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# Utilization Behavior: Clinical Manifestations and Neurological Mechanisms

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This paper describes a variety of motor release phenomena, including manual grasping and groping, imitation behavior, utilization behavior, and alien hand sign, their clinical manifestations, and proposed neural mechanisms. One of these specific neurobehavioral disorders, initially described by Lhermitte (Brain [1983] 106: 237–255), and termed *utilization behavior*, is addressed in more detail. Patients with this disorder are described as reaching out and using objects in the environment in an automatic manner. The current paper provides a comprehensive review of studies that have documented utilization behavior in individuals with a variety of pathologies, all having a specific predilection for the frontal lobes and frontal-striatal systems. Goldberg's (Behavioral and Brain Sciences [1985] 8: 567–616) theoretical framework for understanding motor release phenomena, which conceptualizes these behaviors as resulting from an imbalance between proposed medial (voluntary, goal directed, and future directed) and lateral (automatic, stimulus bound, and visually based) motor systems, is also discussed. Utilization behavior may prove to be a common underlying cause of high levels of excessive and intrusive motor behaviors within various clinical populations. A more comprehensive understanding of the neural systems underlying utilization behavior may prove highly useful for the differential diagnosis of conditions involving the mesial frontal cortex and fronto-striatal connections. Recent studies have started to investigate utilization behavior in clinical populations with known or suspected frontal system dysfunction, including adult patients with a variety of psychiatric conditions and children with ADHD.

**KEY WORDS:** Utilization behavior; frontal lobes; neuropsychological syndromes.

The frontal lobes of the brain, through rich connections to other brain systems, subserve a number of complex functions that are critical for goal-directed behavior. Great interest has arisen in the functions of the frontal areas of the brain, with the recognition that patients with frontal lobe lesions often demonstrate impairments in the selection, production, and organization of goal-directed behavior, sometimes even in the context of intact intellectual ability and spared functioning within other cognitive domains. Indeed, patients with frontal lobe dysfunction often exhibit a perplexing array of spared and impaired cognitive abilities following frontal lobe damage, ranging from striking behavioral alterations in initiation and inhibition, to subtle problems with higher order abilities involved in goal selection, planning, and problem solving (Stuss and Benson, 1986).

Frontal lobe injury can result in both losses/restrictions of motor responding, as well as excesses of responding. Patients with frontal lesions (1) may not exhibit behaviors spontaneously that they are potentially capable of under cued or prompted conditions (e.g., akinesia, abulia, mutism, motor neglect); (2) may have difficulty producing movement sequences or gestures consistently or accurately (e.g., motor impersistence, various forms of apraxia); or (3) may exhibit behaviors that would not normally be exhibited; or may have a combination of these. In this latter category are a number of strikingly abnormal behaviors including manual grasping and groping responses, imitation behavior, utilization behavior, and alien hand phenomena (Lhermitte, 1983). The current paper will provide a brief summary of these behaviors, a

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detailed review of clinical research findings with respect to utilization behavior in particular, and a discussion of proposed mechanisms underlying this domain of abnormal motor movements.

## HISTORICAL PERSPECTIVES AND DEFINITIONS

A number of different motor responses and behaviors have been identified in conjunction with frontal lobe pathology. Motor control difficulties, motor programming problems, and compulsive motor behaviors in frontal lobe patients were described early on by Denny-Brown and Chambers (1958). A set of these involves the production of involuntary movements that are not the result of intended or "willed" action on the part of the patient. The motor activity is generally seen as involuntary and nonpurposeful, and outside of the person's control. As such, it is often seen and described as reflexive in nature. Although widely referred to in the neurological literature, distinctions between the terms and constructs often blur, and the use of terms is not always consistent. In the following sections basic descriptions of several pathological motor responses are discussed.

### Grasp Reflex (GR)

The GR was described by Denny-Brown and Chambers (1958) as the automatic tendency to grip objects or other stimuli, such as the examiner's hand. It is often elicited by passing a tactile stimulus across the palm between the patient's thumb and index finger. If the GR is present, the patient's thumb and fingers grasp in tonic flexion. Attempts to withdraw the stimulating object result in strengthening of the grasp. When tested for by placing the examiner's hand or fingers in the patient's hand, the patient will grip the examiner's hand and not be able to let go, even when requested to do so. The reflex is normal in infants, but is considered abnormal in older children and adults. A similar reflex, involving tonic flexion of the foot may sometimes be seen with stimulation of the plantar surface. Alternative terms that have been used to describe the same phenomena include instinctive grasp reaction, manual grasping behavior, and magnetic apraxia.

### Manual Groping Behavior (MGB)

MGB, sometimes termed the groping reflex, is also evident in infants, but is an abnormal sign in children and adults. The hand (and often the eyes as well) tends to follow an object under examination, again in a somewhat magnetic fashion. Following tactile stimulation, automatic manual manipulation is observed. The patients may, for example, hold, rub, or manipulate objects placed in front of them or on their own person (e.g., buttons, the fabric of collars, etc.). The behaviors do not appear to be volitional or purposeful, and are very repetitive and stereotypic. The involuntary motor activity may cease briefly if the patient's attention is drawn away, but usually resumes in response to the visual or tactile stimulation or both if it is not withdrawn.

### Mechanisms of GR and MGB

Unrestrained motor responses to tactile or visual stimuli in the form of GR and MGB were historically interpreted by Denny-Brown and Chambers (1958) as arising from a sensorimotor imbalance between the frontal and parietal lobes, which they argued represented the internal and external worlds respectively. Frontal lobe damage, and the loss of normal inhibitory functions of the frontal lobes, was believed to release parietal lobe activity elicited by visual, auditory, and tactile sensory information. From an evolutionary and developmental perspective, these abnormal behaviors were seen as resulting from a disinhibition of more primitive exploratory reflexes and motor responses. Both GR and MGB are thus regarded as signs of the release of these reflexes from normal inhibitory control exerted by the frontal lobes, and thus as signs of frontal lobe dysfunction.

GR and MGB tend not to be differentiated meaningfully in clinical research and often are described as manifestations of a single disorder, primarily related to the damage to the mesial frontal area. More recent research, however, has suggested a differential localization of areas on the mesial aspect of the frontal lobes that are associated with these abnormal movement patterns. Hashimoto and Tanaka (1998) examined 28 patients with cerebrovascular accidents. Four patients, with lesions confined to the supplementary motor area (SMA), exhibited GR, primarily, although not exclusively, involving the hand contralateral to the lesion. None of these 4 demonstrated a groping reaction (MGB). In contrast, 3 patients, with lesions primarily involving the anterior cingulate, developed MGB in the hand contralateral to the lesion. These 3 patients demonstrated only very mild GR. Twelve patients, with lesions involving both the SMA and anterior cingulate, demonstrated both GR and MGB. Although the majority of the abnormal reflexes were seen in the hand contralateral to the lesion, in some cases, they were seen in the hand ipsilateral to the lesion. This may be due to the fact that the SMA exerts some degree of bilateral control over both

upper limbs. Patients with lesions involving the medial region of the parietal lobe, the corpus callosum, or the lateral convexity of either the frontal or the parietal lobe demonstrated neither GR nor MGB. Based on these findings, the authors concluded that the SMA is involved in inhibiting abnormal or primitive grasping reflexes, and the anterior cingulate is involved in inhibiting manual exploratory or groping responses.

### Imitation Behavior (IB)

IB, first coined by Lhermitte (1983), refers to a tendency to imitate the examiner's gestures or movements. For example, the patient might cover his/her mouth, wave, or clap hands in response to observing the examiner make these same movements. This tendency to imitate or copy movements persists even after the patients are explicitly told not to imitate or copy, and are provided with negative feedback after they have copied movements. Patients imitate both meaningless and meaningful movements or gestures, but appear to do so without volitional intent or purpose, and appear to be unable to inhibit the response. Imitation is well recognized as a powerful and important behavioral response, particularly during infancy and early development, but is often not recognized or is considered abnormal in adults. Interestingly, however, research in human communication has shown that mimicry, the mirroring of speech, movement, and gesture, is a common and even essential aspect of social interaction (Allport, 1968; Bavelas *et al.*, 1988). This may grow out of more primitive IB, but be more complex and subtle than pathological forms of abnormal IB seen in some neurologically impaired adults. Like GR and MGB, IB is also seen as resulting from a loss of inhibitory control over a more primitive reflex, albeit involving a more complex set of sensory and motor functions. More specific information regarding the localization of lesions associated with IB is provided in the section describing case studies of utilization behavior.

### Utilization Behavior (UB)

UB is another abnormal behavior pattern that has been documented in individuals with dysfunction of frontal areas or systems. Also coined by Lhermitte (1983), the term "utilization behavior" refers to the automatic elicitation of instrumentally correct, yet highly exaggerated and or inappropriate motor responses to environmental cues and objects (Eslinger *et al.*, 1991; Hashimoto *et al.*, 1995; Lhermitte *et al.*, 1986). Patients with UB are described as reaching out and automatically using objects in the environment in an "object-appropriate" manner that is inappropriate for the particular context. For example, a patient may pick up a toothbrush and begin to brush his teeth, in response to a toothbrush being placed in front of him, but in a context or setting in which brushing teeth would not normally be expected or done, such as in an appointment with a doctor. In a neuropsychological testing session, a patient with UB might automatically pick up a pen and paper on a table and begin writing something without being told or asked to do so.

### Environmental Dependency Syndrome

UB has sometimes been described in the context of a more global environmental dependency syndrome (EDS; Lhermitte, 1986). EDS is a term used to describe deficits in personal control of action and a striking overreliance on social and physical environmental stimuli for guiding one's behavior in a more elaborate social context. For example, one patient, upon being told that the examiner's office was an art gallery, began staring and commenting on pictures as if they were on display. He also looked through and selected pictures that had been placed on the floor, which he then hung on nails he pounded in the walls (Lhermitte, 1986). In a less contrived situation, a patient began to undress and get into a bed upon entering a room in which there happened to be a bed in plain sight. EDS is seen as a more florid, contextually based example of UB involving more complex sequences of motor behavior.

### Alien Hand Sign (AHS)

The AHS refers to a somewhat diverse set of abnormal motor behaviors and associated sensations (Brust, 1996; Doody and Jankovic, 1992; Kaufer *et al.*, 1996; Riley *et al.*, 1990). Patients with AHS react with surprise, concern, or perplexity or a combination of these following the production of movements over which they feel no ownership or control. AHS is a component of at least three different constellations or syndromes, each of which has a different neuroanatomical association. The callosal syndrome involves the nondominant hand and is characterized by "intermanual conflict" or "manual interference." In attempting to do a task, the hands work poorly together, with one hand, usually the nondominant one, often interrupting or undoing what the other is doing (Sperry, 1968). The assumption in this form of callosal AHS is that each hemisphere is exerting control of the motor output for different goals. In the mesial frontal form of AHS, there is a release of involuntary exploratory behaviors in the contralateral (and sometimes ipsilateral) limb, including GR, MBD, and UB. This frontal form has been postulated

as a dissociation between will and action. Although the patient feels no direct attachment to his actions, there is no specific sensory distortion or anosognosia. The third category of behavior subsumed under the term AHS is a posterior parietal–occipital form in which the involuntary movements of the hand may involve withdrawal or avoidance responses such as a levitation of the limb away from support surfaces. Patients are often surprised by the new position of their limb, and seem unaware they have made any movement at all. In this posterior form of AHS, sensory impairments are prominent, and feelings of alien control are often seen together with visual and somatosensory dysfunction as well as body schema distortion.

Although many of the abnormal motor responses discussed in these sections can be thought of as unintended by the patient, in AHS the patient makes explicit statements with respect to feeling a lack of agency or control. Patients with AHS often report feeling as if some other "alien" or outside force is involved in producing the movements, and may personify the unregulated limb (e.g., naming it or speaking about it as belonging to another). Although many movement disorders result in loss of motor control, what is specific to AHS is the sense that some other force or 'alien' agent is producing the movement. It is difficult to determine from the literature just how AHS maps onto other disorders, such as GR, MGB, IB, and UB. In many studies there is little or no comment with regard to the patients' associated sensations or feeling with respect to the abnormal movements, and patients with significant cognitive impairment may be unable to perceive or express such feelings. Nevertheless, AHS deserves mention in any discussion of automatic motor behaviors.

## A BRIEF REVIEW OF PROPOSED MECHANISMS FOR IB AND UB

It has long been recognized that frontal lobe lesions can disrupt the ability to inhibit impulsive actions, and to maintain purposive and goal-directed behavior (Luria, 1967). Indeed, patients with frontal lobe damage often appear to be easily distracted and strongly influenced by irrelevant external stimuli, such that they have difficulty behaving in accordance with the plans or intentions that they have created (Luria, 1967). Along the same lines as those adopted to explain GR and MGB, most theoretical perspectives explain IB and UB as resulting from a loss of frontal inhibitory control over more automatic behaviors. Again, for IB and UB, it has been postulated that there is an imbalance between frontal (internally generated) and parietal (externally or environmentally driven) systems (Brazzelli *et al.*, 1994; Lhermitte *et al.*, 1986; Shallice *et al.*, 1989). This distinction between neural systems for internally generated movement and externally elicited/guided movement was made particularly explicit by Goldberg (1985). Abnormal behaviors may be triggered by externally oriented systems responsive to environmental input, if there is a loss of frontally based inhibitory controls that mediate more purposeful, volitional, and internally generated behavior. We will return to this model and discuss the support for it after a more detailed discussion of UB.

Briefly however, we want to comment on a topic of considerable interest with respect to imitation behavior. This is the recent identification of 'mirror neurons' in the premotor area of the macaque monkey (Gallese *et al.*, 1996). So-called mirror cells become active both when the monkeys perform a given action and when they observe similar action performed by the experimenter. The authors proposed that mirror neurons form a system for matching observation and execution of motor actions in the lateral premotor area. A loss of inhibitory control over this system would result in abnormal imitation behavior.

## CLINICAL STUDIES/REPORTS OF UTILIZATION BEHAVIOR

To date, most investigations of UB have been detailed case descriptions. Although the early investigations provided only very general information about lesion localization, recent studies utilizing more sophisticated scanning procedures have provided more detailed neuroanatomical correlates of the disorders. However, the terminology used to designate particular behaviors is still often inconsistent, specific motor responses are not well described, the context in which the responses are observed or elicited is often not well specified, and the consistency/repeatability/recovery of the abnormal behavior is not routinely reported. In most cases, detailed neuropsychological correlates of the motor disorders are not tested or reported. Nevertheless, the literature does provide a rich source of information about UB and its behavioral, neurological, and neuroanatomical correlates. The studies described hereafter are summarized in Table I.

As noted previously, Lhermitte (1983) first clearly described UB based on his observations of 6 patients with unilateral or bilateral lesions of the frontal lobes. With these cases, he extended the notion of what he termed "magnetic apraxia" (including what we have described here as GR and MGB) to include those instances where tactile, visuo-tactile, and visual presentation of objects compelled the patient to grasp and use the objects according to an appropriate motor program. UB was stimulated by placing a common object in the palm and fingers of a

**Table I.** Brain Lesions Associated with GR, MGR, IB, and UB

| Abnormal motor behavior | Area of lesion[a] | Reference |
|---|---|---|
| Manual grasp reflex | Bilateral frontal damage secondary to herpes encephalitis | Brazzelli *et al.* (1994) |
| | Supplementary motor area (SMA) ($n = 4$) | Hashimoto and Tanaka, 1998 |
| Manual groping response | Anterior cingulate ($n = 3$) | Hashimoto and Tanaka, 1998 |
| Manual grasp and groping | SMA and anterior cingulate ($n = 12$) | Hashimoto and Tanaka, 1998 |
| Imitation behavior | Frontal lesions ($n = 40$) | Lhermitte *et al.* (1986) |
| | Bilateral cingulate | Fukui *et al.* (1993) |
| | Upper medial and lateral frontal cortex (±lenticular nucleus, caudate nucleus, internal capsule or all of these) ($n = 17$) | DeRenzi *et al.* (1996) |
| | Lenticular nucleus alone | |
| | Thalamus alone | |
| | Internal capsule | |
| Utilization behavior | Orbitofrontal and caudate lesions ($n = 6$) | Lhermitte, 1983 |
| | Mesial frontal lesions ($n = 35$) | Lhermitte *et al.* (1986) |
| | Bilateral inferior medial frontal lesion | Shallice *et al.* (1989) |
| | Bilateral hypodensities: ACA distribution | Hoffman and Bill, 1992 |
| | Bilateral frontal damage secondary to herpes encephalitis | Brazelli *et al.* (1994) |
| | Bilateral masses in cingulate gyri | Fukui *et al.* (1993) |
| | Cingulate gyrus (ACA infarct); Medial frontal hematoma ($n = 2$) | DeRenzi *et al.* (1996) |
| | Left anterior cingulate & bilateral caudate | Degos *et al.* (1993) |
| | Bilateral medial thalamic infarct | Eslinger *et al.* (1991) |
| | Right anterior thalamic infarct | Hashimoto *et al.* (1995) |
| | Fronto-striatal projections (PSP) ($n = 7$) | Ghika *et al.* (1995) |

[a] $N = 1$ unless otherwise noted.

patient's hand or by holding out an object (e.g., a glass, a jug of water, a plate, a knife and fork, etc.) and enticing the patient to seize it. Patients with frontal lobe lesions automatically grasped and then proceeded to "utilize" these objects in an appropriate manner, even when object use was inappropriate for that particular situation. Patients were also described as occasionally manually grasping and manipulating objects in a nonpurposeful manner as well. As indicated earlier, UB was postulated to reflect an imbalance between frontal-voluntary (i.e., intrapersonal, motivational, reward driven) and parietal-reactive (i.e., environment-driven, extrapersonal, reflex driven) systems. Based on an analysis of lesion sites in these six cases, Lhermitte concluded that UB arises following unilateral or bilateral frontal lesions. More specifically he claimed that UB had an inferior frontal localization, reflecting damage to orbital frontal structures and possibly the caudate nuclei, although his information about localization was quite limited.

In a subsequent investigation, Lhermitte *et al.* (1986) studied 125 patients with focal and diffuse cerebral lesions resulting from a variety of neurological processes (tumor, trauma, vascular lesions, degenerative disorders, etc.). Patients were screened for evidence of environmental dependency, including both IB and UB, through neurological/neuropsychological testing and behavioral observations. Within this sample, 40 patients were identified as displaying IB, 35 patients as demonstrating IB and UB, and 50 patients as neither exhibiting IB nor UB. No patient exhibited UB without IB. Results of clinical examinations, neuropsychological testing, and behavioral observations revealed that all patients exhibiting IB alone or IB and UB had frontal lesions and displayed these behaviors within the context of a more global "frontal syndrome." IB and UB were again believed to reflect an imbalance between dependence on and independence from external stimuli, and to differ only in terms of their level of severity (with UB reflecting a more severe disorder), and their respective dependence on the social (in the case of IB) versus the physical (in the case of UB) environment. Interestingly, individuals with disseminated lesions incorporating both frontal and parietal systems did not demonstrate UB or IB, suggesting these behaviors are associated specifically with damage to the frontal cortex in the context of intact parietal function. The second part of this investigation supported the ecological validity of the IB and UB constructs through their elicitation in real-life situations (Lhermitte, 1986).

Shallice *et al.* (1989) described UB in an individual with bilateral inferior/mesial frontal lobe lesions as a result of an ischemic episode involving both anterior cerebral arteries. This study also identified concerns regarding Lhermitte's original procedures for eliciting UB, particularly that the examiner's placing objects in the clients'

hands may have led patients to a mistaken understanding of what was expected of them. They were concerned that this procedure for inducing UB may have confused Lhermitte's patients and led them to the erroneous assumption that the examiner wished them to use the objects. To address this issue, this study employed two different procedures for eliciting UB; Lhermitte's original procedure (which they termed "induced UB") and an additional 'incidental' procedure in which there was presumed to be no explicit or implicit expectation that objects should be used. During this incidental procedure, the patient was engaged in neuropsychological tests and other tasks in the presence of objects that could act as triggers for UB. Behavioral observations of the patient revealed 23 occurrences of incidental UB during the testing. These were subsequently categorized as (1) toying: a single action in which an object was manipulated but not in a purposeful way, (2) complex toying: actions involving two objects used together but not for the purpose for which they were designed or in a complete way, and (3) coherent activity: a set of actions integrated in an appropriate way with respect to one or two objects. UB was also elicited with the patient using an induced procedure, but no direct comparison of these two conditions was reported. De Renzi *et al.* (1996) investigated IB and UB in 78 neurologically impaired patients with focal CT documented hemispheric lesions. Patients were separated into a "frontal group" ($n = 52$), in which damage encroached on the frontal lobes, and a "nonfrontal" group ($n = 26$), in which the frontal lobes were spared. UB was invoked through both incidental and induced procedures, and IB was elicited through the performance of various gestures by the examiner. IB was present in 39% of the frontal patients; it was generally associated with damage to upper medial frontal cortex, and in some cases with damage to deep frontal striate structures including the lenticular nucleus, the head of caudate, the internal capsule or all of these. Three patients without evidence of frontal cortical lesions also showed IB; all demonstrated lesions confined to deep frontal structures including the lenticular nuclei, the thalamus, the internal capsule or all of these. GR was present in approximately one half of the frontal cases, but the degree of overlap with IB was not high; 65% of cases with IB showed GR whereas 42% of patients without IB showed GR. Within this sample UB was again, as in the Lhermitte *et al.* (1986) study, seen much less frequently than IB, occurring in only 2 patients, both of whom also exhibited IB. One of these patients had a small anterior cerebral artery infarct encroaching on the cingluate gyrus, and the other a hematoma involving the mesial and lateral aspects of the frontal lobe. The authors concluded that UB is always present in association with IB, and that UB reflects a more severe degree of impairment.

UB and concomitant motor neglect were reported in association with bilateral edematous masses of the left and right cingulate gyri in a patient with primary cerebral malignant lymphoma (Fukui *et al.*, 1993). The patient presented with bilateral UB and IB, and motor neglect of the left arm. Pathological grasping on visual or tactile presentation of objects, nonuse of the left arm, and the automatic use of objects, despite prohibitions not to touch them, were reported. In most situations, the patient's reactions consisted of automatic reaching, holding, and using objects for their intended purpose, particularly with the right hand. Following treatment, MGB was still present in the right hand and became more prominent in the left hand, as use of the left hand increased in general. Automatic utilization of objects became less conspicuous in the right hand, but more prevalent in the left hand. Following recurrence of the tumors, the bilateral GR, MGB, and UB again became more prominent in the right hand. The nonuse of this patient's left extremity was believed to be associated with motor neglect. It is interesting to note that UB and IB can be seen in association with motor neglect in the same limb, suggesting that externally driven motor responses can occur in the context of severely impaired, internally generated motor responses.

UB and IB were also prominent signs in a patient with bilateral frontal lobe infarction due to moyamoya disease (Hoffmann and Bill, 1992). Moyamoya disease, also called occlusive vascular disease, is characterized by progressive neurological disability as the result of occlusions and small hemorrhages in the vessels at the base of the brain. The disease has an initial predilection for the anterior circulation. In this patient, CT scans revealed bilateral hypodensities in the distribution of the anterior cerebral artery. Although performance during standard neurological and neuropsychological examination was within normal limits, behavioral sequelae included inappropriate behavior, an apathetic and aloof demeanor, and both IB and UB. EDS was seen when the client was placed in a number of familiar situations. All three categories of abnormal motor responding resolved gradually over a period of 6 months following treatment, coinciding with 60–70% shrinkage of the anterior lobe hypodensities. The authors described EDS as an extension and a more severe form of IB and UB that is more likely to be elicited in the more familiar and complex situations of everyday life. This study suggests that IB and UB may be some of the earlier and subtler signs of bilateral prefrontal lobe dysfunction.

Brazzelli *et al.* (1994) described a 16-year-old girl with herpes encephalitis and bilateral damage of the frontal

lobes. Observations during neuropsychological testing revealed significant motor hyperactivity and high levels of MGB and UB. Interestingly, her performance on several neuropsychological measures of attention and executive function was within normal limits, refuting the notion that UB is only present in the context of a severe "frontal syndrome." In this case, there was a bilateral lesion involving the orbito-mesial cortex, with sparing of dorsolateral prefrontal cortex and the basal ganglia.

A severe frontal syndrome, including UB, was described in a patient found, on postmortem examination, to have an infarct of the left anterior cingulate gyrus-caudate complex and the head of the right caudate nucleus (Degos *et al.*, 1993). Prior to the patient's death from another cause, clinical features included distractibility, docility, emotional unconcern, perseveration, anterograde amnesia, MGB, and UB. When left alone the patient tended to remain silent, motionless, and seemingly indifferent to the situation. However, she demonstrated GR and UB when presented with an object, even when instructed explicitly not to touch it. Bilateral disruption of fronto-striatal (specifically caudate) projections was suggested in the genesis of her UB.

A study of environment driven responses in patients with progressive supranuclear palsy (PSP) provides additional evidence for the role of fronto-striatal mechanisms in UB. PSP is a Parkinsonian syndrome, with supranuclear opthalmoplegia as a prominent feature. The neuropathology of PSP is thought to involve dysfunction in the frontal lobes and basal ganglia (fronto-striatal system). The behavioral sequelae of PSP include classical motor release signs, including complex motor behaviors involving compulsive manipulation of tools and environmental dependency. Results of an investigation by Ghika *et al.* (1995) in 7 patients with PSP indicated numerous instances of GR and UB when objects were placed in front of them. The behaviors persisted even when they were told to refrain from touching the objects. A parallel compulsive attraction of the gaze by objects that might normally elicit 'watching' (e.g., a television screen, a mirror, and a camera) was interpreted by the investigators as a form of "UB of the gaze." In conjunction with evidence from PET scans, UB in PSP was postulated to have resulted from weakened descending inputs from the frontal lobes, which are typically responsible for set, choice, timeliness, and initiation of motor responses. Frontal-striatal dysfunction was hypothesized as leading to disinhibition of a "chain of behaviors" involving automatic orienting, sensing, and tracking.

The potential role of other frontally connected subcortical structures, particularly the thalamus, in the manifestation of UB has also become evident. For example, disinhibited responding to objects and environmental cues was described by Eslinger *et al.* (1991) in a woman with bilateral encephalomalacia affecting the medial thalamus. Behavioral observations indicated high levels of distractibility and disinhibition, and excessive manipulation of objects in the external environment. On examination, with and without induction, the patient excessively utilized objects and demonstrated great difficulty regulating her interaction with the environment. Neuropsychological evaluation revealed executive impairments in concentration and mental control, and significant difficulties with focusing, maintaining, and shifting attention. Significant deficits were also noted in motor programming. The fact that UB can occur with focal damage to the paramedian thalamic region is not surprising given the connection between this area and the cingulate cortex. PET studies have demonstrated hypometabolism in frontal regions after thalamic infarction, supporting a possible diaschisis effect on the medial frontal lobes following damage to anterior and medial thalamic structures (Cummings, 1995).

Consistent with this notion, UB was reported in a patient with right anterior thalamic infarction (Hashimoto *et al.*, 1995). Functional neuroimaging revealed hypoperfusion in the right frontal cortex, likely as a result of interrupted projections from the thalamus to orbital medial prefrontal cortex. Behavioral observations of the patient indicated marked motor impersistence (an inability to sustain a simple motor response like keeping eyes closed or tapping a finger), a bilateral, but right hand predominant, instinctive grasp reaction (GR), and excessive utilization of objects (UB). Either a light stationary contact with any part of the patient's hand or visual stimuli evoked a series of palpating or groping movements. She excessively utilized objects placed in front of her without any instruction to do so. When explicitly instructed not to use the objects, the patient would immediately discontinue the behavior, but resume utilizing the objects after a few minutes during which her attention was diverted. Of note, marked fluctuations in this behavior were noted from session to session. A 2-month follow-up revealed that the motor impersistence and UB had resolved, although mild GR remained. GR and MGB were interpreted as due to disinhibition of instinctive orientating behavior or primitive vigilance systems following right thalamo-frontal dysfunction. It is interesting to note that this patient demonstrated predominant right GR given her right-sided thalamo-frontal lesion. Interestingly, Mori and Yamadori (1986) previously demonstrated a close relationship between motor impersistence and *ipsilateral* instinctive grasp reflex, particularly following right hemisphere damage, as was the case in this patient, although the mechanisms for this association are not clear.

## PROPOSED NEUROLOGICAL MECHANISMS OF UTILIZATION BEHAVIOR

As noted in the introduction, Denny-Brown and his colleagues (Denny-Brown and Chambers, 1958) were among the first to describe and discuss the origins of many abnormal motor reflexes and behaviors. They hypothesized that external tactile and visual stimuli could trigger automatic visual tracking, reaching, grasping, manipulation, and complex utilization responses in individuals with frontal lobe lesions. In discussing these phenomena, they introduced the notion of competitive *tropisms* (biological orientations), a positive one involving approach/exploratory behavior that is dependent primarily on posterior brain systems, and a negative one involving withdrawal/avoidance behavior that is dependent primarily on anterior brain systems (see Vilensky and Gilman, 1997, for a recent review of Denny-Brown's hypothesis). These two systems were hypothesized to act in opposing, but complementary fashions in the coordination of movement at all levels of the neuroaxis.

According to the model, exploratory movements like visual tracking, grasping, and groping are triggered by sensory input processed in cortical and subcortical sensory areas. However, if the response is unnecessary, maladaptive, or inappropriate to the larger external context, or to the organism's internally generated intentions and motor goals, such responses are inhibited before they are produced. This inhibition involves frontal, and particularly mesial frontal structures and their subcortical striatal and thalamic connections. Withdrawal/avoidance/neglect behaviors, though not as clearly specified in the model, are thought to result from lesions involving the posterior temporal and parietal lobes and involve failures to orient or respond to sensory inputs or abnormal movements away from sensory input. More recent theory has elaborated on this concept. A cascade of behaviors involving attention and orientation to external stimuli, approach, exploration, and utilization is believed to be controlled by the posterior parietal cortex and lateral frontal cortex (Ghika *et al.*, 1995). This sequence is inhibited when necessary or appropriate by processes in the mesial frontal cortex.

In understanding these unusual motor behaviors, it is important to recall that the cerebral cortex regulates the activity of spinal neurons, and therefore produces and influences movement, in both direct and indirect ways. Cortico-spinal fibers originate from many parts of the cerebral cortex and send direct projections to the spinal cord. Such fibers extend not only from the primary motor cortex, but also from the premotor cortex, including the supplementary motor cortex (SMA), and from portions of the parietal somatosensory cortex. In addition to these direct spinal connections, other areas are involved in movement planning and control. At the highest level of control are prefrontal and polysensory association areas. Processing at this level is critical for planning actions based on present perceptual information, past experience, and future goals. The premotor and motor cortex, with input from the basal ganglia, thalamus, and cerebellum, translate these action goals into movement.

This kind of distributed, hierarchical organization may be more easily understood from a phylogenetic perspective. Movement in organisms with primitive motor structures is primarily based on simple reflex actions operating at even the spinal level. Additional layers of subcortical and cortical control enable these reflexes to be modified, such that they are only expressed under certain conditions. This additional regulation of lower levels of the motor hierarchy offers an organism greater flexibility of action, and the ability to generate any number of movements in response to sensory signals. Higher subcortical and cortical mechanisms also enable the organism to generate actions that are not dependent on external cues, but on internal intentions (e.g., gesturing, speaking or producing other skilled movements). The cortico-spinal tract, which appears only in mammals, is a late evolutionary adaptation that affords enormous motor flexibility. During simple, overlearned, or repetitive movements, only primary motor and sensory areas are activated (Roland, 1993). During movements with greater complexity, the contralateral premotor, supplementary motor, and prefrontal cortices, the basal ganglia and the ipsilateral cerebellum are activated. There is also activation of the ipsilateral supplementary motor area and prefrontal cortex during both complex movement and imagined complex movements (Gazzaniga *et al.*, 1998; Roland, 1993).

Goldberg (1985) proposed that the control of movement varies as a function of whether the action is internally or externally guided. According to his very detailed model, the mesial portion of the frontal lobes, including the SMA, will dominate when the task is internally guided, as in a self-regulated finger-sequencing task or in producing gestures without visual guidance or cues. In contrast, the lateral portion of the premotor area becomes more involved when the behavior depends on or is primarily made in response to external stimuli or cues. This area is activated when people are asked to perform movements under the guidance of visual, auditory, or somatosensory feedback. The cingulate gyrus, which receives input from the striatum and basal ganglia, projects to the SMA, which projects to the motor cortex, creating the potential to both facilitate and inhibit motor responses. With damage to the mesial system, including the SMA or cingulate gyrus or

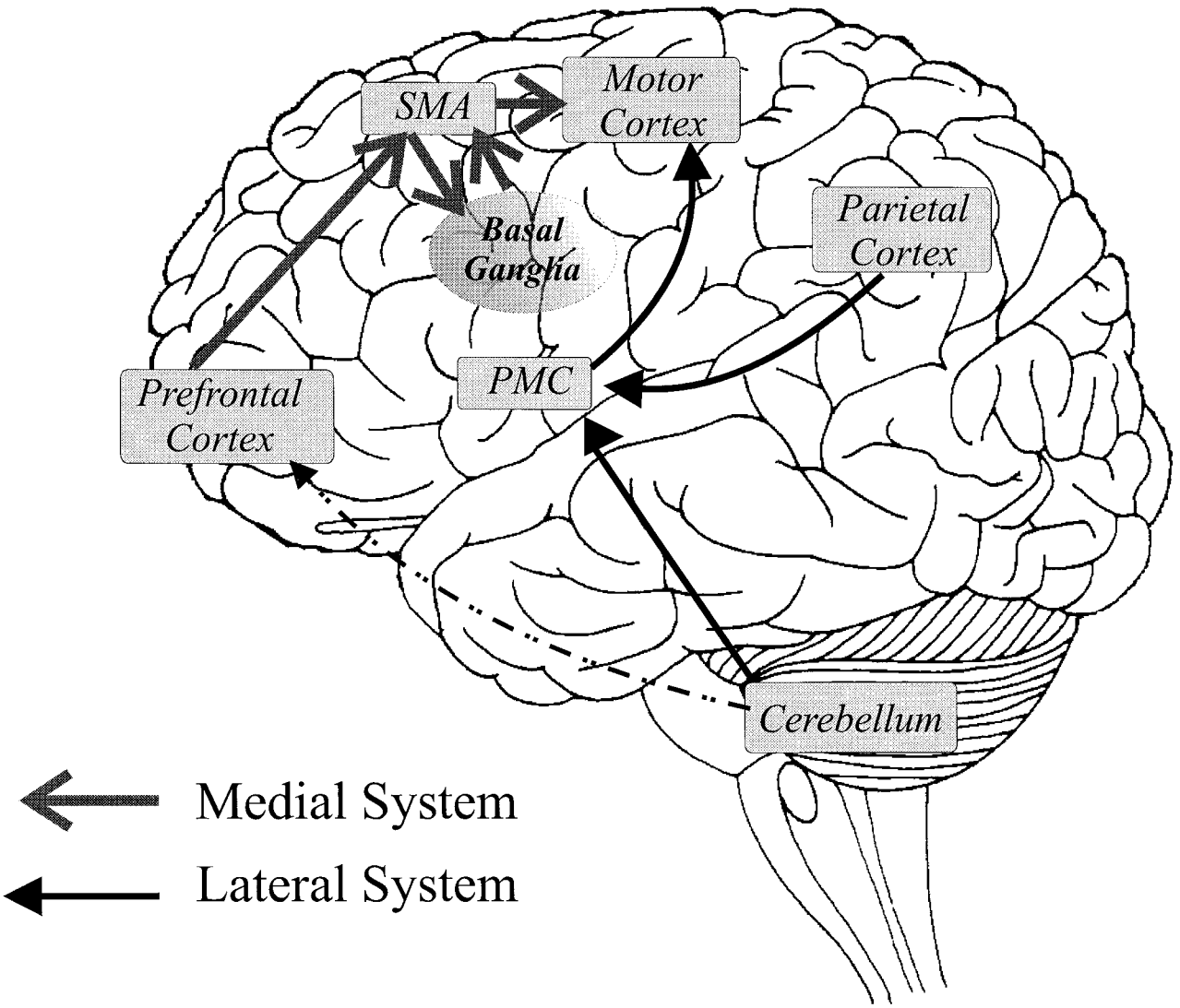


**Fig. 1.** Schemata of Golberg's model of motor control (1983). The lateral (or external) system includes inputs from the cerebellum and parietal cortex to the premotor cortex (PMC), which then influences the motor cortex. This system is hypothesized to dominate during visually guided movements. The medial (or internal) system includes inputs to the supplementary motor cortex (SMA) from the basal ganglia, anterior and medial nuclei of the thalamus, the anterior cingulate, with reciprocal connections to and from the motor cortex. Goldberg hypothesized that this system dominates during self-guided and learned movements. The prefrontal input becomes particularly important during novel task acquisition.

both, there may be both a reduction of normal internally generated, willed movement, manifest as akinesia, motor neglect, motor impersistence or all of these, and a release of unintended reflexive movements in the form of GR, MGB, IB, and UB.

Figure 1 provides an overview of the anatomical basis for Goldberg's external–internal control hypothesis. According to Goldberg, the lateral premotor cortex is extensively innervated by the parietal cortex, ventral lateral thalamus, and the cerebellum, all areas linked to rich sources of sensory information. It is this interconnected system that he claims is responsible for sensory-guided movement driven by primarily external stimuli. Indeed, substantial research has demonstrated exceedingly close interactions between perception and action. In contrast, the SMA receives extensive projections from the prefrontal cortex, the anterior cingulate, the basal ganglia, and the ventral thalamus. These connections allow access to information regarding internalized behavioral goals and plans, and to limbic structures that convey information about the organism's current motivational state. The SMA is seen to play a role in intentional processes whereby the internal context

and internally generated goals influence the elaboration of action. In support of this hypothesis, Orgogozo and Larsen (1979) studied blood flow patterns in relation to performance of various actions. Their results suggested that the SMA participates in the organization of movements in direct proportion to the degree of internally developed "intentionality" associated with the performance of the task, that is, the degree of conscious intentional engagement of the subject in the task.

Goldberg's model suggests that widespread activation of the cortical mantle provides a contextual basis for action. This activity converges on the striatum, passes through the ventral thalamus, and on to the SMA. This transformation from context to intention to act is modulated by the limbic system both within the basal ganglia and via projections to the SMA from the cingulate cortex, which itself receives projections from the anterior and medial thalamus. The SMA, which participates earlier than the primary motor cortex in the translation of motive to intention to act, projects to both the primary motor cortex and corticospinal fibers. The goal is to select a context-appropriate behavioral schema in accordance with internal goals and states. At the same time, sensory information from parietal cortex, lateral thalamus, and direct cerebellar inputs converge on the lateral premotor cortex, triggering motor responses that serve to monitor and explore environmental events. A more detailed diagram illustrating these connections is provided in Fig. 2.

Within this model, GR, MGB, IB, UB, and EDS all reflect some disruption in the inhibitory control of the medial system. GR appears to occur more commonly following SMA lesions, and MGB more often following cingulate lesions. Both IB and UB appear most commonly to involve the cingulate, caudate, and anterior and medial

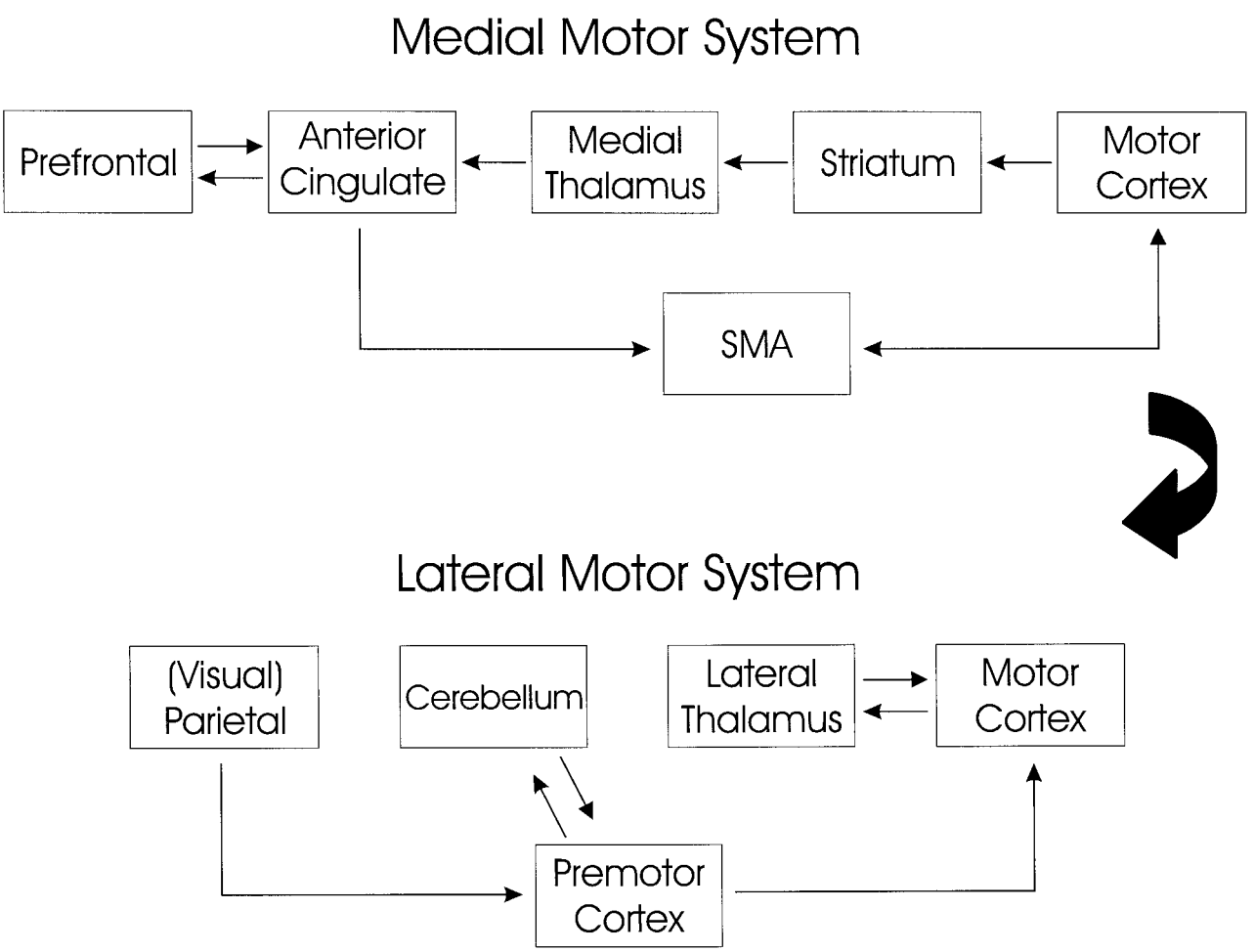


**Fig. 2.** Differing inputs to motor control from the medial and lateral system. The medial system is assumed to have inhibitiory control over the lateral system, and damage to the medial system can result in abnormal motor activity such as utilization behavior.

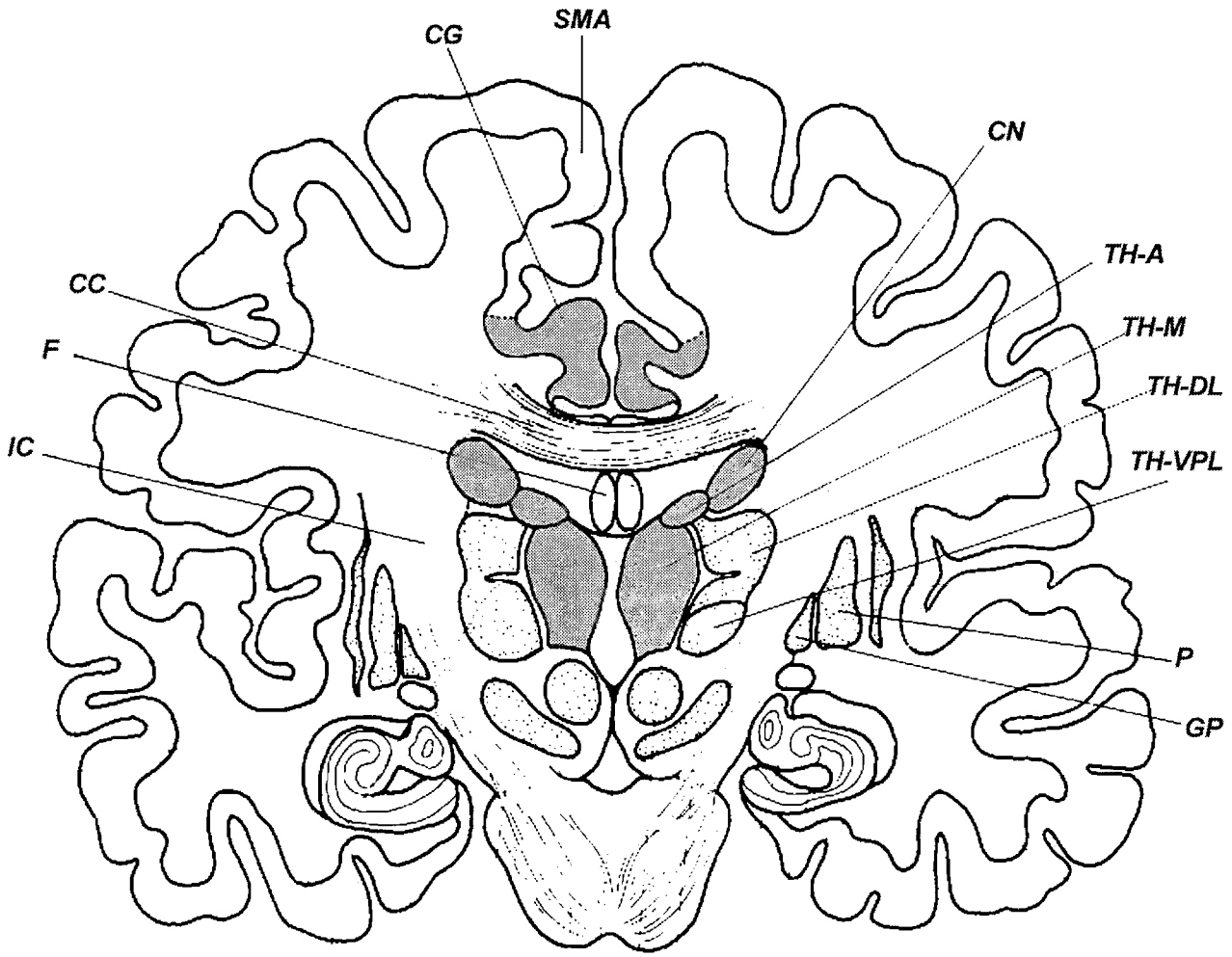


**Fig. 3.** Shaded areas indicate brain areas implicated in abnormal utilization behavior. Many pathological conditions result in bilateral damage to these midline thalamo-fronto-striatal structures. Key: SMA, supplementary motor area; CG, cingulated gyrus; CC, corpus callosum; F, fornix; IC, internal capsule; CN, caudate nucleus; thalamic nuclei (TH-A, anterior nucleus; TH-M, medial nucleus; TH-DL, dorsolateral nucleus; TH-VPL, ventral posterior nucleus); lenticular nuclei (P, putamen; GP, globus pallidus).

nuclei of the thalamus, all of which are closely connected. Figure 3 is a coronal illustration on which the brain areas implicated in utilization behavior have been identified. Lesions of this area are often related to vascular or infectious processes and, as such, frequently involve multiple structures, so that the specific contributions of individual areas to behavior are difficult to discern. It is important to consider, however, that considerable differentiation of function within mesial frontal areas and connected subcortical structures is likely. Indeed, recent studies have suggested at least six additional "motor maps" representing the body are evident within the structures comprising the mesial frontal cortex, many having both ipsilateral and bilateral connections (Picard and Strick, 1996). These various motor homunculi have probably emerged phylogenetically and allowed for increasingly flexible control of motor output. It may also be that the more contextually based disorders, including IB, UB, and particularly EDS, may emerge with disruption of more complex and socially mediated motor planning functions involving more anterior portions of the prefrontal cortex.

## CLINICAL IMPLICATIONS AND NEW DIRECTIONS

From a clinical perspective, observation and/or elicitation of this constellation of unusual motor signs and symptoms may be helpful in gaining a better understanding of conditions involving the mesial frontal cortex and

fronto-striatal connections. If these disorders are to be better understood, it will be important to develop and use clearer definitions and descriptions. It will also be important to note possible distinctions between induced and incidental approaches to elicitation of the behaviors and to incorporate this information into clinical observations and reports. These disorders typically occur in the context of intact parietal function, and some of the patients exhibiting these abnormal motor behaviors are intact on traditional neuropsychological testing.

It is interesting to note that a few studies to date suggest that UB may be a subset of IB, in the sense that IB appears to be more frequently observed, and that UB is rarely if ever seen without IB when both have been studied. Replications will be important in substantiating the generalizability of these preliminary observations. However, the findings suggest that IB may be a more sensitive indicator of pathology than UB and that both should be examined clinically.

To date, motor behaviors such as GR, MGB, IB, and UB have rarely been described or studied outside the context of striking, acquired pathology in adults with clear indication of central nervous system involvement. However, there are some reports suggesting that subtle or less obviously pathological behaviors in these same domains are evident in other populations believed to have primary involvement of frontal structures. Frith and Done (1989), for example, documented problems with control of internally generated, voluntary motor responses in individuals with schizophrenia. They argue that a similar failure to monitor internally generated thoughts and self-statements may be the basis for psychotic symptoms such as hearing voices. Lhermitte (1993) studied IB and UB in 60 psychiatric patients. IB or UB were present in every one of 18 cases diagnosed as having a major depressive episode and in 15% of 19 cases diagnosed as having a dysthymic episode. These were unexpected focal neurological signs in individuals with depressive illness. However, the findings were felt to be consistent with the frontal hypoactivity shown by PET studies in individuals with major depression.

Finally, in a recent study of children with attention deficit hyperactivity disorder (ADHD), children with ADHD were found to have a significantly higher incidence of utilization behavior compared with control children (Archibald, 2001). UB was noted in response to objects seen in the visual environment, but not in response to items the children knew about, but that were not in their line of sight. In contrast, self-touching movements did not occur with a higher frequency in children with ADHD as compared to controls, suggesting that these children were not just more active or restless. Instructions not to touch items made no difference in the manifestation of the behavior. ADHD has been shown to be associated with hypofrontality and other frontal lobe abnormalities (Casey, in press; Casey *et al.*, 1997; Filipek *et al.*, 1997; Sieg *et al.*, 1995; Zametkin *et al.*, 1993).

Barkley (1997) has described ADHD as reflecting a disturbance of executive functions. In his view, the development of executive functions, from both an ontogenetic and phylogenetic perspective, allows shifting overt (public) responses to covert (private) responses as a means of self-regulation. The development of inhibition allows one to override otherwise automatic responses to external stimuli. Humans have evolved to regulate and monitor their own behaviors conferring a distinct advantage in highly social group-living environment. Without such inhibitory control, behavior would appear impulsive, reflexive, and potentially inappropriate to the moment. Indeed, Barkley proposed that some of the behavioral manifestations of ADHD might reflect aspects of a UB syndrome, similar to that seen in adults with neurological damage, a hypothesis borne out in recent work by Archibald (2001). More recently, Barkley (2001) has elaborated on his model of executive function indicating that executive functions serve to shift the control of behavior from the immediate context and the 'temporal now' to self-regulation by internal representations regarding the 'hypothetical future.' The external and internal motor control systems described in this paper with respect to UB might serve as a neurological mechanism for such distinctions. The medial system may be necessary to inhibit inappropriate externalized behaviors triggered by environmental stimuli.

Finally, Saver and Damasio (1991) proposed the notion of 'acquired sociopathy' in the context of frontal lobe lesions. According to this model, individuals with orbitomedial prefrontal cortex lesions seem to be led by immediate gratification from external sources or an inability to profit from the negative consequences of their behavior or both, often resulting in significant personal and social difficulties. UB (or a more generic EDS) could be considered in the context of broader behavioral manifestations, in that individuals with injury or damage to orbitomedial regions may not be able to inhibit approaching and utilizing environmental triggers, resulting in behaviors that may be risky or dangerous to themselves or others.

## SUMMARY

The pathophysiology of UB, as well as other abnormal motor responses such as GR and MGB, appears to involve dysfunction in structures of the mesial frontal lobe and fronto-striatal pathways. In particular, the SMA,

cingulate gyrus, and both basal ganglia thalamocortical circuits, appear to be involved. UB is hypothesized to result from an imbalance between a medial motor system, which is responsible for internally generated control of movement and goal-directed action, and a lateral motor system, which facilitates monitoring and responding to external environmental stimuli. The medial pathway is thought to function upstream from and to exert control over the lateral pathway.

UB and other motor release phenomena have been well described in cases of focal neurological disease or disorders involving the mesial frontal lobe, and interconnected subcortical structures including the basal ganglia and thalamus. Given the prevalence of frontal brain system involvement in many neurological and psychiatric disorders, future studies may wish to investigate aspects of UB within other acquired and developmental disorders with documented or suspected frontal-striatal pathology (e.g., Parkinson disease and other frontal dementias, schizophrenia, depressive disorders, attention deficit hyperactivity disorder, traumatic brain injury). Indeed, UB may prove to be related to high levels of intrusive, dysruptive, and inappropriate motor behaviors often seen in individuals with frontal system damage or dysfunction.

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