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The Orbitomedial Frontal Syndrome

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An orbitomedial frontal syndrome is proposed, characterized by anosmia, amnesia with confabulation, Go-NoGo deficits, personality change, and hypersensitivity to pain. The orbitomedial frontal syndrome is distinct from the clinical picture that results from dorsolateral frontal damage. Aspects of orbitomedial damage have been discussed previously in isolation, but we argue that recognition of this syndrome in toto is clinically important. It appears to be associated with poor social and vocational adjustment after brain injury, and the co-occurrence of features of the syndrome provides clues to underlying mechanisms for disinhibition and confabulation in frontal lobe patients.

The goal of this paper is to propose a behavioral syndrome resulting from dysfunction of the orbitomedial frontal lobes. Although aspects of orbitomedial (OM) frontal dysfunction have been previously described in isolation, the complete clinical syndrome has not been clearly delineated. The OM syndrome will be illustrated with a case study, depicting the essential features of the disorder. It will be argued that recognition of this syndrome is important both clinically and heuristically, because it is associated with poor postinjury adjustment and can contribute to our understanding of fronto-limbic functional systems.

Many aspects of the functional neuroanatomy of the frontal lobes in humans remain to be delineated, but a number of functional divisions have been identified: The *primary motor* areas (Brodmann's area 4) are critical to pyramidal motor functions; the *premotor* areas (areas 6,43,44,45) are involved in sensorimotor integration and praxis (Heilman, 1979); the *frontal eye fields*

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(area 8) are necessary for voluntary gaze and visual search (Crowne, 1983); and the *supplementary motor* areas and *anterior cingulate gyri* (area 24) comprise a dual system controlling environmental exploration, and initiation of volitional movement (Goldberg, 1987).

The *prefrontal* cortical divisions (anterior to the aforementioned zones) have been of greatest interest to clinicians. Clinical observations have suggested that lesions to the dorsolateral (DL) and OM divisions of the prefrontal lobes result in distinct patterns of cognitive deficits and personality change (Blumer & Benson, 1975). DL lesions seem to cause deficits in temporal and sensory integration, planning, maintenance of goal-directedness, and behavioral flexibility. Lesions of the OM frontal division, on the other hand, result in disruption of inhibitory and emotional mechanisms, with impulsive and socially inappropriate behavior resulting (Luria, 1980; Stuss & Benson, 1983). These functional distinctions are the reflection of distinct evolutionary, architectonic, and anatomical trends that define DL and OM zones (Pandya & Barnes, 1987). The DL system is extensively connected with secondary sensory association areas in parietal, occipital, and temporal lobes, a reciprocal relationship that fits with the presumed role of DL frontal zones in integrating sensory information from multiple modalities. The OM zone is connected with limbic structure in the cingulate and anterior temporal lobes, and hence is well situated to integrate motivational and emotional processes. Mesulam (1986) has referred to the DL zone as *heteromodal* cortex and the OM zone as *paralimbic* cortex, terminology that captures the functional differences well.

Although many previous authors have commented on these functional divisions conceptually, few studies have attempted to differentiate DL from OM frontal lesioned patients clinically. To our knowledge, none have included all of the clinical indicators of OM dysfunction presented here. In fairness, it is difficult to collect groups of patients with frontal lesions strictly limited to the frontal lobes. For example, it required 2 years of reviewing CT scans in a major medical center to identify 20 focal right and left frontal patients for a previous study of frontal functions (Malloy, Webster, & Russell, 1985). More discrete lesions involving solely the OM areas are even more difficult to identify, because naturally occurring lesions frequently involve large areas of the frontal lobes.

Another barrier to understanding OM functions is that most widely used clinical tests of frontal lobe function are most sensitive to executive functions subserved by DL prefrontal zones. For example, the Wisconsin Card Sorting Test (Heaton, 1981), and word or figure fluency tests (Benton, 1968; Jones-Gottman & Milner, 1977) are designed to measure executive functions such as generation of multiple response alternatives, cognitive flexibility, and maintenance of set. Few measures of OM frontal functions in humans exist, and none have been developed into psychometric neuropsychological tests. As a result, clinicians may have difficulty specifying common behavioral sequelae of OM frontal damage.

Despite these difficulties, review of the literature suggests several signs which are reliable indicators of OM frontal dysfunction. These well-described clinical features include anosmia, amnesia with confabulation, deficits on Go-NoGo tasks, disinhibited personality change, and hypersensitivity to pain. Although any one of these signs can occur with lesions of other brain areas, we suggest that when they occur together, they constitute a specific OM frontal syndrome. Recognition of this syndrome by clinicians is important, in that OM frontal dysfunction has dire prognostic implications. Close examination of the syndrome may also help explain neuropsychological phenomena (e.g., confabulation) that remain poorly understood.

CHARACTERISTICS OF ORBITOMEDIAL FRONTAL SYNDROME

Anosmia

Loss of smell discrimination is a common result of OM frontal dysfunction. Orbital cortex receives direct projections from the olfactory tract (Turner, Gupta, & Mishkin, 1978) and secondary olfactory projections from temporal lobe regions (Potter & Nauta, 1979). Poor odor discrimination has been reported in animals following orbitofrontal cortex lesions (Tanabe, Yarita, Iino, Ooshima, & Takagi, 1975), and in humans with neurological disease affecting the frontal lobes. For example, Potter and Butters (1980) reported that five patients with orbitofrontal space-occupying lesions were impaired on an odor discrimination task, whereas patients with posterior damage performed normally. More recently, Jones-Gotman and Zatorre (1988) reported olfactory discrimination deficits in patients following frontal lobectomy, when surgery involved the orbitofrontal cortex. Damage to this region of the frontal lobe appeared sufficient to cause olfactory discrimination deficits, and additional damage to the temporal lobe did not exacerbate this deficit. Finally, deficits in olfactory discrimination have been reported in Korsakoff patients, in whom pathology involving frontal systems has been demonstrated (Jones, Butters, Moskowitz, & Montgomery, 1978).

Shearing of the olfactory nerve against the cribriform plate is a common sequelae of closed head injury (Levin, Benton, & Grossman, 1982), and is usually associated with damage to adjacent OM frontal cortex (Jennett & Teasdale, 1981). Varney (1988) found that anosmia, used as a sign of orbitofrontal damage, had negative implications for vocational prognosis in head trauma. Despite having no clear neurological, intellectual, or memory deficits to explain their unemployment, nearly all of the totally anosmic patients showed obvious vocational problems.

Anosmia, then, is a reliable sign of OM frontal damage, and has prognostic significance. Unfortunately, most neurologists do not routinely test olfaction. This is probably due to the fact that some patients partially lose their sense of

smell due to infection, smoking, and normal aging, necessitating somewhat cumbersome procedures for detecting clinically significant deficits in olfactory abilities (see below).

Amnesia and Confabulation

Damasio, Graff-Radford, Eslinger, Damasio, and Kassell (1985) have described a series of patients with lesions involving the orbital and inferior medial frontal lobe, as well as the basal forebrain (i.e., septal nuclei, nucleus accumbens, diagonal band nuclei, and substantia innominata). These patients experienced profound amnesia that shared characteristics with Korsakoff's syndrome, including tendencies to make impulsive and perseverative errors, difficulty with "temporal tagging" of information (determining when it was learned), and bizarre confabulations. The authors argued that amnesia with confabulation is typical of patients with this sort of frontal lesions, and distinct from the memory disorder seen following temporal lobe damage.

Despite the frequent use of the term "confabulation" in neurology and psychiatry, it has been defined in a variety of ways and has been used to describe apparently different phenomena (Whitlock, 1981). Confabulation most commonly refers to "the production of erroneous and fabricated verbal material and is thought to be a failure of self-critical capacity, rather than a desire to mislead" (Stuss, Alexander, Lieberman, & Levine, 1978). However, there appears to be more than one type of confabulation, and there is some debate over how to parse the various forms.

The earliest division of confabulation has, in fact, gained the widest acceptance. Bonhoeffer (1901; as reported in Berlyne, 1972) described two types of confabulation in amnesia. *Momentary confabulation* refers to the common, usually transient, provoked form of confabulation that occurs when a patient is asked a specific question (Berlyne, 1972). For example, an amnestic patient may attempt to "fill in the memory gaps" by providing incorrect (albeit plausible) information about his recent past. Often the confabulatory responses contain elements of truth but are taken out of their proper temporal or spatial context. This form of confabulation is thought by some researchers to be a normal consequence of impaired memory and indeed, it has been replicated in nonbrain-damaged subjects by asking them to recall information after a very long delay period (Kopelman, 1987). In contrast, *fantastic confabulation* is much less common and tends to be spontaneous, more persistent, and bizarre or grandiose in nature (Stuss et al., 1978). For example, the patient who has never been in the service produces a personal history involving heroic acts and decorations.

A number of mechanisms underlying confabulation have been proposed (Berlyne, 1972), though few have been precisely delineated or rigorously tested. Confabulation has been attributed to suggestibility of the patient or other aspects of personality structure (Williams & Rupp, 1938), psychological

defense against a catastrophic reaction (Zangwill, 1953), memory loss (Barbizet, 1963), disturbance of chronology (Van der Horst, 1932), and disinhibition or inability to self-monitor responses (Stuss et al., 1978). In their review, Stuss and his colleagues (1978) argued convincingly that most of these explanations for confabulation are inadequate and unsupported by clinical data. For example, confabulation in amnestic patients has not been found to be related to heightened suggestibility (Mercer, Wapner, Gardner, & Benson, 1977), and no relationship has been found between premorbid personality and confabulation in a series of Korsakoff patients (Berlyne, 1972). Although impaired memory may contribute to confabulation, it is well known that amnesia per se is not sufficient to produce confabulation and the two are dissociable. In fact, it has been commonly observed that confabulation is present in the early stages of the Wernicke-Korsakoff Syndrome, but the confabulation disappears despite a chronic and very profound amnesia (Victor, Adams, & Collins, 1989). Moreover, results of the Mercer et al. study (1977) failed to support the hypothesis that severity of memory impairment is related to confabulation.

The strongest evidence has been garnered for the view that deficits in self-regulation coupled with memory impairment underlie confabulation (Luria, 1976; Mercer et al., 1977; Stuss et al., 1978). Indeed, confabulation often occurs in patients with frontal lobe impairment who display deficits in self-regulation. For example, in the Stuss et al. (1978) sample, all five patients with confabulation of the "fantastic" type had cortical and/or subcortical frontal lesions and demonstrated cognitive deficits associated with frontal lobe dysfunction. The most prominent neuropsychological deficits of these confabulatory patients included memory impairment, depressed verbal fluency, perseverative responding, stimulus boundedness, poor planning, failure to self-monitor, and flat affect with blatant unconcern. The results of studies of confabulation are consistent with the Stuss case reports: Confabulation has been most closely associated with disinhibition (Mercer et al., 1977) and perseveration of response set (Shapiro, Alexander, Gardner, & Mercer, 1981). Further evidence for the view that confabulation is related to frontal lobe functioning was provided by Kapur and Coughlan (1980) who found that as "fantastic" confabulation resolved, performance on neuropsychological tests of frontal lobe function improved.

Go-NoGo Deficits

In Go-NoGo paradigms the subject is required to make a response to the Go signal, and to withhold or inhibit response to the NoGo signal. The task can be made more difficult by reversing the designated meaning of the signals during testing, or by requiring the subject to overcome the habitual meaning of a stimulus (e.g., Go to a red light, NoGo to a green light).

In animals, it has been found that OM-lesioned animals show the greatest deficits on Go-NoGo, whereas DL-lesioned animals differentially fail delayed response tasks (Rosenkilde, 1979; Fuster, 1989). Four studies have examined Go-NoGo performance in humans on the Go-NoGo task. In a study utilizing topographic evoked potential mapping in normals, Malloy, Rasmussen, Braden, and Haier (1989) found increased activity in OM frontal areas during a Go-NoGo task. Malloy et al. (1985) found that the Go-NoGo task was failed significantly more often by frontal than by non-frontal patients, and that failures were worse in OM-lesioned patients. Drewe (1975) and Leimkuhler and Mesulam (1985) also found that patients with medial frontal lesions were selectively impaired on this task. Thus, in both animals and humans, Go-NoGo failures are strongly related to OM frontal dysfunction.

Disinhibited Personality Change

This aspect of OM frontal dysfunction has received considerable attention from clinical observers (Blumer & Benson, 1975). In the aforementioned series of frontal patients with amnesia (Damasio et al., 1985), prominent personality change was also noted in several patients. This was characterized by sexual and verbal disinhibition, jocularity, lack of concern, and unstable mood.

Other case studies have demonstrated that OM lesions can result in profound and devastating changes in motivational and social behavior, while leaving executive and specific cognitive functions intact. For example, Eslinger and Damasio (1985) described a patient who premorbidly was a well-adjusted, successful accountant. Following bilateral ablation of orbital and lower medial frontal lobes due to a meningioma, he experienced a striking personality change. He made impulsive investments resulting in bankruptcy, lost multiple jobs due to tardiness and absenteeism, stockpiled useless items in his home, and was divorced due to his personality change. An important aspect of this case was his normal performance on extensive batteries of cognitive tests, including traditional psychometric tests of frontal lobe executive function. These investigators did not assess other aspects of the OM syndrome as described here, such as anosmia or Go-NoGo performance.

Hypersensitivity to Noxious Stimulation

While studying the postoperative behavior of prefrontal lobotomy patients whose OM areas had been ablated, Rose (1950) observed an interesting phenomenon. The elementary neurological examination was usually normal in these patients, except for transient reduced muscle tone and bladder control

which typically resolved in a few weeks. However, virtually all the lobotomized patients displayed persistent hypersensitivity to noxious stimulation. This was especially notable when the sole of the foot was stimulated by pin prick. Frontal patients responded with exaggerated withdrawal of the leg, loud complaints, and defensive posturing. Similar hypersensitivity was observed in response to attempts by nurses to give injections, to elicitation of the cremasteric reflex, and even to shaving with a dull razor. Chapman, Solomon, and Rose (1950) confirmed this clinical observation in a quantitative study using a standardized pain threshold apparatus. They documented that frontal lobotomized patients displayed reduced pain thresholds, and exaggerated wince and withdrawal responses to heat. These changes persisted from 1 to 2 years after the operation.

CASE DESCRIPTION

In order to illustrate the OM frontal syndrome, we will describe the case of a young man who suffered bilateral OM frontal lobe trauma, resulting in a constellation of changes in sensory, emotional and personality functions. We argue that this case represents a frequently occurring but unrecognized combination of symptoms (anosmia, amnesia with confabulation, Go-NoGo deficits, personality change, and hypersensitivity to noxious stimulation) typical of an OM frontal syndrome.

History

JC was a 32-year-old, right-handed man with 9 years of formal education, employed as a manual laborer. The patient was educated in a Spanish-language country, but had been living in the United States for many years. He was involved in a motor vehicle accident and sustained a severe head injury, resulting in skull fractures and requiring intubation. He was in a coma for 6 weeks and hospitalized for 2 additional months for therapy. There was no previous history of psychiatric or neurological disease in the patient, and his past medical history was unremarkable. He did not abuse alcohol or other drugs. Family history was also negative for neuropsychiatric disorder.

Three months after the accident he was admitted to Butler Hospital due to his poor impulse control and intermittent explosive behavior directed toward his family. On admission to the hospital, the phenobarbital which this patient had been prescribed prophylactically for seizure control was tapered and replaced with therapeutic doses of carbamazepine. Elementary neurological examination revealed a left upper motor neuron facial nerve palsy, mild bilateral weakness, nonsustained right end gaze nystagmus, and mild ataxia with wide-based gait. There were no "frontal release" signs (snout, suck, glabellar,

grasp, root, or palmomental). CT scan revealed bilateral contusions in the OM frontal region, worse on the left than on the right (see Figure 1). EEG was abnormal with paroxysmal slowing, left greater than right. Routine blood chemistry and urinalysis were normal.

Anosmia

The patient was given the University of Pennsylvania Smell Identification Test (UPSIT; Doty, 1983). The UPSIT consists of four "scratch and smell" booklets with a total of 40 multiple choice items. The test has been extensively normed, and controls for such factors as age-related changes, familiarity, and nonolfactory (trigeminal) identifiability of the stimuli. JC readily selected one of the four multiple choice alternatives and never expressed difficulty with the instructions for the test. Yet he achieved a score of 7 out of a possible 40, essentially a chance performance, placing him in the "Total Anosmia" range of functioning.

Amnesia and Confabulation

The patient had severe verbal and nonverbal memory deficits. For example, on the California Verbal Learning Test (Delis, Kramer, Kaplan, & Ober, 1987) he was only able to recall 3 out of 16 words on the first trial and 5 words after five repetitions of the list. The patient was unable to recall any of the words after a 20-min delay, and categorical cuing did not aid performance.

Although the patient was severely impaired on both verbal and nonverbal memory tests, he did not confabulate in the context of structured memory testing. JC did display confabulation in other realms, however. The patient denied that he had been involved in an accident. He stated that he was in the hospital to "gather information for my boss" concerning an accident, but that the accident did not involve him. Despite severe neurological deficits, JC denied any changes in sensory or motor skills, mental functioning, personality or mood. He displayed this profound denial even when directly confronted with his disabilities.

Confabulation was also observed during testing for anosmia. When specifically asked about his performance on the UPSIT, JC denied being anosmic and said that he could smell as well now as he ever could. In order to further explore this phenomenon, we modified the UPSIT testing procedure. In one condition, JC was asked to make same/different judgments about two target smells. He again responded below chance level and voiced a confabulated smell even when the target was not placed below his nose (the patient was blindfolded). In another condition, the patient was presented with 10 "scratch and sniff" items and asked to identify the smell. He insisted that the initial

FIGURE 1. CT scan of Patient JC, demonstrating low density defects in the orbitomedial frontal lobes bilaterally.

item was "green pepper," then perseverated on this response for the remaining nine items.

Go-NoGo Deficits

The patient was administered a version of Luria's (1980) Go-NoGo tasks. In the first task, the subject is asked to tap once when the examiner taps twice, and to tap twice when the examiner taps once. In the second task, the subject is instructed to tap to the word "Stop" and not tap to the word "Go." Both tasks require the subject to inhibit his automatic or habitual response to the stimulus at hand. JC's performance on the Go-NoGo task was characterized by mirroring of the examiner's movements, indicating an inability to inhibit inappropriate responses. That is, he began by correctly complying with the instructions, but then began to tap the same number of times as the examiner.

Disinhibitory deficits were noted on other tasks as well. For example, when asked to produce words beginning with the letter "A" on the Controlled Oral Word Fluency Test (Benton, 1968), he impulsively produced phonemically unrelated words (e.g. "doctor," "empty," "hand"). Confrontational naming was severely impaired due to responses with no obvious semantic relationship to the target. When provided with phonemic cues, he impulsively produced unrelated words beginning with the target phoneme (e.g., "Raymond" for rhinoceros), a disinhibited response style characteristic of frontal patients.

Personality Change

Upon his return home from his initial hospitalization, the patient's wife noticed marked personality changes. JC had previously been a patient and understanding man, but was now irritable and easily angered by his children. Concerns about his escalating temper outbursts had led to the present hospitalization.

JC's demeanor in the hospital was remarkable for "inertia," or lack of spontaneous activity, alternating with periods of jocularity. He typically sat quietly on the unit unless verbally directed, and then responded slowly, and was described as extremely docile by unit staff. During testing he occasionally became quite talkative and inappropriate, laughing at the test stimuli and rambling in a perseverative manner.

In order to clarify these personality changes, the patient and his wife were administered versions of an experimental Frontal Lobe Personality Scale currently under development by our research group (FLOPS; Grace & Malloy, unpublished manuscript). This questionnaire consists of four scales designed to measure personality features characteristic of frontal patients, namely

"Social and Emotional Disinhibition," "Inertia/Apathy," "Executive Dyscontrol," and "Poor Self-regulation and Monitoring." The subject indicates the frequency of problem behaviors on a 5-point Likert scale ranging from "Never or almost never" to "Always or almost always." JC responded in a characteristically stereotyped fashion, choosing the midpoint "Sometimes" for virtually all items. In contrast, his wife indicated significant problems in all domains. Her ratings ranged from 40 to 51 out of a possible 60 points on the subscales. The discrepancy between the patient's rating and those of his wife illustrate his characteristic lack of awareness or concern about his deficits.

Hypersensitivity to Noxious Stimulation

Light plantar stimulation resulted in a markedly exaggerated withdrawal response. There was no evidence of a general hyperesthesia from observation on the unit or family report, however.

Other "Frontal Lobe" Features

The patient's behavior throughout testing was notable for severe perseveration. On the Wisconsin Card Sorting Test (Heaton, 1981), he produced 104 perseverative responses, with no attempt to correct his erroneous initial match. Attempts to reproduce Luria's alternating graphic sequences were marked by within-task perseverations (i.e. repetition of peaks, rather than alternations of peaks and plateaus as in the model).

Many simple linguistic and spatial functions were intact, although deficits in frontal functions made it difficult to assess these more basic cognitive abilities. JC's spontaneous speech was fluent, but his perseverative tendencies interfered with verbal communication. The patient had some difficulty with comprehension of complex questions and serial commands, and repetition was impaired for lengthy phrases, but this seemed to be due to problems in directed attention and memory rather than to a linguistic deficit per se. The patient was severely impaired on a test of complex visual perception, focusing on isolated parts, unable to integrate the parts to form a whole. His copies of two and three-dimensional objects, although performed very slowly, were adequate. His drawing of a clock showed some lack of planning, but the patient was able to place the hands on it, and set it to a specified time.

DISCUSSION

The case of JC illustrates an OM frontal syndrome, characterized by anosmia, amnesia with confabulation, Go-NoGo deficits, personality change, and

hypersensitivity to noxious stimulation. Recognition of this OM syndrome has both theoretical and clinical import.

The OM Syndrome and Frontal Systems

Since it is rare to sustain discrete damage to only the OM region, it is likely that this syndrome frequently occurs in the presence of more general neuropsychological impairment (especially DL frontal dysfunction). Indeed, in the case of JC, a specific cluster of deficits associated with OM prefrontal damage emerges in the context of other cognitive deficits. Although the two commonly co-occur, we suggest that the features of this syndrome are distinct from the executive deficits that result from DL frontal damage.

In normal behavior, of course, the OM area acts in concert with many other frontal and nonfrontal brain areas in complex systems. This dynamic interaction is necessary for the execution of smooth, integrated behavior (Pandya & Barnes, 1987). For example, on the Controlled Oral Word Fluency Test (Benton, 1968), the subject is required to name as many different words as possible beginning with a given letter, excluding proper nouns or different forms of the same word. The DL frontal zones are probably responsible for generating multiple response alternatives (e.g., "fly, fox") on this task, while the OM areas act to inhibit extraneous associations which do not comply with task requirements (e.g., "Francis," names of other animals). In the intact individual, these interactions are dynamic, and change on a moment-by-moment basis depending on environmental demands.

OM frontal areas have extensive connections with limbic structures involved with motivation, and appear to be crucial in the *modulation* of both affective and motor responses (Fuster, 1989, p.74). In the OM-lesioned patient, deficits may therefore be seen due to direct dysfunction of OM areas, due to disruption of more widely organized brain systems of which OM zones form a part, or due to *disconnection* of other frontal zones from limbic input (Malloy, 1987).

Because the OM system acts as a modulator, dysfunction in the system can result in *over*-responsiveness or *under*-responsiveness. This concept of OM deficits may explain some phenomenon that seem paradoxical. Eslinger and Damasio (1985) noted that OM-lesioned patients are not spontaneously motivated for appropriate actions, although they can often describe correct behavior when prompted. This is consistent with the concept that they suffer from a disconnection of DL executive functions from limbic motivational systems, resulting in under-responsiveness. On the other hand, many of the clinical deficits observed in OM patients appear to be due to over-responsiveness or disinhibition. Deficits on Go-NoGo tasks, for example, typically involve the inability to inhibit responses to salient stimuli in favor of the correct response. Thus, when the patient is asked to withhold a response to a

stimulus with an habitual "Go" meaning (e.g., a green light), the task is made more difficult.

Similarly, Chapman et al. (1950) were puzzled by their finding of increased sensitivity to pain, when lobotomy was known to be an effective treatment for intractable pain. The explanation for this apparent paradox may be that severing the white matter connections to OM frontal lobes can result in both abulia and disinhibition. While the patient may still experience painful sensory input, this fails to have normal emotive significance due to disconnection of the limbic system from cortical zones responsible for conscious awareness. However, because of disinhibition of motor responses, the patient displays exaggerated withdrawal and defensive reactions to minimal stimulation, even though they may not be experienced consciously as more "painful."

The OM Syndrome and Confabulation

Confabulation can similarly be conceptualized as disinhibited speech — the patient speaks the first thing that comes into his mind, without regard to the accuracy or appropriateness of the material. Confabulation may therefore represent one class of disinhibited or impulsive behavior characteristic of the OM frontal patient. The confabulatory patient apparently has difficulty withholding answers and spews out irrelevant associations without monitoring the accuracy of his responses. This disinhibited style, coupled with unawareness of his deficits and impaired memory for information, sets the stage for confabulation in the OM frontal syndrome.

The case of JC exemplifies a number of features that have been highlighted in previous research on confabulation. First, JC had demonstrable bilateral frontal damage. His most prominent neuropsychological deficits were memory impairment and a cluster of "frontal" behaviors, which included disinhibition, perseveration, and unconcern. These deficits have been reported in other cases and appear to be highly characteristic of confabulators (Mercer et al., 1977; Shapiro et al., 1981; Stuss et al., 1978). The patient's confabulation concerning his recent personal history was also typical of previous patients. He clearly had no recollection of his accident, but had probably been told that it involved work (the patient was driving a truck as part of his job, when he was involved in a motor vehicle accident and sustained the head injury). Instead of responding that he didn't know why he was in the hospital, he presumably pieced together recollections and associations to arrive at an explanation: He was in the hospital to collect information for his *employer* about an *accident*. While there were elements of truth in his response, his memory impairment and inability to self-monitor his verbal response resulted in a fictitious tale.

The patient's confabulations about his olfactory experiences are of particular interest. Confabulation limited to a given sensory modality has been observed in Anton's syndrome, in which the patient confabulates visual expe-

rience. The case of JC, like Anton's syndrome, is consistent with Fisher's (1989) proposal that anosognosia is a prerequisite of confabulation. JC is also reminiscent of the case described by Sandson and her colleagues (Sandson, Albert, & Alexander, 1986) in which a fluent aphasic patient displayed two types of confabulation: Confabulation of recent personal history and confabulation of word meaning. The authors argue that this latter form of confabulation reflects gaps in semantic representation. So too, does JC's confabulations reflect gaps in olfactory experience. It is the unique combination of lack of olfactory experience coupled with disinhibition that resulted in "olfactory-specific" confabulations in Patient JC.

Directions for Future Research

As noted above, there has been a dearth of studies reporting groups or series of patients with circumscribed frontal lesions. Future studies should determine whether the signs of OM dysfunction reported here can discriminate groups of OM and DL frontal patients at clinically useful levels. Hypersensitivity to pain, in particular, requires further validation since it is the least well-documented of the signs.

A number of localizational questions remain unanswered. Future studies could address the contention that prominent behavioral changes are seen more frequently in bilateral than in unilateral frontal lesions (e.g., Damasio & Van Hoesen, 1983). The effects of lateralization of damage could also be examined, although this variable has yielded remarkably few reliable differences in studies of DL frontal functions such as card-sorting performance (see Heaton, 1981, for review). It will also be important to determine whether involvement of so-called basal forebrain structures (i.e., nucleus accumbens, diagonal band, substantia innominata) is necessary to produce amnesia in inferior frontal lesions (Damasio et al., 1985), or if orbital cortical lesions are sufficient.

Clinical Implications

Certainly any one of JC's deficits could exist as a result of damage to diverse regions of the brain. However, we suggest that when seen together, these signs represent a distinct neuropsychological syndrome associated with OM frontal dysfunction. It should be clear that we are not claiming to have "discovered" the functions of the OM zone, but rather we have highlighted the disparate clinical signs of the disorder.

Clinicians are well-advised to assess for signs of OM syndrome routinely (especially when patients have a history of head injury or other disorder that often involves OM damage). If OM syndrome is present, it may impact the rehabilitation plan significantly. Family therapy and other environmental

interventions are likely to be much more effective than individual therapy in remediating inhibitory deficits such as sexual aggressiveness or confabulation. On the positive side, patients with amnesia due to OM frontal lesions may be capable of retaining motor learning (Cermak, Lewis, Butters, Goodglass, 1973). Hence, they may be employable in settings such as sheltered workshops that require repetitive assembly skills and provide considerable external structure.

As Mesulam (1986) has pointed out, it is often difficult to document frontal deficits, because the patient may be able to inhibit inappropriate behavior for brief periods, the examination itself may provide needed structure (masking behavioral deficits), and the patient may be able to give adequate responses to social judgment questions but be unable to apply that knowledge in "real world" situations. The clinician who was heretofore hard-pressed to document the relatively subtle (but profoundly disabling) deficits present in OM frontal patients now has a number of tools at his disposal for this difficult task.

REFERENCES

Barbizet, J. (1963). Defect of memorizing of hippocampal-mammillary origin. *Journal of Neurology, Neurosurgery, Psychiatry*, **26**, 127–135.

Benton, A. L. (1968). Differential behavioral effects in frontal lobe disease. *Neuropsychologia*, **6**, 53–60.

Berlyne, N. (1972). Confabulation. *British Journal of Psychiatry*, **120**, 31–39.

Blumer, D. & Benson, D. F. (1975). Personality changes with frontal and temporal lobe lesions. In D. F. Benson & D. Blumer (Eds.), *Psychiatric aspects of neurologic disease*. New York: Grune & Stratton.

Bonhoeffer, K. (1901). *Die akuten Geisteskrankheiten der Gewohnheitstrinker*. Jena: Gustav Fischer.

Cermak, L. S., Lewis, R., Butters, N., & Goodglass, H. (1973). Role of verbal mediation in performance of motor tasks by Korsakoff patients. *Perceptual and Motor Skills*, **37**, 259–262.

Chapman, W. P., Solomon, H. C., & Rose, A. S. (1950). Measurement of motor withdrawal reaction in patients following frontal lobotomy. In M. Greenblatt, R. Arnot, & H. C. Solomon (Eds.), *Studies in lobotomy* (pp. 386–392). New York: Grune & Stratton.

Crowne, D. P. (1983). The frontal eye fields and attention. *Psychological Bulletin*, **93**, 232–260.

Damasio, A. R., Graff-Radford, N. R., Eslinger, P. J., Damasio, H., & Kassell, N. (1985). Amnesia following basal forebrain lesions. *Archives of Neurology*, **42**, 263–271.

Damasio, A. R., & Van Hoesen, G. W. (1983). Emotional disturbances associated with focal lesions of the limbic frontal lobe. In K. M. Heilman & P. Satz (Eds.), *Neuropsychology of human emotion* (pp. 85–110). New York: Guilford.

Delis, D. C., Kramer, J. H., Kaplan, E., & Ober, B. A. (1987). *California Verbal Learning Test: Research edition*. San Antonio, TX: Psychological Corporation.

Doty, R. L. (1983). *The University of Pennsylvania Smell Identification Test Administration Manual*. Philadelphia: Sensonics.

Drewe, E. A. (1975). Go-NoGo learning after frontal lobe lesions in humans. *Cortex*, **11**, 8–16.

Eslinger, P. J., & Damasio, A. R. (1985). Severe disturbance of higher cognition after bilateral frontal lobe ablation: Patient EVR. *Neurology*, **35**, 1731–1741.

Fisher, C. M. (1989). Neurologic fragments. II. Remarks on anosognosia, confabulation, memory, and other topics; and an appendix on self-observation. *Neurology*, **39**, 127–132.

Fuster, J. M. (1989). *The prefrontal cortex: Anatomy, physiology, and neuropsychology of the frontal lobe (2nd ed)*. New York: Raven Press.

Goldberg, G. (1987). From intent to action: Evolution and function of the premotor systems of the frontal lobes. In E. Perecman (Ed.), *The frontal lobes revisited*. New York: IRBN Press.

Greenblatt, M., Arnot, R., & Solomon, H. (1966). Studies of lobotomy. *Proceedings of the Association of Research in Nervous and Mental Disease*, **36**, 19–34.

Heaton, R. K. (1981). *Wisconsin Card Sorting Test Manual*. Odessa, FL: Psychological Assessment Resources.

Heilman, K. M. (1979). Apraxia. In K. M. Heilman & E. Valenstein (Eds.), *Clinical neuropsychology* (pp. 159–185). New York: Oxford University Press.

Jennett, B., & Teasdale, G. (1981). *Management of head injuries*. Philadelphia: F. A. Davis.

Jones, B. P., Butters, N. M., Moskowitz, H. R., & Montgomery, K. (1978). Olfactory and gustatory capacities of alcoholic Korsakoff patients. *Neuropsychologia*, **16**, 323–338.

Jones-Gotman, M., & Milner, B. (1977). Design fluency: The invention of nonsense drawings after focal cerebral lesion. *Neuropsychologia*, **15**, 653–674.

Jones-Gotman, M., & Zatorre, R. (1988). Olfactory identification deficits in patients with focal cerebral excision. *Neuropsychologia*, **26**, 387–400.

Kapur, N., & Coughlan, A. K. (1980). Confabulation and frontal lobe dysfunction. *Journal of Neurology, Neurosurgery, and Psychiatry*, **43**, 461–463.

Kopelman, M. D. (1987). Two types of confabulation. *Journal of Neurology, Neurosurgery, and Psychiatry*, **50**, 1482–1487.

Leimkuhler, M. E., & Mesulam, M.-M. (1985). Reversible Go-NoGo deficits in a case of frontal lobe tumor. *Annals of Neurology*, **18**, 617–619.

Levin, H. S., Benton, A. L., & Grossman, R. G. (1982). *Neurobehavioral consequences of closed head injury*. New York: Oxford University Press.

Luria, A. R. (1976). *The neuropsychology of memory*. New York: John Wiley.

Luria, A. R. (1980). *Higher cortical functions in man*. New York: Basic Books.

Malloy, P. F. (1987). Frontal lobe dysfunction in obsessive-compulsive disorder: Evidence from evoked potential mapping and neuropsychological tests. In E. Perecman (Ed.), *The frontal lobes revisited*. New York: IRBN Press.

Malloy, P. F., Rasmussen, S., Braden, W., & Haier, R. (1989). Topographic evoked potential mapping in obsessive-compulsive disorder: Evidence of frontal lobe dysfunction. *Psychiatry Research*, **28**, 63–71.

Malloy, P. F., Webster, J. S., & Russell, W. (1985). Tests of Luria's frontal lobe syndromes. *International Journal of Clinical Neuropsychology*, **7**, 88–95.

Mercer, B., Wapner, W., Gardner, H., & Benson, D. F. (1977). A study of confabulation. *Archives of Neurology*, **34**, 429–433.

Mesulam, M.-M. (1986). Frontal cortex and behavior. *Annals of Neurology*, **19**, 320–325.

Pandya, D. N., & Barnes, C. L. (1987). Architecture and connections of the frontal lobes. In E. Perecman (Ed.), *The frontal lobes revisited* (pp. 41–72). New York: IRBN Press.

Potter, H., & Butters, N. (1980). An assessment of olfactory deficits in patients with damage to prefrontal cortex. *Neuropsychologia*, **18**, 621–628.

Potter, H., & Nauta, W. J. H. (1979). A note on the problem of olfactory associations of the orbitofrontal cortex in the monkey. *Neuroscience*, **4**, 261–367.

Rose, A. S. (1950). Postoperative behavior. In M. Greenblatt, R. Arnot, & H. C. Solomon (Eds.), *Studies in lobotomy* (pp. 75–82). New York: Grune & Stratton.

Rosenkilde, C. E. (1979). Functional heterogeneity of the prefrontal cortex in the monkey: A review. *Behavioral and Neural Biology*, **25**, 301–345.

Sandson, J., Albert, M. L., & Alexander, M. P. (1986). Confabulation in aphasia. *Cortex*, **22**, 621–626.

Shapiro, B. E., Alexander, M. P., Gardner, H., & Mercer, B. (1981). Mechanisms of confabulation. *Neurology*, **31**, 1070–1076.

Stuss, D. T., Alexander, M. P., Lieberman, A., & Levine, H. (1978). An extraordinary form of confabulation. *Neurology*, **28**, 1166–1172.

Stuss, D. T., & Benson, D. F. (1983). Frontal lobe lesions and behavior. In A. Kertesz (Ed.), *Localization in neuropsychology*. New York: Academic Press.

Tanabe, T., Yarita, H., Iino, M., Ooshima, Y., & Takagi, S. F. (1975). An olfactory projection area in orbitofrontal cortex of the monkey. *Journal of Neurophysiology*, **38**, 1269–1283.

Turner, B. H., Gupta, K. C., & Mishkin, M. (1978). The locus and cytoarchitecture of the projection areas of the olfactory bulb in Macaca Mulatta. *J. Comparative Neurology*, **177**, 381–396.

Van der Horst, L. (1932). Ueber die psychologie des Korsakowsyndroms. *Msschr. Psychiatr. Neurol.*, **83**, 65–84.

Varney, N. (1988). Prognostic significance of anosmia in patients with closed-head trauma. *Journal of Clinical and Experimental Neuropsychology*, **10**, 250–254.

Victor, M., Adams, R. A., & Collins, G. H. (1989). *The Wernicke-Korsakoff syndrome (2nd ed)*. Philadelphia: F.A. Davis.

Whitlock, F. A. (1981). Some observations on the meaning of confabulation. *British Journal of Medical Psychology*, **54**, 213–218.

Williams, H. W., & Rupp, C. (1938). Observations on confabulation. *American Journal of Psychiatry*, **95**, 395–405.

Zangwill, O. L. (1953). Disorientation for age. *Journal of Mental Science*, **99**, 698–701.