to this paper is that perseveration may result from inability to shift attention away from a previous problem and well established routines may be required if one cannot shift attention so as to rapidly acquire, or shift to a contextually appropriate, a motor pattern.

Counterevidence. However, there is some evidence that argues against drawing a parallel between frontal lobe syndrome and autism. One problem is that digit span is the part of the WAIS IQ test on which autistics are relatively facile (Lincoln, et al, 1988), arguing against an attentional deficit. However, neurophysiological work suggests that autistic people may indeed have a deficit in their ability to rapidly shift their attention (Courchesne, 1990). There are several possible explanations for this inconsistency. Perhaps, autistics have a compensatory attentional mechanism that allows them to perform near normal on the digit span task. Alternatively, digit span may not require shifting, but rather sustaining, attention, or perhaps digit span does not involve attention to any great degree. However, it is beyond the scope of this paper to determine how to assess the involvement of attention. It is assumed that attention tasks, such as digit span, do assess attention.

Nevertheless, there are some frontal lobe symptoms that are clearly not present in autistics. For example, frontal patients are known to confabulate and have persistent mood changes, including pseudodepression and pseudopsychopathy, autistics may only exhibit extremes of emotion and then only occasionally. Frontal patients also may have problems akin to autotopagnosia, being deficient at behaviors related to egocentric spatial orientation (Mayes, 1988). For example, they cannot point accurately to parts of their bodies as instructed, while having no trouble finding their way around a room via a map. Autistic people are better at spatial tasks than verbal (Lincoln, et al 1988), as evidenced by their performance on the block design and object assembly sections of the WAIS IQ test, whereas frontal patients performed relatively poorly on block design, as well as picture arrangement and digit span (Janowsky, et al, 1989).

Resolution of Discrepancies. However, that there should be significant differences between autistic behavior and that of frontal patients should be expected, even if autism does involve abnormalities in frontal lobe function, After all, the frontal lobe syndrome is usually seen as the result of an extensive cortical lesion in adulthood, whereas autism is thought to be a developmental disorder (Gillberg, 1990). The former results from damage to a mature, normally developed system. The latter may result from the development of abnormal connectivity. Abnormal neuronal connectivity of the frontal lobes, and/or subcortical structures subserving the frontal lobes, may result in symptoms that mimic extensive lesions to frontal cortex in adulthood. In relation to this idea for mediotemporal lobe amnesia, it may also be that some of the differences between autism and organic amnesia are due to the differences that result from a developmental lesion as opposed to one received after maturity. Nevertheless, autism seems to share more cognitive abnormalities in common with frontal lobe syndrome, than with mediotemporal lobe amnesia, though there may in fact be a composite of causes of the autistic disorder, involving both mediotemporal lobe and frontal lobe function. The diversity of lesions classified as frontal, as well as difficulties with diagnosing autism, may then contribute further to discrepancies between frontal lobe syndrome and autism.

Emergence of Higher Order Cognitive Impairment

Assuming that at least some of the symptoms of autism may be attributed to frontal lobe dysfunction, it is relevant to ask whether this could account for any, some, or all of their deficits in higher order cognitive abilities, such as language acquisition, concept formation, categorization, and symbolic representation. Such comparisons are complicated by the fact that in neither autism nor frontal lobe syndrome are the fundamental cognitive deficits characterized, let alone any more complex abilities.

However, one way in which an attentional deficit could result in the complex of cognitive deficits found in these groups may be suggested by a study by Cohen, Ivry, and Keele (1990). They have shown that attention is required for the learning of complex sequences of stimuli. Simpler sequences, in which the previous symbol determines identity of the next symbol, eg. 123123···, may involve the construction of only simple associations between stimuli, but more complex sequences, in which there are multiple, yet constrained, possibilities for the identity of the next symbol, eg. 132312132312···, require the formation of a hierarchical representation. Such less constrained sequences cannot be achieved without attention. Attention may serve to break down the sequence into component parts to which a higher level description may be attached, these higher level descriptions being more determined. This description acts as an additional cue that makes manipulation of the underlying information easier.

These ideas are consistent with the findings of Hermelin (1976) that autistics do not tend to engage in rule extraction to aid them in learning visual sequences. Rather, they learn them by rote. Hermelin's work also suggests that autistics tend to order sequences spatially rather than temporally, the most common strategy in controls. This is consistent with the frontal lobe deficit with temporal order (Shimamura, Janowsky, and Squire, 1991). Additional evidence for a parsing disability comes from work that suggests that autistics may process sensory information centrally in a fundamentally different way (Ornitz 1975, O'Connor, 1975, DeMyer, 1975), resulting in an inability to organize information into modality independent codes. Normally, through the extraction of rules and redundancies, one may arrive at an appropriate integration and interpretation of the components of a perceptual experience. Such codes could be used to reduce information load (Hermelin, 1976), or to facilitate understanding or whatever behavior in which one is engaged. Overall, this research supports the contention that autistics may not be able to use attention to parse an experience in a way necessary to generate such simplifying codes or rules, and is consistent with Prizant's (1983) finding that autistics are impaired at sentence segmentation. Therefore attention plays an essential part in determining the structure of the mind's knowledge. This structure is less a property of the static entity of memory than the result of the dynamic action of attention on the learning experience over space and time. It is with such dynamics that autistics have particular difficulty.

In terms of higher cognitive functions, several studies have shown that autistics have trouble manipulating information, including symbols (Ricks and Wing, 1976). Tager-Flusberg (1985) found that autistic people do not use their acquired cognitive skills in a flexible and appropriate way. While she also found that organization of their semantic knowledge for concrete objects is largely intact, this need not warrant the conclusion that autistics are not deficit in their use of attention at encoding. After all, it takes them an unusually long time to learn these concrete words, and this study did not examine their abilities with abstract words. Prizant's (1983) work on the autistic style of language acquisition suggests that they have a general deficit in using their knowledge and cognitive abilities. While work on concrete versus abstract words are sparse (Hobson & Lee, 1989; Eskes et al, 1990), most researchers consider autistics to be impaired at making abstractions (Ricks & Wing, 1975; Prizant, 1983; Tager-Flusberg, 1985; Hobson & Lee, 1989). Another demonstration of this disability is the tendency of autistic persons to use a holistic rather than an analytic approach (Prizant, 1983). This is true of their language acquisition and of other learning domains. They do not tend to parse their experiences into meaningful, structured components. Rather, autistics form context-bound holistic representations with little meaning, especially for the parts. Inflexible use of any representation formed may indicate that they cannot focus attention on their internal representations efficiently enough to manipulate them, nor can they parse and code representations. Thus, the flexibility of cognition may depend upon attentional capacity.

Conclusions

It has been seen that the autistic syndrome results in a complicated composite of cognitive deficits. It has been argued in this paper that the best way to understand such complexity may be to strip it down to its most fundamental elements. While most researchers have focused on language, this is not likely to be the most basic cognitive ability (Bates, 1990). Thus, it seems reasonable that one should look elsewhere for more basic aspects of cognition. Memory and, particularly, attention are put forth as the most fruitful alternatives. Additionally, comparisons between autistic performance and other

patient populations may serve to generate ideas as to the nature of the deficit in either disorder. In terms of cognitive mechanisms, research into autism has tended to be like the deficit itself. Its approach perhaps tends to be holistic and devoid of the foundational meaning that could be provided by an emphasis on cognitive abilities, like attention and memory. Study of the cornerstones of cognition may ground research on autism, contributing to the elucidation of the primary behavioral characteristics which give rise to the more complex behavioral deficits in language and thought seen in the autistic syndrome.

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