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A Role for Volition and Attention in the Generation of New Brain Circuitry

Toward A Neurobiology of Mental Force

Obsessive-compulsive disorder (OCD) is a commonly occurring neuropsychiatric condition characterized by bothersome intrusive thoughts and urges that frequently lead to repetitive dysfunctional behaviours such as excessive handwashing. There are well-documented alterations in cerebral function which appear to be closely related to the manifestation of these symptoms. Controlled studies of cognitive-behavioural therapy (CBT) techniques utilizing the active refocusing of attention away from the intrusive phenomena of OCD and onto adaptive alternative activities have demonstrated both significant improvements in clinical symptoms and systematic changes in the pathological brain circuitry associated with them. Careful investigation of the relationships between the experiential and putative neurophysiological processes involved in these changes can offer useful insights into volitional aspects of cerebral function.

Introduction

Advances in the field of neuroscience over the past several decades have greatly enhanced our ability to demonstrate systematic and experimentally verifiable relationships between a wide array of conscious experiences and brain mechanisms which can reasonably be thought to underlie them. A prime example of this kind of work involves recent advances in our understanding of obsessive-compulsive disorder (OCD), a condition affecting approximately 2% of the population (Rasmussen & Eisen, 1998). OCD is characterized by intrusive thoughts and urges that often result in the performance of dysfunctional repetitive behaviours such as excessive handwashing or ritualistic counting and checking. There is now a broad consensus among neuropsychiatrists that brain circuitry contained within the orbital frontal cortex (OFC), anterior cingulate gyrus and the basal ganglia is intimately involved in the expression of the symptoms of OCD (for recent reviews see Schwartz 1997a & b, Rauch & Baxter, 1998).

The manner in which OCD manifests its symptoms renders it a particularly apt subject for the investigation of natural phenomena which occur at the mind–brain interface. People who are affected by this condition are generally quite aware that the bothersome intrusive thoughts and urges with which they are suffering are inappro-

priate and adventitious in the literal sense i.e., the symptoms are experienced as unwanted and extraneous intrusions into consciousness, and have a quality which has classically been described in the clinical literature as 'ego-dystonic' or 'ego-alien' implying 'foreign to one's experience of oneself as a psychological being' (Nemiah & Uhde, 1989). Because of this, people afflicted with OCD can frequently give clear and precise descriptions of how the symptoms are subjectively experienced, which allows investigators to perform studies of how patterns of cerebral activity change with symptom onset. Scott Rauch and the brain imaging group at Harvard University performed studies of just this kind, utilizing both PET and functional MRI to demonstrate changes in cerebral activity after acute symptom exacerbation in patients with OCD. Exposure of patients to stimuli which elicit the intrusive thoughts and urges typical of OCD (e.g., an intense sense of dread and contamination evoked by being visually exposed to a dirty glove) was accompanied by markedly increased activity in the OFC and the anterior cingulate gyrus, brain regions with a well demonstrated capacity to generate alerting 'error-detection' type signals in response to unanticipated alterations in the environment, as well as in the caudate nucleus, a key component of the basal ganglia and the major sub-cortical projection site involved in the functional modulation of the OFC and cingulate (Rauch *et al.*, 1994; Breite *et al.*, 1996).

The ability of OCD patients to clearly describe their symptoms also allows the investigation of how their conscious experiences change with treatment. Since there are now very effective means of alleviating OCD symptoms through the utilization of both pharmacological and psychological interventions (Jenike, 1998; Tallis, 1995; Van Oppen *et al.*, 1995), as well as significant evidence that each of these treatments independently cause similar changes in patterns of cerebral glucose metabolism in patients who respond to them (Baxter *et al.*, 1992; Schwartz *et al.*, 1996), it has become possible to track how post-treatment changes in the cerebral metabolism of functionally well-characterized brain circuits relate to changes in the internal conscious experience of clearly defined neuropsychiatric symptoms.

The use of cognitive-behavioural therapy (CBT) as a means of enabling people suffering from OCD to overcome their repetitive responses to bothersome intrusive conscious phenomena offers a valuable source of data to those interested in the study of the mind-brain interface. Because there is strong evidence that the core experience common to essentially all OCD symptoms — a gnawing, intrusive, inescapable and predominantly passively experienced sense that 'something is wrong' — is generated by faulty brain circuitry (for review see Schwartz, 1997a; 1998a), a close examination of the mental processes used by people learning how to wilfully alter their behavioural responses to OCD can yield significant insights about the processes whereby changes in the meaning or value one places on distressing conscious phenomena can result in active changes in how one responds to those phenomena. The training techniques which are used to accomplish that clinical goal explicitly encourage OCD sufferers to arrive at a new understanding of the relationship between their brain, their conscious experience, and their choice of behavioural responses to that experience (Schwartz, 1996). Since an understanding of the basic brain circuitry involved in OCD forms a key part in the theoretical basis of the cognitive training techniques we utilize at UCLA, and since significant alterations in the metabolic activity of those circuits occurs in response to successful treatment, a brief review of the basic brain mechanisms of OCD seems in order.

OCD: Basic Brain Circuitry

There is, as mentioned above, a broad consensus that brain circuitry contained within the OFC, anterior cingulate gyrus and the basal ganglia is intimately involved in the expression of the symptoms of OCD. The findings of cerebral metabolic rate changes in this circuitry after successful CBT are especially relevant to questions concerning mind-brain relations in light of recent advances in the fields of anatomy and physiology concerning the functional connectivity of these structures. The major findings of this research have been summarized in several elegant review articles (Graybiel *et al.*, 1994; Saint-Cyr *et al.*, 1995; Zald and Kim, 1996a,b). A brief overview of some of this work may help both to elucidate cerebral mechanisms relevant to the pathophysiology of OCD, as well as begin to clarify the relationship between brain function and internal experience in ways that can enhance our understanding of the topology of the mind–brain interface.

A. Micro-anatomy of the caudate nucleus

The basal ganglia, which include the striatum (comprised of the caudate nucleus and putamen), have been implicated in numerous studies of OCD (see Rauch, Whalen *et al.*, 1998 for review). Recent advances in our understanding of the micro-circuitry of the striatum are potentially of great relevance to understanding brain mechanisms of OCD and its treatment.

The entire striatum (fig. 1) contains a profoundly complex system of interacting neuronal micro-circuits or 'modules' comprised of neurochemically specialized zones called striosomes dispersed within a larger compartment called the matrix (see Graybiel *et al.*, 1994 for review). These striosomes, which have a patchy appearance, receive inputs primarily from limbic system structures such as the amygdala and project to the dopamine-containing neurons of the substantia nigra pars compacta (Gerfen, 1992). This pattern of connectivity strongly suggests a role for striosomes in mediating aspects of striatal modulation of emotional arousal (see Graybiel, 1995 for review). Moreover, the prefrontal cortex, which plays a primary role in assessing the behavioural relevance of environmental inputs, also projects into this system. Interestingly, the two prefrontal structures which send the densest projections into the striosomes of the caudate nucleus are the OFC and anterior cingulate gyrus (Eblen and Graybiel, 1995). This seems especially noteworthy given the very substantial data base implicating these two structures in OCD pathology (Rauch and Baxter, 1998) as well as in both pharmacological (Saxena *et al.*, 1998) and psychological treatment response (Schwartz, 1998a).

In contrast to the select striosomal projections of the orbital cortex and cingulate gyrus, projections from the rest of the prefrontal cortex go primarily to the matrix compartment. Projections from areas of the lateral convexity of the cerebral hemispheres, which play a key role in processing information for high order tasks such as anticipation and planning (Stuss and Benson, 1986; Fuster, 1989), seem to be of particular interest, however. These projections form other quite distinct patchy distributions within the matrix, which have been termed matrisomes (Flaherty and Graybiel, 1994). These matrisomes are consistently found in close spatial proximity to striosomes. This sort of micro-anatomical arrangement could be quite conducive to functional interactions between the limbic associated striosomes and matrisomes made up of frontal association cortex projections. This is especially so given the discovery of

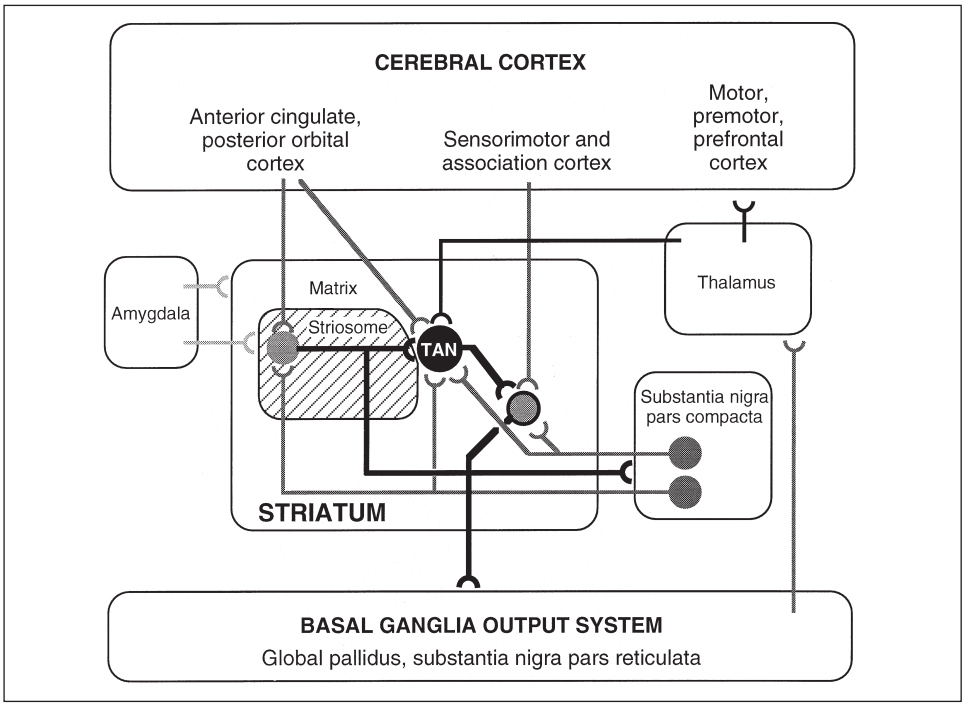

Figure 1. Tonically active neurons (TAN), which tend to localize adjacent to striosomes, appear to integrate information related to limbic system function with other cortical inputs to the striatum. Changes in TAN response patterns during behavioural learning could modify output functions of the basal ganglia. (Localization of matrisomes within the matrix is not shown in this diagram. See text for details.) Adapted from T. Aosaki, M. Kimura and A.M. Graybiel, *J. Neurophysiology*, **73**, 1234–52 (March 1995); schematic revision courtesy of *Science & Medicine*, 1997, **4** (2), p. 20.

cells of a highly specialized nature which seem to localize at striosome-matrix borders, and are in a position to integrate information from both striosomes and matrisomes. These cells, called tonically active neurons or TANs (figure 1), are of great interest insofar as they demonstrate a distinct pattern of firing when cues linked to reward delivery are presented during behavioural conditioning (Aosaki *et al.*, 1995). Modification of TAN activity by behaviourally meaningful stimuli could potentially serve as a gating mechanism for re-directing information flow through the striatum during learning. In this way TANs could potentially help select and generate new patterns of striatal activity in response to the integration of behaviourally significant information. This kind of functional re-gating of neuronal activity patterns could be profoundly important during the acquisition of new behavioural skills in the course of doing CBT which, as we will see, requires the applied use of behavioural planning to modulate one's responses to the powerful emotional feelings mediated by limbic-related areas of the brain.

B. The caudate's role in gating thoughts and emotions

A number of researchers who have studied how cortical circuitry is modulated by the caudate during the preparation for and initiation of behavioural activity have used the concept of gating to help explain this process (Baxter, 1995; Swerdlow, 1995). How the flow of information is gated through the complex circuitry of the basal ganglia

will help determine whether the signals received by the thalamus result in subsequent excitation or inhibition of behaviourally critical cortical areas. Imbalances in gating the flow of cortical output can thus affect relative levels of cerebral excitation and inhibition in ways which profoundly influence an organism's conscious experience. The study of OCD provides a striking example of this, as demonstrated by the data of the Rauch group at Harvard (see above) in which the essentially contemporaneous onset of intrusive OCD mental phenomena and markedly increased activity in the OFC, cingulate, caudate and thalamus was seen.

Basic research in behavioural neuroscience by the groups of Okihide Hikosaka of Tokyo and Jean Saint-Cyr of Toronto provides further insight into the potential relevance of this circuitry to the symptoms of OCD. In a series of elegant experiments into the mechanisms of saccadic eye movements occurring in response to reward-related visual cues, Hikosaka *et al.* (1989) demonstrated that the caudate can rapidly modulate the activity of neural circuits involved in the preparation for and guidance of environmentally relevant behaviours. Saint-Cyr *et al.* (1995) have proposed that analogous mechanisms involving caudate-prefrontal cortex interactions could enable what he metaphorically calls a 'cognitive grasp reflex' for circuitry used in habitual behaviours. The Saint-Cyr group's research indicates that the caudate is likely to be involved in the development of 'habit patterns' or behavioural responses which can be rapidly mobilized without a large amount of conscious thought or awareness. While these patterns of habitual response are potentially quite adaptive, they could form the basis for symptoms of a disease such as OCD if behaviours of this type were pathologically and repetitively generated by dysfunctional basal ganglia circuits.

Baxter (1995) and Swerdlow (1995) have similarly noted how imbalances in the internal circuitry of the basal ganglia could lead to excessive disinhibition of thalamo-cortical pathways resulting in the self-sustaining activation of a 'worry circuit' involving the OFC and cingulate. The key point on which all of this reasoning is based is that a malfunction in the caudate and associated basal ganglia structures could lead to the dysfunctional gating of cortical circuitry, with resulting intrusions of pathological thoughts and sensations resulting in compulsive behavioural responses.

C. A role for the OFC and cingulate in error detection

The now well established (Rauch & Baxter, 1998) finding of increased metabolic activity in the OFC and anterior cingulate in people suffering from OCD, a disease state characterized by inappropriate repetitive thoughts and behaviours, is quite consistent with the results of prior research on the behavioural physiology of the these two key structures of the limbic cortex. Neurologists have long recognized that patients with damage to these areas of the cortex demonstrate behavioural perseverations, the repetitive performance of behaviours no longer serving a useful function. These patients are now well documented to show deficits in assessing the future consequences of their actions as well as problems associated with socially inappropriate behaviours (Bechara *et al.*, 1994). Studies on the cellular physiology of OFC and cingulate in behaving monkeys provide a firm foundation for interpreting these clinical findings.

In a classic set of studies done in an attempt to clarify the behavioural physiology of the frontal lobe, several groups (Niki and Watanabe, 1979; Rosenkilde *et al.*, 1981; Thorpe *et al.*, 1983) investigated neuronal firing patterns in the OFC and anterior cin-

gulate in monkeys trained to respond to various visual cues in order to receive juice as a reward. These experiments revealed several important aspects of OFC and cingulate function. First, neurons in these structures change their firing pattern in response to visual cues depending on whether these cues are associated with rewarding stimuli. Seeing something associated with a reward triggers neuronal firing in both OFC and anterior cingulate. Thus one aspect of neuronal signalling in these structures is related to informing the organism about the presence of stimuli in the environment which are behaviourally significant. Further, and especially important for understanding behavioural perseveration, responses in both of these areas are very sensitive to the expectations the organism has concerning the stimuli to which it is exposed. For instance, if a monkey comes to expect that a light is associated with receiving juice, but no juice is delivered after the light appears, bursts of neuronal firing will occur in OFC and anterior cingulate. These cellular responses are not elicited after trials in which the expected reward is delivered. These responses, which can be understood as an 'error detection' mechanism, can underlie an internal sense in an organism that 'something is wrong' in the environment. If damage to these structures, with a resulting loss of 'error detection' signals, impairs an organism's capacity to realize that things have changed in the environment and that stimuli previously associated with reward no longer are, it becomes quite understandable why behavioural responses to those stimuli could become repetitive and demonstrate much slower and less effective behavioural adaptation.

In contrast to perseverative behaviours performed as the result of a *failure* of OFC and cingulate to generate appropriate 'error detection' signals, obsessions and compulsions can be understood as behavioural perseverations corresponding to signals of this type being *excessively* generated in a repeated and inappropriate manner due to hyperactivity of these structures. The clinical manifestation of this overactivation, and the associated generation of adventitious 'error detection' signals, would be an internal sense of dread accompanied by an intractable feeling that 'something is wrong.' This kind of an internal feeling state closely approximates how patients describe the experience of OCD symptoms. There is a significant consensus among neurobiologists (see Saxena, Brody *et al.*, 1998; Rauch, Whalen *et al.*, 1998 for review) that an impairment in the modulation of OFC and cingulate activity by the caudate nucleus is a key aspect of the pathophysiology of these symptoms in OCD.

D. Overview

The perspective of the Graybiel group at MIT on the physiology of the cortico-striate system forms an overview which can serve to coherently integrate the large body of neuroscience data presented above (fig. 1). These investigators have generated a body of work which allows us to view the caudate nucleus as an extraordinary mosaic composed of juxtaposed micro-anatomical modules with striosomes, receiving inputs from the OFC, anterior cingulate and other brain structures intimately related to emotional expression, and matrisomes receiving inputs from areas related to behavioural planning. Lying between these two modules are specialized cells which can change their firing patterns in distinctive ways when the organism is presented with stimuli which are perceived to have behavioural significance. This arrangement of anatomical elements could be quite conducive to adaptive changes in the gating of cortical signals related to modifications of behaviour in response to new information.

While much work remains to be done to further elaborate the detailed workings of this system, the relevance of these findings to formulating a possible neuroanatomical foundation for the physiological integration of thought and emotion is a very exciting development in the field of behavioural neuroscience. Further, conceptual models of this sort may well help us to better understand the neural mechanisms which are involved in the acquisition of new behavioural skills by patients with OCD during the course of doing CBT.

The applied use of anticipation and planning to alter behavioural responses to OCD's powerful intrusive thoughts and urges is the key element to success in overcoming the symptoms of this potentially debilitating neuropsychiatric condition. As reviewed above, there are strong reasons to believe that aberrant 'error detection' messages resulting from faulty gating of neuronal information within circuitry involving the OFC, anterior cingulate and the striosome compartment of the caudate nucleus are intimately involved in the generation of the pathological intrusions into consciousness that are the core element of OCD symptoms. In addition, circuits operating within the lateral convexity of the frontal lobe, and projecting into the caudate nucleus at least in part in the form of matrisomes, are very likely involved in the thought processes required for the execution of CBT strategies. These neuronal elements exist in close juxtaposition to highly specialized TAN cells in the caudate which appear to be sensitive to changes in the perceived relevance of sensory inputs and are extremely well positioned to alter the gating of information needed to alter behavioural responses to those inputs. All the necessary neural ingredients seem in place for constructing a meaningful theory of the cerebral mechanism of CBT response in OCD, and the associated and well-documented alterations of cerebral function that accompany it (Schwartz *et al.*, 1996, and below). Yet, to an experienced clinician who actually works with people afflicted with this problem, one huge element of the theory seems totally unaccounted for — the element of effort which is so critical to driving the treatment forward in a real-life situation. For all our discussion of cognition, information processing, and cerebral processing in turn of the century models of mind-brain relations in neuroscience and philosophy of mind, the critical role of effort as a necessary component for keeping the machinery on track and functioning does not seem to adequately enter the picture. But in the case of OCD, a condition involving real people with real and now reasonably well-studied brains, we have an opportunity to take a closer look at the real-life process that occurs during therapeutic manoeuvers which result in systematic alterations of brain function. And, in so doing, we may uncover an opportunity to gain fresh insights into what promises to be one of the major new fields of investigation in the coming century — the role of volition in brain function.

CBT Interventions for OCD: An Active Approach at the Mind–Brain Interface

Behavioural approaches to the psychological treatment of OCD are based on the principle, empirically validated by three decades of research (Marks, 1987), that people suffering from the disorder can learn to perform adaptive behaviours instead of pathological ones in response to the intrusive thoughts and urges which comprise the core symptoms of the condition. To successfully complete therapy patients must effectively tolerate and re-direct their responses to the acutely uncomfortable feeling states

that arise as a result of OCD pathophysiology. There is now substantial evidence that the acquisition of specific cognitive skills by patients enables them to perform behavioural therapy techniques more effectively by increasing their ability to maintain their attentional focus on functional activities when confronted by the intensely uncomfortable thoughts and urges that arise during treatment and the profound distractions they understandably cause (Steketee *et al.*, 1998).

At UCLA we have developed a four-step cognitive-behavioural training method (Table 1) which is specifically designed to help patients successfully refocus their attention away from the steady bombardment of intrusive symptoms into their conscious awareness. The educational approach of the method is organized around the working hypothesis that the intrusive thoughts and urges of OCD are caused to a significant degree by a biomedical disease state (Schwartz, 1996; 1997b), and an active attempt should be made to re-interpret the significance of these intrusions so as to better understand how to behaviourally respond to them.

One major goal of this training is to deepen the appreciation of the OCD sufferer to the nature of the relationship that exists between the distressing thoughts and urges intruding into their consciousness and what are basically 'false brain messages' which can safely be ignored. A critical underlying assumption of this process is that an electro-chemical malfunction is causing serious distracting phenomena to intrude into conscious awareness — an assumption which is, of course, now buttressed by a sizable scientific data base, which is presented as part of the educational component of the therapy in ways commensurate with the individual sufferer's cognitive capacity. The goal of treatment is, of course, to learn to respond to these 'false brain messages' in new and much more adaptive ways. This is accomplished through the utilization of techniques of behavioural refocusing, usually applied within a largely self-directed training paradigm in which functional activities are systematically performed in place of habitual OCD responses. These cognitive-behavioural training techniques enable patients to utilize improved self-monitoring capabilities in order to more accurately interpret their conscious experience, resulting in an improved ability to manage their emotional and behavioural responses to the intense anxiety caused by OCD symptoms. This results in an enhanced ability to maintain attentional focus on the performance of consciously chosen adaptive behaviours, rather than capitulating to automaton-like compulsive responses like repetitive washing and checking, when besieged by the fearsome thoughts and urges of OCD.

Table 1: The Four Steps of Cognitive–Behavioural Treatment for OCD

1. Relable	Recognize the intrusive obsessive thoughts and urges as a *result of OCD*.
2. Reattribute	Realize that the intensity and intrusiveness of the thought or urge is *caused by OCD*; it is probably related to a brain biochemical imbalance. Remember: *It's not me, it's the OCD*.
3. Refocus	'Work around' the OCD thoughts by focusing attention on something else at least for a few minutes, i.e., *do another behaviour.*
4. Revalue	Do not take the OCD thought at 'face value'. It is not significant in itself.

(Adapted from Schwartz, 1996)

As the patient's understanding that intrusive OCD symptoms are merely 'false brain messages' is increasingly well integrated into his/her cognitive framework, an extremely important transition begins to take place. The very nature of the conscious experience of the uncomfortable feeling of an OCD symptom begins to change in ways that allow him/her to increasingly create a mental distance or space between the experience of self and the experience of the symptom. While this change in the perception of the nature of the symptom is in some sense an accentuation of the 'ego--dystonic' or 'ego-alien' aspect of it, that is only one small component of the therapeutic process. The essence of this adaptive change in perspective is that the person with OCD becomes increasingly able to experience the intrusive symptom from the point of view of a clear-minded observer, and thus comes to see the symptom as merely the result of a malfunctioning mechanical process in the brain which, while unpleasant, is not of any great personal concern.

It is the ability to observe one's own internal sensations with the calm clarity of an external witness that is the most noteworthy aspect of this experience. Within the terminology of traditional Buddhist philosophy this sort of mental action is called mindfulness or mindful awareness (Silananda, 1990; Schwartz, 1998b). The German monk Nyanaponika Thera, a major figure of twentieth century Buddhist scholarship, coined the term 'Bare Attention' in order to precisely explain to Westerners the type of mental activity required for the attainment of mindful awareness, which by the practice of meditation can be developed into what is called in Pâli *vipassanâ,* or insight. 'Bare Attention is the clear and single-minded awareness of what actually happens *to* us and *in* us, at the successive moments of perception. It is called 'bare,' because it attends just to the bare facts of a perception as presented either through the five physical senses or through the mind. . . without reacting to them.' (Nyanaponika Thera, 1962, p. 30) As a practical matter, shifting one's perspective in this way requires substantial and quite directed effort, especially when it is done in the presence of significant anxiety and fear. It is certainly not a shift that tends to occur spontaneously — it requires significant anticipation and forethought. Yet it is the mental act of adverting attention in this manner which enables sufferers of OCD to develop the insight necessary for consciously choosing new and more adaptive responses to the intrusive and intensely bothersome thoughts and urges which bombard their consciousness.

Mental states of this kind have been described in modern Western philosophy at least since the eighteenth century. Noting the sense in which conscious activity of this kind involves clear observation by, as it were, 'a man within,' the Scottish philosopher Adam Smith (1976) described the associated mental experience as the perspective of 'the impartial spectator.' During CBT utilizing the Four Steps in Table 1, the terms 'mindfulness,' 'mindful awareness,' and 'impartial spectator' are all commonly used to help clarify for patients how to apply their new insight into the biomedical nature of OCD symptoms to help them create a distance between the conscious experience or feel *of* the symptom and their self-concept (Schwartz, 1996 and 1997b). Thus, for example, patients learn to stop making self-statements like, 'I feel like I need to wash my hands again,' and instead make statements of the type, 'That nasty compulsive urge is bothering me again.' This process, which requires profound and painstaking effort, can significantly enable patients to more effectively manage the fears and anxieties associated with OCD symptoms and improve their

ability to refocus attention away from the symptoms and onto functional activities. This results in a markedly enhanced ability to prevent the mind from succumbing to the intense distractions that OCD thoughts and urges create, and so increases the capacity to consistently alter behavioural responses in increasingly adaptive ways.

Effects of Cognitive-Behavioural Treatment on Cerebral Function

Systematic changes in cerebral glucose metabolism accompany the clinical improvements achieved using this method of cognitive-behavioural therapy (Schwartz *et al.*, 1996). We investigated cerebral metabolic rate changes in eighteen drug-free subjects studied with PET scans before and after ten weeks of outpatient treatment. Twelve of them demonstrated clinically significant decreases in OCD symptom severity during the treatment period, and six did not. There were two main findings in this study:

(1) Bilateral decreases in caudate nucleus metabolism, divided by ipsilateral hemisphere metabolism (Cd/hem), were seen in responders to treatment compared to non-responders. This finding was more robust on the right (p = .003) than on the left (p = .02). (See fig. 2)

(2) In the right hemisphere, prior to treatment, there were highly significant patho-

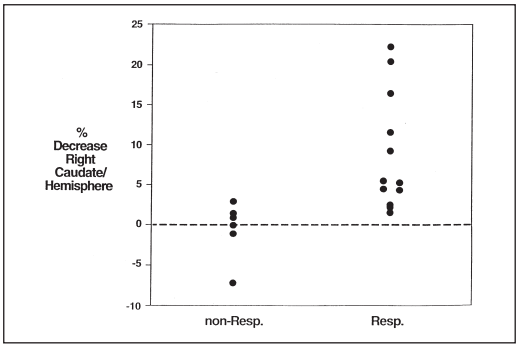

Figure 2. Change in Right Cuadate Glucose Metabolic Rate in Non-responders and Responders to Cognitive-Behavioural Therapy.

Plot of percent change after cognitive-behavioural treatment ((pre-post/pre) x 100) in right head of caudate nucleus metabolic rate divided by ipsilateral hemisphere (caudate/hem) for responders and non-responders to treatment. There is a significant difference between responders and non-responders to treatment (p = .003).

logical correlations between the metabolic rate in OFC and the metabolic rates in the caudate nucleus, anterior cingulate, and thalamus. These four brain structures acted as if they were functionally locked together. After effective treatment with CBT, these pathological correlations were significantly reduced. (See Table 2)

Table 2: Extent of Correlation (*r* Values) Between Metabolic Rates in Brain Structures

Left hemisphere	*Before*	*After*	**Right hemisphere**	*Before*	*After*	
Left orbit to left caudate	0.46	–0.01	Right orbit to right caudate	0.74	0.28	*
Left orbit to left cingulate	0.11	0.58	Right orbit to right cingulate	0.87	0.22	*
Left orbit to left thalamus	0.34	0.05	Right orbit to right thalamus	0.81	0.14	*
Left caudate to left thalamus	0.66	0.36	Right caudate to right thalamus	0.69	0.41	

Data from 12 patients who successfully responded to drug-free cognitive-behavioural therapy. * = Significant ($p<.05$) difference between before-treatment and after-treatment correlation. Adapted from Schwartz (1997a).

Mindful Awareness, Attentional Refocusing, and Cerebral Change

The reasoning on which the CBT model described above is based derives to a significant degree from developments over the past few decades in the field of behavioural neurobiology (see literature review above). Given the large data base demonstrating that the basal ganglia are involved in the modulation of cortical circuitry involved in the expression of previously learned behaviours, and the growing evidence implicating frontal aspects of that circuitry in the expression of OCD symptoms, it seems reasonable to teach OCD patients a technique intended to enable them to activate more functionally adaptive circuits within the vast array of neuronal connections comprising the cortico-striate system. If OCD symptoms are related to a malfunction in cortical circuit activation, and in particular to an 'error-detection' mechanism in the OFC and cingulate (Schwartz, 1997a and 1998a), then activation of alternative circuitry through the focused performance of adaptive alternative behaviours might, over time, ameliorate the discomfort related to the faulty brain mechanism. On this basis the third step of this cognitive-behavioural method, the Refocus step, gradually evolved. In this step the concept of 'working around' OCD symptoms is stressed. This means learning *not* to wait for the urges to subside before changing behaviour. Instead, patients must learn to change behavioural responses while the uncomfortable intrusive urges are still present in conscious awareness. By shifting to another task as a systematic response to the inner experience of OCD symptoms, new adaptive responses to intrusive OCD thoughts and urges are learned. In conjunction with that process, significant changes are seen in the activity of circuitry which a growing body of research finds related to OCD pathophysiology. The fact that the metabolic activity in this circuitry changes in a manner which is significantly related to alterations in symptom expression (fig. 2 and table 2) underscores the possibility that there is a *causal* relationship between the changes in experience and in brain function.

It is of the utmost importance to appreciate that the changes in behavioural response to the conscious intrusions that characterize OCD are not in any way a passive process, but are, in the most intuitively meaningful sense, active and purposeful choices to respond differently to profoundly distracting aspects of conscious experience. To view this critical therapeutic process, the essence of what the term self-help genuinely refers to, as merely the preordained outcome of an abstract neuronal vector analysis passively occurring in some vast matrix which we generically call the 'nervous system,' reflects a serious misunderstanding about the nature of what actually occurs during the course of a real-life treatment of a case of OCD. In an attempt to clarify this critically important point, it is worth examining in overview some of the clinical data that comprise a typical treatment history.

Let's consider, for purposes of illustration, the therapeutic process in the case of a man with typical OCD. (The clinical aspects of this case will, to save space, be markedly abbreviated. See Schwartz, 1996 for details) At the outset of treatment this man is besieged by very intrusive and persistent thoughts and urges associated with a gnawing gut-level feeling of dread that his hands are contaminated with germs. This almost invariably leads to hand washing of such severity that it causes the skin to become red, raw and chapped. Although he knows that his concerns about germ contamination are excessive to the point of being nonsensical, the gnawing anxiety associated with the obsessive thoughts of possible contamination is so intense he almost

invariably succumbs to it with bouts of hand washing. A large body of research data is consistent with the statement that the intrusive gnawing fear that 'something is wrong' with his hands (e.g., they are 'contaminated by germs') is caused by error-detection circuitry which is generating an inappropriate signal. This faulty signal is probably causally linked to excessive neuronal activity in circuitry connecting his caudate nucleus, OFC and anterior cingulate. When we explain this to the man, and give him associated instructional materials, emotional support and time, he comes to readily understand it. This learning process comprises the first two steps, Relabel and Reattribute, of the Four Step Method (see Table 1) which is used as part of the treatment process.

From a clinical perspective, what has the man learned: what is the essence of his new understanding? The practical core of his new knowledge is that the reason his intrusive persistent thoughts and urges are so damnably persistent, the reason they *don't go away,* is due a biochemical imbalance in his brain which results in his consciousness being bombarded with a steady stream of 'false error messages' or (as many patients prefer to call them) 'false alarms.' In other words, he has significantly improved his ability to *correctly interpret* the signals being generated in his conscious awareness by the faulty operation of his brain. One major benefit that accrues to the man on account of this enhancement in the accuracy of his signal interpretations (e.g., from 'My hands feel dirty!! I better go wash them again!!' to 'That bothersome urge to wash caused by my OCD is acting up again.') is that it markedly alters his perspective concerning the *next question* he addresses to himself — the one which will guide the course of his next action, 'Should I wash my hands, or not?' As Stapp points out in detail in his related article in this issue, changing one's perspective with regard to the *next question* posed to nature, i.e., what aspect of nature one *focuses attention on,* has profound implications concerning the quality of the physical processes occurring at the mind-brain interface.

Clearly understanding the mental actions which are performed when our OCD patient applies his training in the first two steps of Relabel and Reattribute will help clarify why this process is so important. By Relabeling we mean making assertive self statements describing accurately the nature of the feeling you're experiencing when suffering from an attack of OCD (e.g.,'my hands don't *really* feel dirty — that's just OCD'). By Reattributing we mean working to remain aware of the fact that the bothersome feeling of OCD symptoms is caused by (and thus properly attributed to) a pathological, but treatable, brain-related imbalance (i.e., *not* my own goal directed mind — thus the popular saying among OCD sufferers 'It's not me, it's the OCD'). Another way of accomplishing these goals is through the systematic use of mental notes, a technique originally described in connection with the performance of vipassanâ mindfulness meditation in the Burmese tradition (Silananda, 1990). In this approach, the practitioner very systematically notes with brief self-statements just where the focus of attention is *in the present moment.* An example of this technique being applied to, say, walking (a common meditation subject in vipassanâ practice) would be, at the moment when lifting the foot, make the quick precise mental note 'lifting,' when moving the foot forward, 'moving,' when placing it down, 'placing' and so on (as one proceeds in meditation practice the noticings and mental notes become *much* more finely grained than in this simple example, especially with respect to the inner experiences associated with movement). When this meditation

technique is refined for clinical use in treating OCD it involves patients training themselves to take note of the fact that the bothersome feeling now intruding into their mind is 'OCD', or 'false message', or 'bothersome feeling', etc.

What this accomplishes is a change in perspective *away* from automatic responses (exactly the sort of activity the basal ganglia is wired by many millennia of evolution to perform (Graybiel 1995 and 1997)) and *toward* a more precise, considered, and consciously goal-directed interpretation of the present moment's experience — which is, of course, a much more cortically directed activity. In other words, from a neuroanatomical perspective, what the mind is doing while performing the first two steps of CBT or basic vipassanâ mindfulness meditation on walking is wilfully *shifting* the predominant brain mechanism which is utilized in performing the action going on *right now.* In the case of treating OCD, the mental action is performed to shift out of faulty basal ganglia circuitry and into activities which are more adaptive. In the case of working to sharpen vipassanâ, it is done to shift out of mindless habitual activity and convert to activity which is precisely observed and examined with mindful awareness. In both cases great effort is involved, not least because the brain, which very much has its own agenda, is bombarding consciousness with a series of distractions which must be overcome (i.e., *not* attended to) if success at one's chosen mental training is to be achieved. In the case of CBT the distraction is pathological, threatening and intense ('You're contaminated! Go Wash!), in meditation it's usually pretty mundane and benign ('This is boring. What's for dinner?'), but in both cases the focus of attention must be shifted away from where the brain 'wants' to take it — and in both cases there are important alterations in the biology (and physics — see Stapp, this issue) of the situation in response to the effort at refocusing attention being actualized *in the present moment* by the practitioner involved.

A key point to grasp when considering the experiential field in which the OCD patient is working is that meaningful changes in the raw sensation of the *actual feelings* of fear and anxiety that constitute OCD symptoms generally do not occur early on in treatment. As our man works to master his responses to the discomforting urges by refocusing his attention on alternative adaptive behaviours, his gut is still churning and the gnawing sense that 'You're contaminated!' is very definitely still present. A very profound and directed *effort* is required for him to refocus his attention in the face of such intense and (neurophysiologically speaking) almost literally gripping distracters. That's almost certainly because the brain mechanisms underlying the inner experience of his symptoms have not changed in any significant way. What *has* begun to change, and the factor that underlies the effort the man is now willing to make to resist those symptoms, is the *value* he now puts on those feelings and sensations. What he is beginning to do is to learn how to control his *emotional reactions* to those thoughts, feelings and sensations, by which I specifically mean the kinds of interpretations and meanings he attributes to them. In the early stages of treatment he basically *feels* the same — but he has begun to change in a critical way how he *understands* those feelings. With that change in understanding he has set the stage for making different choices about how to act on those feelings and sensations — choices which, in the conceptualization of the mind-brain interface formulated by Stapp (this issue), actually change the nature of the *physics* of the situation. Now, by consistently instituting steps three and four of the CBT method outlined in Table 1 — i.e., by Refocusing his behavioural output onto healthful rather than pathological behaviours,

while systematically Revaluing his inner experience of OCD symptoms — he can consciously establish new and much more adaptive response patterns to internal experiences which, prior to treatment, were almost always followed by nearly automatic pathological handwashing. And controlled scientific data now exist associating those new consciously chosen response patterns with statistically significant changes in energy use by the very brain circuitry which most probably underlies his painful intrusive thoughts and urges, changes which bring with them a marked amelioration of the intensity and severity of his mental suffering.

The Volitional Modulation of Cerebral Function

As discussed above, the groundbreaking study of Aosaki *et al.* (1995) demonstrated that highly specialized cells called tonically active neurons (TANs) display a distinctive pattern of firing during behavioural conditioning. Specifically, after monkeys were trained to associate the delivery of a juice reward with audible clicks or a light flash, TANs displayed a pause in tonic firing followed by a burst of rebound excitation upon presentation of these stimuli. Since, within the micro-anatomy of the striatum, TANs tend to localize at the interface of limbic and frontal association cortex input, it is hypothesized that these cells could serve a critical role as a gating mechanism for efficiently directing information flow through the striatum in response to behaviourally significant information (or, to use the exact terminology of the investigators, 'conditioned stimuli predictive of reward') (Graybiel *et al*., 1994). A brain mechanism such as this would also have the potential to provide internally experienced sensations which could function as cues enabling an organism to recognize a sensory stimulus as related to behaviours associated with the delivery of a reward.

Reflecting on how these cutting edge findings in the field of behavioural neuroscience relate to the process of therapeutic change that occurs during the psychological treatment of a person with OCD can shed significant light on the sorts of phenomena that occur at the mind-brain interface. To those committed to the investigation of volitional aspects of brain function, maintaining keen awareness of the critical role of *valuation* in the processing of conscious experience is perhaps the key factor for attaining a clear understanding of the available data. For, while the question of what constitutes a 'reward' (or 'behaviourally significant information') is generally a quite transparent one for non-human animals, for human beings it can raise issues of tremendous complexity. To understand these issues with even a modicum of clarity one must not lose sight of the fact that human consciousness exhibits tremendous latitude and variation with regard to not only the selection of what constitutes a 'reward' at any given moment, but also to the types of responses generated to the phenomena so chosen. This critical process of *valuing* experience has tremendous relevance to a proper understanding of the relationship between the quality of attention (e.g., mindful or non-mindful) directed toward both internal and external stimuli and the role of signal interpretation in the volitional initiation (or inhibition!) of motor responses to experiences generated by activity within the nervous system.

For a monkey, the association of a light flash with juice results in a predictable change in the firing pattern of TANs when the monkey is exposed to the light flash. As a result of its association with a clear-cut reward such as juice, the light flash becomes behaviourally significant information for the monkey. The striatum can then rapidly

and efficiently process input from relevant cortical circuitry in order to prepare and initiate effective behavioural responses (Hikosaka *et al.*, 1989; Graybiel, 1995). For our man with obsessional fears of contamination, a substantial data base now supports the hypothesis that upon exposure to a cloth soiled with axle grease the striatum will respond in a pathologic fashion. As reviewed above, work by the Rauch group indicates that circuitry connecting the OFC, anterior cingulate and striatum will become metabolically activated in such a situation. As a result the patient could be bombarded by feelings of impending catastrophe with resulting behavioural responses consisting of repetitive handwashing — behaviours which, to the symptomatic patient, are linked to a sense of 'reward' insofar as they give him an extremely brief and evanescent feeling that his contamination is momentarily 'alleviated'. Unfortunately for the poor OCD sufferer, this momentary relief is quickly terminated by the intrusion of still more biologically generated sensations of fear and contamination. It is the role of CBT to help the man improve his ability to accurately interpret the signal conveyed to him by visual exposure to stimuli which tend to induce OCD symptoms, such as greasy cloths (e.g., 'That's just axle grease. It's anxiety caused by OCD that's making me feel like washing my hands'). This change in the valuation of the stimulus, and the resulting change in the nature of the behaviourally significant information conveyed to the man by the greasy cloth, enables the man to volitionally inhibit his ingrained pathological rituals and instead actively generate an entirely different set of behavioural responses to such stimuli — behaviours, such as helping his wife in the garden, which the man, with much effort, comes to systematically link with a new and much more accurate and sustainable sense of reward. With this change in the valuation of his sensory experience, the man is performing a mental action which characterizes the human species — through the hard work of self-directed volitional training he is activating and bringing to fruition the potential for sapience that characterizes Homo sapiens at its best. At that moment of making a new choice he is unique among all of the Animal Kingdom. For his wilful change of his response to the intrusions of OCD into consciousness yields not only the rewards of adaptive as opposed to self-destructive behaviours, it also results in an extremely rewarding sense of true self-esteem — that empowering inner awareness that the utilization of knowledge has enhanced one's capacity for self-control.

Prior work by Libet (1985; 1998) has demonstrated cerebral activity which appears to indicate 'the possibility of a role for the conscious function . . . to be one of potentially blocking or vetoing the volitional process so that no actual motor action occurs. Veto of an urge to act is a common experience for individuals generally (1998, p. 215).' As we have seen, for a person with OCD to reliably and consistently veto the urge to perform the motor action involved in a compulsive ritual requires tremendous effort and considerable training. But it is certainly of interest that a large data base exists in normal subjects demonstrating brain mechanisms so clearly consistent with the kinds of volitional modulation of behaviour performed by OCD patients doing CBT. It also provides an extremely relevant context for understanding the data displayed in Figure 2 and Table 2, which demonstrate that the reductions in the bothersome symptoms that accompany successful CBT are associated with statistically significant changes in brain function. Changes in the valuation and interpretation of both inner and outer experience form a critical aspect of how these kinds of systematic changes in both volitional expression and cerebral function take place.

It seems a reasonable working hypothesis that cells such as TANs may play an important role in how this occurs by influencing the probability of the firing of sets of surrounding neurons in the striatum, resulting in more adaptive gating of neuronal information flow as more accurate and insightful interpretations of sensory stimuli lead to the preference of adaptive over maladaptive 'rewards,' and the learning of new responses to replace old OCD behavioural patterns takes place (Graybiel, 1995; 1997). However, from the perspective of understanding the neurobiological dynamics of a process such as this, the key point to focus on is that the modification of TAN activity that would accompany the change in sensory preference would not be likely to occur spontaneously — under the circumstances of a typical treatment it is hypothesized that such a change would occur gradually, step-by-step, over several weeks and require a significant focusing of brain activity generated by the concerted, sustained and highly directed effort exerted by the patient during the peformance of CBT.

Generating New Brain Circuits: The Role of Mindfulness and Mental Force

The finding that CBT alters brain function in circuits now well established to be related to OCD pathophysiology has the potential to help clarify the nature of the relationship that exists between the *experiential* data that OCD patients so vividly relate concerning their very real discomfort and sense of being 'locked in' to maladaptive behavioural patterns and the types of *effort* required to generate the cerebral changes that occur when these patients show clinical improvement. The observation that cognitive training about biomedical aspects of OCD can enhance a patient's ability to alter behavioural responses to intrusive symptoms reflects the fact that improvements in the cognitive processing of internal information can lead to more adaptive behavioural output. However, as stated above, it is of the utmost importance to clearly realize that no element of this process of systematic change occurs without significant effort and work — and that this requirement for work is directly related to the need to generate energy for the purpose of altering powerfully entrained patterns of cerebral activity which are causally linked to the symptomatic experiences which the OCD sufferer is striving to overcome.

Let us return to our man with OCD and reflect on exactly what may be happening in his brain as he struggles to go and assist his wife as opposed to capitulating to his urge to compulsively wash his hands. In scientific terms, it is uncontroversial to assume that newly forming patterns of cerebral activity must be related to the man's growing awareness that 'a faulty brain message' is generating the bothersome urge he's now struggling with. However, while fully granting that quite plausible assumption, it is also clear that in *experiential* terms a task of immense difficulty now confronts our man: for he now must *actualize* his new awareness in behavioural terms by systematically Refocusing his attention on useful adaptive behaviours. This action, if done regularly, will enable him to alter the patterns of neuronal gating in his caudate in scientifically verifiable ways. Given the well-documented scientific basis for believing that changes in TAN firing patterns are related to changes in caudate gating, it may be useful to frame the man's task as one in which he must change the response contingencies of the TANs in his caudate.

In *experiential* terms, how does he go about completing that task? Let us focus for a moment on the newly forming patterns of brain activity which are beginning to

emerge in association with the enhanced cognitive processing of sensory input that our man, through his considerable effort, has begun to achieve (e.g., 'If I wash my hands just because I saw some axle grease, in the long run it'll only make these OCD urges even more intense.'). Thought processes such as these, when performed regularly, probably have some effect on the gating of messages through the caudate (Graybiel, 1997) and thus could involve changes in TAN cell response patterns to the axle grease, but the process is still very weak and undependable, since the man is still in the early stages of treatment. What sort of process, what kind of *energy input*, will it take to amplify this process sufficiently to systematically alter the cellular firing patterns, very likely genetically mediated from the start and then further established through decades of repetitive responses, generating the intense intrusive feelings of contamination which so persistently afflict our man — and to do it in an entirely drug-free manner in less than ten weeks?

To put it in the plainest of terms, how does he focus his attention on the 'true message' those new and still frail and inchoate circuits are sending to his consciousness ('Go help your wife in the garden. That's what you need to do to get better.') when he is simultaneously being bombarded by intense, powerful and extremely distracting 'false messages' ('You better go wash! That filthy grease might be on your hands!') which are very much still being 'transmitted' by his hyperactive cortical-basal ganglia circuitry? Where does he find the energy to strengthen the 'good message' signal now forming in his cortex and attempting to forge a new 'gating path' via the fragile and still developing new circuitry beginning to arise in his caudate? And, further, how does he now activate motor circuitry which will take him *away from* rather than *towards* the bathroom sink? The momentous nature of this question becomes especially apparent in light of the fact that for many years now movement *towards* the sink, followed by further damaging handwashing, has been our man's habitual motor response to the OCD urge generated by the 'false brain message.' As a result, that particular set of motor responses will itself have a very well-established brain circuitry with it's own well-developed gating and TAN response patterns, and will thus be generating its own associated drives and urges.

So how does the man with OCD begin to stabilize new striatal gating patterns in response to OCD symptoms? The answer to this question, as every good OCD therapist knows, is that he generates the energy necessary to activate, strengthen, and stabilize his new health-giving and life-affirming circuitry, and the new gating patterns associated with it, through the *exertion of his will*, and the *power of his striving* — a power which can generate a real and palpable force which I propose to term **mental force** (Schwartz, 1999). This force is similar in kind to what Lindahl & Århem (1994) (following Popper *et al.*, 1993) have called 'mind as a force field,' and what Libet (1994; 1996) has termed the 'conscious mental field (CMF).' However, consistent with the basic principles of quantum physics described by Stapp (this issue), I specifically take the strongly felt effort which is clearly experienced as a necessary aspect of the onset of the action of this force to accurately reflect the true nature of its etiology — my working hypothesis is that it is a genuine physical force generated by real mental effort. This mental force, associated in our OCD patient's case with the effort he must exert in order to *mindfully direct and focus* his attention on his new 'true message' and *wilfully actualize* the adaptive behaviour towards which it orients him, will functionally amplify his new brain circuitry and enable it to generate more adaptive

patterns of neural gating. This leads to meaningful decreases in the intensity of his intrusive OCD symptoms as well as to improvements in the behavioural and signal interpreting (a.k.a. cognitive) functions with which those newly formed adaptive neural patterns will be associated.

While this newly proposed term, mental force, represents a still largely hypothetical entity, there does seem to be a theoretical need for a force of this kind in nature. The clinical fact that changing one's behaviour in the midst of a barrage of intense and intrusive thoughts and feelings produced by pathological OCD brain circuitry requires a profoundly active process leads naturally to a consideration of the type of mechanism that would be required for such a process to cause the kind of systematic energy use changes those pathological circuits have undergone in the data presented in Fig 2 and Table 2. Forthrightly addressing the question of how that highly targeted effort and those systematic brain changes are related could well lead a reasonable observer to conclude that a mental force, or something very much like it, is necessary if one is to establish a causal relationship between those two quite different sorts of data — and by far the most intuitively clear and satisfying way of viewing how such intense efforts could lead to the kinds of cerebral changes observed is by means of a causal relationship.

Without reference to a naturally occurring mental force, the observed changes in cerebral energy use, which are statistically significant only for those OCD subjects who demonstrate clinically meaningful improvement, would have to be autonomously generated by an entirely *passive* process — but that is plainly inconsistent with a very large amount of clinical data, most especially the verbal reports of patients who have actually undergone these treatments. To precipitously reject such verbal reports as an unimportant or misleading source of data is not only scientifically and methodologically unjustified, it reflects an ad hoc perspective adopted merely to protect a profoundly counter-intuitive way of thinking. The notion of a naturally occurring mental force arises directly as a result of properly attending with an open mind to *all the relevant data* collected in the clinical and basic science studies discussed above. It seems an entirely legitimate means of parsimoniously explaining the data at hand without a need for resorting to reductionist approaches which irrationally prefer materialist as opposed to experiential perspectives.

The Distinction Between Active and Passive Phenomena in Consciousness Research

As was stated at the outset of this paper, the aspect of OCD which renders it a particularly valuable source of information about the mind-brain interface is the so-called 'ego-dystonic' nature of its symptoms — the fact that people who are affected by the condition experience the repetitive intrusive thoughts and urges which comprise the core phenomena of the disorder as unwanted and extraneous intrusions into consciousness. Because of this, OCD symptoms can be viewed as a painfully amplified version of the sorts of desultory and unpredictable mental events that pass through the mental life of essentially all people innumerable times in the course of a day — mental events which are, to a very large degree, passively experienced as essentially arbitrary phenomena not subject to any obvious volitional control. Phenomena which, in effect, pass into and out of consciousness in a quite fleeting and transitory manner.

Of course, in the case of OCD, the very nature of the disease state renders the symptomatic thoughts repetitive and intrusive rather than fleeting and transitory. They also cause significant distress since they are accompanied by a very bothersome sense that 'something is wrong,' a feeling that very much is passively experienced insofar as the person having it is painfully aware that it is false, bothersome, and inappropriate. Not infrequently they experience their behavioural responses to these sensations (e.g., compulsive handwashing) as also having a robotic and disturbingly passive quality. To overcome this disease state, as we have seen, they must take decisive and profoundly active countermeasures, with resulting alterations in their brain's habit generating mechanisms.

This naturally occurring 'amplification effect' on otherwise normal experiences, while pathological and burdensome to those afflicted by it, renders OCD symptoms potentially quite valuable phenomena for modeling several aspects of consciousness in a practical and coherent way. Specifically, the study of OCD can help us address more general questions concerning the crucial difference between passive and active phenomena in conscious experience. The work of the eighteenth century French philosopher Maine de Biran, a seminal figure in the history of modern psychology (Moore, 1970), sheds great light both on these issues and on the kinds of problems which arise in connection with an exclusively 'brain-based' theory of volition. In particular, Biran demonstrates the lack of clarity with which the concept of causation is utilized by philosophers who attempt to reduce volitional phenomena to the realm of material processes.

After three centuries of nearly unchallenged dominance, the position of those philosophers and scientists who uncritically assume that all causal efficacy resides in the material realm has begun to come into question in recent decades, not least due to the advances in the field of quantum physics detailed by Stapp and others in this issue. Some of these recent developments were anticipated by Biran who, in response to Hume's skepticism concerning the nature of causation, observed that our inner experience of a causal relationship between the will to movement and bodily motion is so vivid and direct that it transcends the possibility of reasonable doubt. While honest investigators must acknowledge that Biran's position on the causal efficacy of volition elicits as immediate and intuitive a sense of agreement as any statement concerning causation in the outer world of five sense experience possibly could, it is also an observation whose significance is now further strengthened by the well-documented inadequacy of the classical pre-quantum physics conception of material causation to account for all the data which science must try to explain. Inflexibly clinging to a notion of causation that is both counter-intuitive for conscious phenomena and demonstrably incomplete in the world of material processes can no longer be justified on rational grounds.

The debate on causation between Hume and Biran, and its relevance to current developments in consciousness research, is admirably elucidated in the article on Biran by Philip Hallie in The Encyclopedia of Philosophy:

> According to Maine de Biran, Hume mistakes our *pensées* for our *effort voulu,* confuses disparate outward impressions and their images with intimately related, inwardly simultaneous willing and movement.
>
> Hume's . . . objection is that no connection or 'means' connecting the will to the body is present in willed effort. By 'means' Hume chiefly meant physiological means that can be

> demonstrated through outward impressions and derived hypotheses concerning the connection between the willed effort and bodily movement. Maine de Biran answered, however, that in the face of the plainly felt experience of inward causation, one need not ask for 'connecting' entities deviously derived from a different sort of experience. Hume, in doing so, simply reasserted his old prejudice in favor of outward impressions and their images. . . .[O]ur certainty in experiencing the *effort voulu* lies in this experience itself, not in any hypothetical structures based on quite different experiences (Hallie, 1967).

For those seeking rigorously data based explanations of conscious phenomena there is much to recommend in Biran's perspective. One can only add that if those committed to the materialist position and its many-fold variants would simply acknowledge that the idea of causal efficacy in the material world is both every bit as hypothetical as any account of causation derived from inner observation and *much less directly experienced* (i.e., much less empirically based), then rigidly asserted modes of reductionist explanation (which recent developments in quantum physics have demonstrated to be radically incomplete) would be more amenable to much needed updating and expansion. That simple acknowledgment would also open up the field of consciousness studies to more precise, pragmatic and experientially based explanations, a development that would both increase the light and decrease the considerable heat which frequently accompanies debates on these issues.

Using the subjective experiences of OCD sufferers as a model for more precisely clarifying our intuitive grasp of the critical distinction between active and passive phenomena in conscious experience can enable us to more clearly delineate the pragmatic grounds for positing a causal role for volition in the process of systematic cerebral change. As described above, the OCD patient in early stages of CBT is faced with a situation in which two systems of brain circuitry are competing for dominance in a present moment of real time. One set of circuitry underlies the passively experienced pathological intrusions into consciousness which characterize the phenomenal *feel* of OCD; the other encodes information which can be utilized to enhance one's cognitive awareness of the true significance of those intrusions. The relative strength of these two neural systems is markedly incommensurate if they are compared with respect to either utilization of metabolic energy or acute effects on conscious experience — the pathological circuitry is clearly dominant with respect to both of these variables. However, from the perspective of the patient doing CBT, the most important difference between them lies in their subjective quality, in the *feel* with which they are associated. For the pathological phenomena are experienced largely as passive and disturbing intrusions which if attended to elicit monotonous, robotic, enslaving types of responses, while the capacity to use knowledge as a basis for resisting the symptoms is very much experienced as potentially active in nature — especially insofar as it provides an informational template for the kinds of responses which can provide, with the painstaking exertion of appropriate effort, adaptive alternatives to life as an 'OCD robot.' *It is the critical difference between these two types of feels that makes genuine volitional choice a real possibility.* And, as the data presented in Table 2 and Figure 2 demonstrate, the conscious choice to exert effort in the service of resisting the false messages and misdirected urges of OCD is accompanied over several weeks by systematic changes in the very neural systems which are implicated in the generation of those symptoms.

In the present context the term *mental force* is defined as a naturally occurring force generated by the volitional effort required to refocus one's attention away from the

experiential phenomena of OCD, and associated with the cerebral alterations which are correlated with that attentional refocusing. It is a force which is necessary for the activation of the new patterns of neuronal gating which volitional effort generates, as well as for the stabilization of all the associated adaptive alterations in brain circuitry which occur in concert with the sustained effort required for meaningful behavioural change. Through the regular exertion of directed mental force the reliable operation of what are initially inchoate processes gradually develops, and this accounts for the observed changes in brain function which accompany clinical improvement after the successful application of CBT in OCD sufferers.

A Causal Role for Mindfulness

Because the predominant *feel* of sensory phenomena is essentially passive in nature, the realm of sensory experience (and particularly visual experience) is one in which the analytical power of the scientific method can be imagined to at least potentially provide a genuinely meaningful dissection of so-called 'qualia' of experience into brain-related data points. However, the idea of a similar dissection for the intrinsically *active* feel of volitional phenomena seems lacking in any intuitive basis for what might even be considered face validity. And yet those committed to explanations based in the classical physics of the material world seem unfazed by the profound discrepancies in these two types of data. If nothing else, this provides compelling testimony for the power of 'scientific materialism' to generate a sense of having achieved an adequate explanation in the minds of modern philosophers.

Perhaps the biggest category error committed by philosophers who attempt to advocate a 'scientific' determinism, based on the reduction of volitional phenomena to neuroscience data, is the identification of the conscious elements of our passively experienced thought stream with either the totality of consciousness itself or with its actively experienced volitional component (mistaking 'our *pensées* for our *effort voulu*,' as Biran so insightfully described it). One of the major benefits accruing to philosophers and scientists from the use of the OCD experience as a model is that, through it's 'amplification effect' on normal conscious phenomena, the study of OCD enables us to more clearly discern the difference between the passively experienced thoughts and urges of OCD symptoms and the extremely active nature of the attentional aspect of cognitive-behavioural Relabeling and Refocusing that OCD patients utilize when performing CBT. Cambridge University psychologist John Teasdale, who has independently devised a method for treating depressed patients that has many similarities to the CBT approach described above for OCD (Teasdale *et al.*, 1995), has coined the term 'meta-awareness' to describe the key feature of active attentional processes of this type (Teasdale, 1997). Using a five-point scale to assess the degree of meta-awareness before and after cognitive therapy for depression, his research team rated the extent to which patients report 'a wider perspective on thoughts and feelings as mental events in the field of awareness, rather than as phenomena with which patients identified personally as aspects of themselves.' The subjects who were rated four or above on the scale 'saw their thoughts and feelings in a wider perspective; there was a discrimination of self from thought and feelings.' Depressed patients scored significantly higher on this scale after cognitive therapy compared to a medication only group. As patients' scores on the scale increase they

begin to demonstrate 'a different view and relationship to depressive experience in general: "thoughts and feelings as mental events that can be considered and examined", rather than "thoughts as self-evident facts"'.

This conscious perspective of meta-awareness that Teasdale has been investigating and training patients with depression to actively utilize is identical in its main features to the mental state which has been described above as 'mindfulness' or the perspective of 'the impartial spectator.' Discerning clearly the characteristics which distinguish this type of consciousness from its contrasting mental state, which might appropriately be termed 'unmindful' or 'mere' awareness, is greatly enhanced by training within one of the mindfulness-based traditions of meditation which are common to many of the ancient Asian schools of mental development, particularly those based within the Buddhist tradition. The Pâli canon of the Theravâda school of Buddhist tradition places an especially strong emphasis on mental development of this sort, and includes numerous texts which elaborate on distinctions between various sorts of mental attention. Perhaps the most important of these is between what are termed *yoniso* and *ayoniso manasikâra,* which can be loosely translated as wise (or proper, reasoned, methodical) attention (Nyanatiloka, 1980) and unwise attention. Within the Theravâda tradition, and especially with respect to the teachings of the vipassanâ or insight branch of meditation practice, the key distinction stressed between these two types of attention hinges on the presence of mindfulness or Bare Attention in so-called 'wise attention' and absence of it in the 'unwise' variety (Mahâsi Sayadaw, 1983).

A very helpful further clarification of the intimate relationship between mindfulness/Bare Attention and *yoniso manasikâra* has recently been provided to me in a personal communication from Bhikkhu Bodhi, President of the Buddhist Publication Society, Kandy, Sri Lanka, '*Yoniso manasikâra is* not synonymous with mindfulness, though the two work closely together. *Yoniso manasikâra* is close and careful consideration of things taken up for scrutiny. Mindfulness is what makes these available for consideration, but *yoniso manasikâra* goes beyond 'Bare Attention' and actively examines things in a way intended to bring their real characteristics to light. The product of *yoniso manasikâra* is wisdom (*paññâ*). Thus we might consider *yoniso manasikâra* to be the bridge between *sati* (mindfulness) and *paññâ*.'

It is worth noting a key Pâli scriptural text in which Gotama Buddha, through the use of an elegant simile, clarifies the critical implication that the type of attention utilized in the present has for future states of consciousness:

> Just as this body, monks, is supported by food and stands in dependence on it, stands not without it, — even so monks, the five hindrances [to the development of wisdom] are supported by [their own type of] food, and stand in dependence on it, stand not without it…
>
> And what, monks, is food for the arising of ill will not yet arisen, or food for the increase and growth of ill will that has already arisen? There is the aversive feature of things. Unwise attention frequently applied to that, this is food for the arising of ill will not yet arisen, or food for the increase and growth of ill will that has already arisen. (Saµyutta Nikâya, Pâli Text Society edition, Vol. V, p.64; Translation modified from Woodward, 1930, p. 52.)

(Analogous application of this simile is used in this text for the other four hindrances: sensual lust, sloth & torpor, restlessness & scrupulosity, excessive doubt.)

The key point of this canonical passage is that the *quality of attention* that one places on a sensory or mental object has critical implications for the *types of conscious states* that arise with respect to that object. The particular example I have chosen to quote was selected because of its great relevance to the mental actions performed by a person with OCD when working on the Four Steps of CBT in Table 1. If the man with OCD described in the clinical example above views the aversive feature of the cloth soiled with axle grease with unwise, unmindful, non-methodical attention (i.e., he does not Relabel its dirty appearance as something that elicits his OCD symptoms), the 'ill will' that arises in him with respect to the cloth (e.g., palpable feelings of disgust and a visceral fear that the cloth can contaminate him) will very quickly intensify and become unmanageable, greatly increasing the probability that he will respond with handwashing compulsions. As Rauch's group at Harvard has convincingly demonstrated, this rapid increase in disgust and fear will be accompanied by marked increases in the pathological OCD circuitry contained within the OFC, anterior cingulate, and caudate nucleus.

On the other hand, if our man makes a conscious effort to be mindful and methodically practices the Relabel Step in order to remain aware that the aversive feature of this soiled cloth is nothing but harmless axle grease and that his urge to wash is an OCD symptom that he can Refocus his attention away from, he will in all probability perform an adaptive behaviour instead, such as going to help his wife in the garden. The volitional effort to do so will, through the generation of mental force, amplify and strengthen alternative circuitry developing in his brain which enables him to re-gate his cerebral responses to the aversive sensory stimulus, which will result over time in a growing ease and automaticity in the adaptive behavioural responses he chooses to initiate as an alternative to repetitive handwashing. With several weeks of sustained effort at CBT the man will Revalue the greasy cloth stimulus in a way which will render it non-aversive — i.e., the 'ill will' he harbours towards the cloth will dissipate. There is now significant evidence that alterations of this sort will be followed by measurable changes in his OFC-anterior cingulate-caudate circuitry, and that the application of mindfulness, painstaking volitional effort, and systematic attentional refocusing by the man is necessary for these cerebral changes to take place (see fig. 2).

Conclusion

A reasonable working hypothesis to coherently explain all these data is that the volitional effort and attentional refocusing that occur in conjunction with mindfulness and wise attention at the critical moments of CBT when OCD patients *actively* change their responses to the intensely anxious feelings of obsessive thoughts and compulsive urges generate a *mental force* capable of re-gating entrenched pathological neural circuitry and establishing new and adaptive patterns of neuronal response in their place. The pragmatic clinical conclusion is simply this: with proper training and volitional effort pathological brain circuitry is susceptible to adaptive alteration. To provide that training to real people with real suffering caused by a now reasonably well understood cerebral malfunction requires using a mode of communication which utilizes their entirely natural and empirically validated belief in the efficacy of their own wilful actions — explanations using radically mechanistic terminology derived from theories based on outdated concepts of causation no longer supported by modern

physics (see Stapp, this issue) are entirely impractical and totally inappropriate for explaining to actual OCD patients the steps they must follow for the purpose of systematically changing their own brain chemistry. The clinical practice of modern data-driven behavioural medicine absolutely requires the use of the data of inner experience — *very much including the directly perceived reality of the causal efficacy of volition* — in order to operate in an effective manner. This plain fact carries with it the profoundest implications, for it means that the age-old belief that human beings have in their capacity to act as genuinely self-directed agents capable of instituting real self-directed change can now be rationally justified not just on practical, but also on scientific and philosophical grounds.

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APPENDIX: The Implications of Psychological Treatment Effects on Cerebral Function for the Physics of Mind-Brain Interaction

Henry P. Stapp and Jeffrey M. Schwartz

The data emerging from the clinical and brain studies described above suggest that, in the case of OCD, there are two pertinent brain mechanisms that are distinguishable both in terms of neuro-dynamics and in terms of the conscious experiences that accompany them. These mechanisms can be characterized, on anatomical and perhaps evolutionary grounds, as a lower-level and a higher-level mechanism. The clinical treatment has, when successful, an activating effect on the higher-level mechanism, and a suppressive effect on the lower-level one.

Certain conscious thoughts accompanying the lower-level process are experienced by the subject as intruders into his stream of consciousness: intruders that are not subject to conscious control in a manner commensurate with the rest of his thoughts. On the other hand, the conscious thoughts associated with higher-level process are experienced as integral parts of a stream of consciousness that is able under normal circumstances, with the application of sufficient wilful effort, to exercise control over the course of both bodily and mental actions (e.g., motor responses and attentional focus). Thus these OCD studies exhibit, in sharp relief, two different aspects of the mind-brain connection, one being the effects of the subject's brain upon his thoughts, the other being the effects of his thoughts upon his brain: the OCD studies juxtapose, and relate, exemplars of these two opposite sides of the apparent mind-brain connection.

The key question at issue, in this discussion of volition, is whether the mechanical picture suggested by intrusive OCD symptoms is, in some important sense, the full true picture of the causal structure of the mind-brain connection, or whether, on the other hand, our thoughts and volitions have effects that lie beyond what the brain itself is doing: do our thoughts and volitions themselves really enter into the causal structure in some way that is not fully reducible to mechanical brain processes alone; or is our deep-seated intuition that our thoughts and associated wilful efforts influence our actions an illusion?

This question can be posed at two levels: the pragmatic, and the ontological. At the level of pragmatic clinical practice it appears advantageous to postulate that the causal connection goes both ways: in the clinical treatment the mechanical origin of the intrusive thoughts was emphasized, in order to separate that aspect from the idea of 'self', thereby weakening its power; yet the supra-mechanical power of thoughts and volitions was implicit in the injunctions to resist, by wilful effort, those mechanically generated intrusions.

Certainly, a wholesale abandonment of the notion that our thoughts and volitions have causal efficacy would seriously cripple the sort of communication between subject and therapist that this successful method of treatment depends upon. Yet recognition of the existence of strong mechanical-type effects is also important in pragmatic clinical practice. Thus, within an ideal pragmatic theory of the mind-brain connection, both aspects of the causal connection should be accommodated without contradiction.

If one poses, on the other hand, the ontological question of what really exists then there is again a strong requirement of logical consistency: the entire ontological picture must hang together as a logically coherent whole that must also be compatible with the findings of other branches of science, and in particular with the principles of physics.

Before the OCD studies reported above there already was a major problem at this ontological level. According to the principles of relativistic classical physics, physical reality consists of nothing but a collection of tiny localized realities each causally connected only to its very close neighbours at earlier times. At the ontological level there is, to the extent that relativistic classical physics is valid, no 'emergence' of anything else: one may have good *practical* reasons for wishing to identify various complex structures, or certain approximate properties, and to give them names, but the only realities that are *needed*, dynamically, are the micro-realities defined by the basic physical principles. These microscopic realities and their micro-local connections to close neighbours are all that exist in the fundamental form of modern classical physics: any added elements constitute an epiphenomenal appendage that is causally gratuitous as far as the behaviour of the physical universe is concerned.

A typical moment of conscious experience has a complexity that makes it nonidentical to any one of the basic atomic micro-realities of classical physics: insofar as conscious experience lies within the classical-physics ontology it can *only* be a collection of these ontologically distinct micro-entities; a collection that is in every detail reducible to, and nothing but, this collection, and that has no property that does not follow as a strict logical consequence of the explicitly posited physical properties. Yet the experiential properties of 'greenness' and 'redness' and 'sourness,' for example, are not logically reducible, within the precepts of classical physical theory, to the spatio-temporal properties in terms of which the classical physical principles are formulated. Moreover, the existence of any *physical* reality that can grasp as a whole the macroscopic properties of large collections of the microscopic realities requires an augmentation to the ontology of precisely the kind that classical physics abolished: the very essence of classical physical theory was precisely that it *eliminated* from the physical world all graspings of macroscopic structures as wholes. The basic point of relativistic classical physics was exactly to reduce physical reality to a collection of local properties. Although it is certainly *logically* possible to reinsert now into the ontology some dynamically superfluous macro-entities, that option runs counter both to the core idea of classical physical theory, and also to Occam's razor, which is one of the pillars of good science.

So the question at issue, at the ontological level, is whether, as demanded by the principles of classical physics, all conscious thoughts and volitions are, as regards their connection to brain process, able to do nothing to the physically describable brain that is not done already by that brain and its physical environment alone? Or can a person's conscious thoughts and volitions actually enter into the causal structure in a way concordant with how they seem — subjectively — to act, namely as a force that can focus our thoughts in a way that can oppose and even override, if powered by sufficient volitional effort, the mechanical aspects of brain process?

In the context of this question the main point of this paper is that, whereas it may well be reasonable to postulate that the ego-dystonic elements of an OCD sufferer's experiences — in the form of obsessive thoughts and compulsive urges — have an

epiphenomenal character, postulating the existence of 'epiphenomenal effort' is extremely problematic. It may indeed be *logically possible* for the 'feeling of intense effort' that accompanies the subject's successful overcoming of the lower-level process by the higher-level process to be just a *by-product* of the higher-level brain process rallying the resources needed to overcome the power of the lower-level mechanical process, and hence an after-the-fact, or beside-the-fact, superfluity. But it makes no dynamical sense to have this feeling of intense effort be an epiphenomenal by-product of the needed rallying of resources, rather than a *cause* of this rallying. When the effort flags, and is in danger of failing, more support is needed if the higher-level process is to prevail. Some process that actually rallies support for a course of action that has been assigned great value by the high-level conscious processing would be highly advantageous to the human organism. The intense effort *seems* to do just this. It makes no sense for this feeling to exist if it is a mere passive signal that this needed rallying is already occurring, or has already occurred, rather than being what it seems to be, namely part of the process of making what needs to happen actually happen.

A philosopher might object to this demand for 'sensibleness'. Yet surely it is far preferable, all else being equal, to embrace an ontology that makes sense, rather than one that does not. In a sensible picture of nature there would be no 'effort' that seems to be doing something that needs to be done physically, namely rallying needed resources, but that actually does nothing physical at all. Clearly, a vastly more reasonable alternative is one in which this palpable effort actually contributes to the bringing into being of that which is needed. Why should we believe, without good reason, that nature embraces a senseless ontology rather than a sensible one?

If the sensible possibilities were to conflict with the principles of physics then one might be justified in rejecting them in favour of one that makes no sense. But exactly the opposite is true. The basic principles of physics, as they are now understood, are not the deterministic laws of classical physics. They are laws that determine only probabilities for events to occur: other processes are needed to complete the ontological structure, if some definite sequence of physical events is to be actualized. Moreover, the physical reality now appears to be more like evolving information than like evolving matter (Stapp, this issue). And this 'physical reality' is explicitly tied dynamically into our human experiences by the basic precepts of contemporary physics, as these precepts are actually practiced, and as they were enunciated by the founders. Consequently, these contemporary physical laws can accommodate, in a completely natural way, the property that 'psychological effort' can focus the course of physical brain events in just the way that it seems to do. There is no compulsion from the basic principles of physics that requires any rejection of the sensible idea that mental effort can actually do what it seems to do: namely keep in focus a stream of consciousness that would otherwise become quickly defocused as a consequence of the Heisenberg uncertainty principle, and keep it focused in a way that tends to actualize potentialities that are in accord with consciously selected ends (see Stapp, this issue). Mental effort can, within contemporary physical theory, have, via the effects of the wilful focus of attention, large dynamical consequences that are not automatic consequences of physically describable brain mechanisms acting alone.