Understanding Hysterical Conversion

An Interdisciplinary Approach

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Contents

Summary

Poor hysterics. First they were treated as victims of sexual trouble ... then of moral perversity and mediocrity ... then of imagination.

William James, 1896

According to current psychiatric classificatory systems, 'conversion hysteria' or in its present clinical incarnation, 'conversion disorder', refers to neurological deficits such as paralysis or somatosensory losses, for which an organic medical explanation cannot be found, and for which a psychological rather than organic cause is presumed.

The assumption of psychogenesis on the basis of the absence of disease is misleading. It assumes that if there is a lack of significant structural damage in organ systems implied by the complaints of the patient, the problem must therefore be 'all in the mind'.

Yet explanations are rising in number in terms of the biological, especially neural, and endocrine regulatory processes that underlie the subjective experience of chronic physical symptoms. These processes *are* organic despite the fact that there is no evidence of damage or disease to either the brain and central nervous system, or peripheral organ systems.

The simplistic reduction of complex systemic processes to the psyche or imagination can no longer be sustained in light of such evidence. Current biopsychosocial models of hysteria, and somatoform disorders in general (disorders that take the form of 'real' diseases) explain the somatic phenomena observed in terms of innate defense reactions to threat, seen throughout the animal kingdom.

This conceptualization is not new. In 1926 Kretschmer suggested that conversion reactions were related to the instinct for self-preservation and likened the motor and sensory diperceptions, and in humans, mental entities, such as memories, might activate affective systems, resulting in physiological disturbances that are mistakenly interpreted by the patient as symptomatic of disease. In the present work, I shall argue that to understand the systems and processes involved in creating the illusion of illness is to understand the problem of hysteria.

However, as things stand, a lack of biomedical explanation for such gross abnormalities has led many practitioners to believe that the patient who claims to be ill in the absence of disease, is either attempting to deceive them or is self-deceived. In other words, many practitioners do not accept that the patient's condition is beyond her control. The problem of hysteria, on this view, will not be resolved by explaining symptoms; rather, what needs to be explained is why some individuals, for various motives, continue to insist they are ill despite medical findings to the contrary. Medical practitioners readily admit that they do not like dealing with 'somatizers', setting the scene for irritation and unsatisfactory interactions. As often observed, the patient's condition may worsen as a result of the doctor-patient relationship.

It is the view of the author, that education is the key to resolving both problems. Patient and doctor require conceptual models that will allow them to understand how neurotransmitters and hormones secreted in response to a stressor (physical, psychological, or social) can, under certain circumstances, alter bodily representations creating sensory and motor disturbances. Such models can also explain why it is that normally transient reactions to threat can become chronic in some individuals and the mechanisms involved. To answer these questions requires knowledge of the principles of associative learning (classical and operant conditioning).

Hysterical symptoms will continue to be misunderstood and their sufferers maligned until both practitioner and patient understand the inextricable interconnections between body, mind and the social environment suggested by emergent biopsychosocial models.

To understand the issues and controversies that abound in studies on hysteria, philosophers also need to acquaint themselves with the different aspects of what is a complex phenomenon. The following represents one such attempt.

Declaration

I certify that this thesis does not incorporate without acknowledgement any material previously submitted for a degree or diploma in any university; and that to the best of my knowledge and belief it does not contain any material previously published or written by another person except where due reference is made in the text.

Patricia Albrecht

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1 Introduction

Each year in general practitioners' surgeries around Australia, thousands of patients present with symptoms which, investigations reveal, have no apparent physical cause. The spectrum of complaints ranges from minor symptoms such as headaches, digestive disturbances, lethargy and non-specific aches and pains, through to visual problems, seizures, paralysis, and severe pain. Millions of health dollars and thousands of hours are spent on sophisticated medical tests (CT scans, MRIs, X-rays, blood tests) for illnesses that appear to be all in the patient's imagination (Jureidini & Taylor, 2002). In some cases it will later be shown that the patient was actually suffering from the early symptoms of disease. However, for a significant number of patients, medical science can provide no physical explanation for their condition.

Symptoms that cannot be medically/organically explained are generally diagnosed as 'somatoform', meaning, as the term implies, that they have the form of a somatic disorder (Mace, 2001). 'Hysteria', or in its present incarnation, 'conversion disorder' (I shall use the terms interchangeably throughout the present work), is listed under this umbrella term in the current Diagnostic and Statistical Manual (DSM-IV, 1994). 'Conversion disorder' refers to a disturbance of body function that is characterized by neurological, sensory, or motor symptoms, which mimic those of organic disease (Kozlowska, 2005).

This lack of a biomedical explanation has limited progress in the understanding of such disorders (Jureidini & Taylor, 2002). However, scientific explanations are rising in number in terms of the biological, especially neural and endocrine regulatory processes that underlie the experience of enduring physical symptoms without significant structural damage in organ systems (Henningsen, 2003; Kozlowska, 2005; Damasio, 2003). On this emerging organismic view of conversion disorder (and possibly other somatoform disorders) the symptoms

presented are not indicative of disease but nor are they the products of the patient's imagination (Chalder, 2001; Henningsen, 2003).

Philosophically, these contemporary theories challenge the Cartesian separation of body and mind which has underpinned medical conceptualizations of hysteria over the last four centuries. Conversion symptoms cannot be neatly placed on one side or the other of the Cartesian divide. Mind-body dualism has led to biological theories of hysteria *or* psychological theories disembodied from underlying neurobiological processes (Kozlowska, 2007). In light of recent advances in neuroscience and neuroendocrinology, the existing dualistic medical model is no longer considered heuristic by a number of practitioners. As Wells (1996:126) says: '... a clear demarcation no longer exists between the purely functional and purely organic'.

Kozlowska (2005) has called on her colleagues to acquaint themselves with these current applications of neuroscience to the problem of conversion disorder. On her view, in order to understand conversion reactions, '... clinicians require conceptual models that expand the conventional dualistic mind-body paradigm, integrate multiple theoretical perspectives, and take into account environmental factors that shape evolutionary and individual development' (Kozlowska, 2005:1). The bodily symptoms reported by patients often become meaningful when understood in terms of the individual's developmental history, helping therapists to define such disturbances in less self-deprecating ways than is currently the practice. As Noyes, et al., (2008:14) writes, 'Consistent with biomedical theory and practice, they are viewed as non-legitimate disturbances for which patients themselves are responsible.'

The assumption of psychogenesis that pervades existing conceptions of hysteria is, in part, a product of mind-body dualism. However, Freudian psychoanalysis, which dominated 20th century understanding of conversion symptoms, significantly reinforced Cartesian thinking, despite the fact that Freud was no

dualist. The purpose of this thesis is to examine existing conceptual models of hysteria as they have evolved since Sigmund Freud (1856-1939) coined the term 'conversion' to describe the process by which unacceptable mental contents were transformed into somatic symptoms. The question I seek to answer is whether the conventional dualistic paradigm underpinning the medical model can be expanded to incorporate these new conceptual models as Kozlowska (2005) urges, or will it require what Kuhn (1962) referred to as a 'paradigm shift'—a whole new system of concepts and approaches congruent with the organismic world-view?

1.1 The concept of conversion

In Chapter Two I begin this examination with Freud's concept of conversion. Although the term 'conversion' used in the context of hysteria, did not originate with Freud, it is generally to the Freudian construct that most students turn when tracing the historical roots of the disorder. In late 19th century Europe, the number of females presenting with hysterical symptoms reached almost epidemic proportions. Freud began his study of the neuroses with the problem of hysteria. He hypothesized that the sensory anaesthesias and motor paralyses characteristic of the disorder did not have neurological origins but rather expressed a way of defending against unacceptable unconscious thoughts.

According to Freud, the fundamental feature of hysteria is the presence of massive psychological repression or inhibition of the unacceptable thought (usually of a sexual nature) and the development of a symptom pattern that indirectly or symbolically expresses the individual's repressed needs and wishes. Freud first presented his concept of conversion in *Neuropsychoses of Defense* (1894):

> In hysteria, the incompatible idea is rendered innocuous by its sum of excitation being transformed into something somatic. For this I would like to propose the name of conversion (Freud, 1894:49).

It should be noted that here hysteria is not identified with any specific symptoms

beyond an understanding that these are somatic and the consequence of nervous action. In the 19th century hysteria was a heterogeneous term used in association with not only anaesthesias and paralyses but also diverse entities such a hypochondriasis, anxiety, mild depression, eating disorders, vomiting and heart palpitations (Breuer & Freud, 1895). The idea of a quantitative conversion of 'psychic' energy into a physical disorder is generally thought by commentators *not* to be maintained in later dynamic formulations. In Chapter One I show why this common conception is incorrect. I agree with Kitcher (1995) that on technical grounds Freud did not—could not, in fact—retreat from the idea of a quantitative conversion involving neurophysiological processes despite appearances to the contrary.

Freud's claim that 'hysteria behaves as though anatomy did not exist' (Freud, 1893:29) and his advice to students of psychoanalysis to 'resist temptation to flirt with endocrinology and the autonomic nervous system' (Freud, 1927/1959:257), were an open acknowledgement of the complex nature of hysteria and the futility of pursuing a neurobiological explanation given the current state of ignorance on such matters. As Freud and his colleague Breuer state in their seminal work *Studies in Hysteria* (1895/2004:250):

> How far we still are today from the possibility of any such complete understanding of hysteria! With what uncertain strokes the contours have been outlined, with what crude hypotheses the yawning gaps have been covered up rather than filled in. Only one consideration is of some comfort—that all *physiological representations* of complicated psychical processes are flawed in this way... (italics added).

Freud never intended to deny that 'functional' disorders such as paralysis and language loss had pathophysiological correlates (Marshall, 2001). Rather, in his later works such physico-chemical processes are assumed.

Current neurobiological models of hysteria cogently, if not comprehensively as yet, fill in many of the gaps in our knowledge regarding the neural systems and

processes responsible for creating abnormal (hysterical) responses to perceived threat that we call 'fear'. As Breuer and Freud (1985/2004) argued in the *Studies*, in the pathogenesis of hysteria it makes no difference whether the exciting event is physical injury or some other aversive experience such as being insulted, the effective cause is not the injury or the insult, but the affect or emotion of fright—'*the psychical trauma*' (p9).

Although Freud tended to use the term 'anxiety' rather than 'fear' in later works, fear and anxiety are closely related. As Le Doux (1998) points out, both are reactions to dangerous or potentially dangerous situations. However, anxiety is usually distinguished from fear by the lack of an external stimulus that elicits the reaction. Fear comes from the outside world—anxiety comes from within us or as Breuer and Freud stated, it is a 'psychical trauma'. For example, the sight of a snake or an angry person wielding a knife may elicit fear, but the memory of some unpleasant experience with a snake or an angry person wielding a knife or, the anticipation that you may encounter these stimuli, are conditions of anxiety (Le Doux, 1998).

A number of scientists who study human fear and anxiety, including nonspecific anxiety, agree that it is most likely that the same underlying brain system is involved in the generation of such feeling states (Le Doux, 1998; Damasio, 1994). If correct, this implies that hysterical reactions (symptoms) may differ only phenotypically from other anxiety disorders such as panic attacks, phobias, and post-traumatic stress disorder (PTSD).

The most thorough understanding of fear behaviour has come from studies of fear conditioning in infrahumans as well as humans. Although considered controversial and incomplete, as we shall see, the learning principles identified by early behaviourists are now considered to be fundamental in our understanding of conversion disorders.

1.2 Hysterical symptoms as behavioural responses to threat

The conditioning theory of neurotic disorders arose in the 1920s at a time when psychologists were beginning to explain most aspects of behaviour in terms of learning experiences and particularly in terms of Pavlov's conditioned reflexes. On this early view, phobias, anxiety disorders, and other so-called 'neuroses', including the abnormal reflexes characteristic of hysteria, could be explained in terms of fear conditioning. According to John Watson, the 'father' of behaviourism, neuroses arose as a result of traumatic learning situations and then persisted and influenced behaviour throughout life (Watson, 1926).

By the 1930s, Watson's theory of the neuroses was augmented by another form of learning called instrumental conditioning which had become equally important to behaviourists. Applied to neurotic behaviour, the theory provided an alternative, more complex, explanation of why a conditioned fear response might endure other than through a simple stimulus-response association. Instrumental conditioning theory introduced the notion of 'reinforcement' into our understanding of neurotic behaviour. The response (the symptom in the case of hysteria) may become habitual because it is instrumental in reducing anxiety (learned fear). For example, a child being bullied at school may be so terrified that his legs tremble and give way. His mother, thinking he is sick, keeps him away from school, reducing the probability of harm and concomitant feelings of fear. The symptom (of distress) is reinforced because it is successful in removing the feeling of fear. As Dollard and Miller (1950:62) put it:

> The phobias, inhibitions, avoidances, compulsions, rationalizations, and psychosomatic symptoms of the neurotic are experienced as a nuisance by him and by all who have to deal with him... When a successful symptom occurs it is reinforced because it reduces neurotic misery. The symptom is thus learned as a habit. Any behaviour conducive to avoiding the threat of harm also reduces anxiety.

Mischel (1976) calls this early conception of how neurotic behaviour develops and is maintained 'psychodynamic behaviourism' because, like Freud, proponents

of this view identified the reduction of anxiety as the primary motivational force underpinning the neuroses.

In contrast to psychodynamic behaviourists who saw Freud as scientifically imprecise but basically on the right track, the radical behaviourism of Skinner (1953) and his like-minded colleagues discarded mentalistic entities such as feelings of fear, anxiety and the like in their explanation of how behaviour, including neurotic behaviour, is learned. On this view, a 'successful symptom' or behaviour is simply one that has been reinforced by the environment. If a partial paralysis is successful in removing the threatening stimulus it is reinforced and thus more likely to occur in the context of the conditioned stimulus—period.

In Chapters Three and Four, I reexamine the tenets of behaviourism as they relate to neurotic behaviour introducing insights from neuroscience and cognitive science regarding the brain systems that underlie fear conditioning. It can be shown that much of the psychopathological behaviour Freud observed is explained in a more parsimonious way by learning theory rather than by appealing to a psychological unconscious. So-called hysterical 'symptoms' may emerge as part of an implicitly learned response to threat which, in turn, is mobilized automatically and therefore experienced as involuntary.

Although older children and adults are able to reflect on their behaviour and to change many unwanted habits learned through instrumental conditioning, in classical conditioning there is no voluntary or instrumental response that the organism can make to influence the pairing (Rolls, 1999). This is why anxiety disorders are generally considered intractable and often experienced throughout the individual's life-span, peaking and waning in severity depending on the relative presence or absence of stressors (Le Doux, 1998).

It is important to note then, conversion symptoms, characterized by neurological, sensory, or motor symptoms, are not simply the result of instrumental

conditioning. They are components of innate, universal reactions to threat observed in infrahumans and humans alike and involve universal physiological adjustments (Le Doux, 1998). The response that is perhaps most relevant to our understanding of hysteria (narrowly defined as symptoms affecting voluntary and motor and sensory functions) is often called the 'freeze response' (immobilization) which normally precedes 'fight or flight' in the presence of perceived danger. However, when flight or fight is not an option, a widespread inhibition of movement and vocalization may occur (Kretschmer, 1926; Nijenhuis, et al., 1998) as seen in children and adults diagnosed with conversion disorder (Kozlowska, 2007).

Kretschmer (1926) suggested that hysterical symptoms were related to the instinct for self-preservation and had their phylogentic roots in the immobilization reflex. In the manner of early behaviourists, he hypothesized that the continued association of a particular behaviour with relief from danger or fear would result in a 'smoothing out' of the behaviour until it became habitual and automatic.

Thus, as I argue in Chapter Three, neurotic behaviours in general do not seem to demonstrate instrumental (operant) conditioned random behaviours as Skinner proposed, rather, they constitute innate preorganized dispositions to threat. While the display of these universal defenses may take many different forms depending on the species, the circumstances under which threat takes place, and, in humans, the culture into which the person is socialized, the freeze response is remarkably similar in all animals.

1.3 The opioid-mediated freeze response

Most people are conversant with the 'fight or flight' response, and to some degree may have knowledge of the associated biophysiological adjustments that we self-conscious beings equate with feelings of 'anger' or 'fear', e.g., increased heart-rate, sweating, and other visceral symptoms associated with increased levels of hormones such as adrenaline and noradrenaline. It is also a well-known fact that

extreme threat can induce the freeze response affecting the ability to move our limbs or even to speak if sufficiently terrified. Less well-known perhaps, is that the body's natural analgesics, endogenous opioids, are responsible for such freezing behaviour. Like their synthetic morphine analogues these peptides bind to specific receptors in the brain effectively filtering out normal body signals. Perhaps the most familiar effect of opioids is stress-induced analgesia which occurs in burn victims or those who suffer other horrific injuries.

Damasio (2003:115) suggests that ' ... some so-called hysterical or conversion reactions that allow patients not to feel or move parts of their body, may be the result of ... modifications in the central body maps.' According to a number of contemporary models of conversion disorder, symptoms reflect inaccurate, implicit representations of body state/sensory data. Since this data provides the substrate for the subjective evaluation of body state, such errors, when perceived as symptoms, may cause the person to believe she is suffering the effects of neurological disease (Damasio, 2003; Vuilleumier et al., 2001, 2005; Henningsen, 2003).

It should be stressed that the opioid-mediated freezing response is a reaction to noradrenergic activity elicited by the extreme trauma either of a physical or psychological nature. Endogenous opioids decrease central noradrenergic activity and their activation thus inhibits hyperarousal (Perry, 2001), which might otherwise result in behaviour which draws the attention of a predator, increasing the risk of capture and possibly death.

In humans, opioid mediated hypoarousal states and noradrenergic hyperarousal states may be manifested as disorders of inhibition and anxiety disorders respectively. Such disorders are most likely to occur in infants and young children lacking a familiar caregiver who modulates physiologic arousal by providing a balance between soothing and stimulation (van der Kolk, 1989; Shore, 2003). In particular, childhood abuse and neglect may cause a long-term vulnerability to

suffer hyperarousal or hypoarousal with the individual experiencing the distressing effects of both extreme states at different times depending on the context. Variables such as gender (Perry, 2001) and the person's early learning history (Crittenden, 2006) appear to influence whether the individual tends to respond to life stressors in ways characteristic of hyperarousal or hypoarousal.

Thus, in addition to various physical, emotional and mental states associated with the more traditional symptoms of hysteria (disorders of inhibition) we should perhaps also include signs of hyperarousal (anxiety disorders) in the category of phenomena classified under conversion disorder. As we shall see in our examination of the *Studies* (1895/2004), hyperarousal states such as heart palpitations, hyperventilation, and other phenomena characteristic of anxiety, were considered more minor features of hysteria. A number of modern clinicians also claim that anxiety disorders, behavioural disorders such as Attention Deficit Disorder (ADD), and the general inability to modulate affective behaviour are signs of hyperarousal (Perry, 2001; Shore, 2001). It may turn out to be the case that many phenotypically different signs and symptoms presently classified under various psychiatric disorders, are actually due to affect dysregulation caused by early childhood trauma.

While Freud confined his later works on the neuroses to the analysis of psychological trauma and repression, modern neuroscientific findings suggest that conscious feelings of rejection by conspecifics utilize the same neurobiological systems implicated in physical trauma. In other words, the same systems that are engaged in response to extreme physical pain, also appear to react to psychological pain (Mac Donald & Leary, 2005; Panksepp, 2003; Eisenberger, et al., 2003). This observation is consistent with notion that in evolution more complex processes arise out of the simple or, as Damasio puts it, there is 'a nesting of the simple within the complex' (Damasio, 2003:37). More will be said on this subject in Chapter Four.

Thus, current neurobiological theories of conversion disorder that suggest symptoms are part of the organism's biological and behavioural response to threat were preceded by 20th century thinkers from various fields of endeavour. However, their explanations were less complete given the relative absence of knowledge regarding the neurobiological correlates of such disorders, particularly the effects of peptides (e.g., hormones and neurotransmitters) on regions of the brain involved in representing body states. If, through further investigation, these theories are shown to be correct, they would explain not only hitherto biomedically unexplained symptoms in patients currently diagnosed with somatoform disorders, importantly they would provide a way for both doctor and patient to understand the etiology of this class of complaints.

1.4 The limitations of psychological theories of hysteria

The failure of some patients to accept that the source of their problem is psychological rather than organic, would be understandable on this view. However, for many physicians this refusal has become the central problem defining hysteria (Taylor, 1989; Jureidini & Taylor, 2002). In other words, understanding the physiology of hysterical disorders is not as 'crucial' to our understanding of hysteria as how and why:

> ... for a variety of motives, some people transiently or chronically ... lay claim to physical ailments, impairments and disorders which they do not have, and for which they are prepared at times, to manufacture the evidence (Taylor, 1989:391).

On this view, hysteria is at best a form of self-deception (Turner, 2001) or at worst, constitutes an attempt on behalf of the patient to 'fool' or deceive the doctor (Taylor, 1989). In the literature hysterics are variously described as individuals who are held in the sway of a false belief (Taylor, 1989); pretenders who do not know they are pretending (Jureidini & Taylor, 2002), or dissemblers who do not know they are dissembling (Halligan et al., 2001). The underlying assumption common to these views is that the patient is ignoring 'normal' bodily signals for a variety of motives (Jureidini & Taylor, 2002).

The above assumption is challenged by contemporary models of conversion disorder yet is so deeply entrenched in the minds of some orthodox clinicians that such novel, albeit plausible explanations, are not generally discussed in psychiatric journals. Healthy skepticism is a good thing. These new models have yet to be tested rigorously in the context of hysteria. However, in a recent review, Charlton (1995) attacked psychiatry for failing to contribute significantly to either the practical or theoretical understanding of major functional disorders such as hysteria. The main problem identified relates to the lack of a coherent, theoretically motivated neurophysiological account concerning the pathophysiology of such disorders (Anthwal, et al., 2001).

Results from recent attempts to map the neural correlates of hysteria using functional brain imaging appear to have met with the same disregard from the psychiatric fraternity as neurobiological models of conversion disorder. According to Anthwal and his colleagues:

> Given the desire to provide a principled account of hysterical symptoms capable of linking a putative abnormal psychological state with the production of physical symptoms, there is a surprising dearth of relevant neuropsychological or anatomico-physiological reports in the literature (Anthwal, et al., 2001:217).

Similarly, Spence (2000) has criticized the failure of psychiatrists to address contemporary developments in cognitive neuroscience which, as he sees it, renders existing theories on hysteria and its management increasingly anachronistic.

1.5 Neuroimaging hysteria

Neuroimaging is yet another step towards constructing a scientific theory of hysteria, placing less reliance on the judgment of the physician as to whether or not the patient is feigning his or her symptoms. For example, one way of excluding conscious intention may be to observe whether a region of the brain that appears to be uniquely associated with the subjective experience of deciding

when to act and which action to perform (Spence & Frith, 1999) is activated when the patient is asked to move, for example, a 'paralysed' limb. This region of the brain is the left hemisphere dorsolateral prefrontal cortex (LHDLPFC) also found to be activated in memory suppression (Anderson, et al., 2006). In support of this measure, it was shown that when subjects were asked to feign paralysis, LH frontal regions were activated including the more dorsolateral areas (Spence, 2000).

Conversion symptoms, on the other hand, appear to reflect the operation of brain systems outside of conscious awareness that are responsible for calculating the reward/punishment value of a stimulus, or the costs and benefits associated with goals for action (Rolls, 1999). If this is correct, then those systems identified as integral to this process should be activated when patients attempt to move an affected limb. Recall that according to learning theory, it is likely that with such patients, the threat elicited freezing response has become implicitly learned (through the principles of classical conditioning). The systems known to be involved in this type of computation are the amygdala and, particularly in primates, the orbitofrontal cortex (OFC) (Rolls, 1999). This region of the prefrontal cortex is also known to be integral to the inhibition of an inappropriate action, that is, an action which is not rewarded or one that is associated with punishment (Le Doux, 1998). Whereas the LH appears to be involved in the conscious memory of a threatening experience, the right hemisphere (RH) is known to be implicated in the learning and memory of the physiological responses threatening stimuli (Le Doux, 1998).

In Chapter Five, I examine various neuroimaging studies carried out on individuals diagnosed with conversion disorder within the context of existing neurobiological knowledge. The results tend to support the above predictions based on what is reliably known about the function of brain regions implicated in hysteria. Although the number of neuroimaging studies are few in number and sample sizes are small, there is converging evidence to support the claim that

hysterical symptoms are correlated with hypoactivation of the LHDLPFC and a corresponding activation of the RHOFC (Marshall et al., 1997; Spence, 2000). However, as I point out in this chapter, there are methodological problems that preclude any definite conclusions regarding a neural 'fingerprint' for hysteria at this stage.

Despite such problems, researchers seem to be quietly confident that a neurphysiological account of hysteria is possible in the not too distant future. This account is likely to be consistent with the more general theory that conversion symptoms are learned emotional reactions to threat. As Vuilleumier (2005:309) reports from his review of the current research, findings point to symptoms being the result of:

> ... a modulation of sensorimotor representations by primary affective or stress-related factors, involving primitive reflex mechanisms of protection and alertness that are partly independent of conscious control, and mediated by dynamic modulatory interactions between limbic and sensorimotor networks.

Here Vuilleumier uses the term 'limbic' in a general sense to include the orbitofrontal cortex, amygdala and cingulate cortex (often referred to collectively as the 'rostral limbic system' (Shore, 2003; Rolls, 1999; Anthwal et al., 2001) responsible for affective evaluations based on current as well as past experience (Vuilleumier, 2005). Lesion studies graphically demonstrate the importance of the ventral or orbitofrontal regions in the choice of appropriate decisions and actions based on past learning (Damasio, 1994, 1999; Rolls, 1999).

Thus, a picture is beginning to emerge of the neurobiological correlates of hysteria which as Vuilleumier (2005:323) states:

> ... may help reassure the patients as well as their caretakers, including nurses and doctors who sometimes show negative or unsympathetic reactions when confronted with complaints unaccompanied by visible organic pathology.

1.6 Emotion regulation and hysteria

If conversion symptoms are part of the emotional response to threat, then emotion regulation strategies that serve to reduce the individual's reactions to life stressors may protect him or her from developing stress-related somatoform disorders as well as anxiety disorders. Emotion regulation theorists show that some strategies commonly used by people to mitigate negative emotional responses are more conducive to health and well-being than others (Gross, 1998a, 1998b; John & Gross, 2004; Richards & Gross, 2000; Oschner et al., 2004). In Chapter Six I examine different emotion regulation strategies within the framework of a general process model which holds that different strategies can be differentiated along the timeline of the unfolding emotional response, a theory at the heart of the past work of a number of emotion theorists (e.g., Arnold, 1960; Ekman, 1972; Frijda, 1986).

On this view, an emotion begins with an evaluation of emotion cues. When attended to and evaluated in certain ways, emotion cues trigger a coordinated set of response tendencies that involve experiential, behavioural, and physiological systems. Once these response tendencies arise, they may be modulated in various ways. Because emotion unfolds over time, emotion regulation strategies can be distinguished in terms of when they have their primary impact on the emotion-generative process (John & Gross, 2004).

Curiously, because repression is understood to occur *before* the emotion response tendencies have become fully activated, that is unconsciously or implicitly, some theorists believe that this strategy may promote resilience to developing various anxiety-related disorders (Coifman et al., 2007; Tomarken & Davidson, 1994). This position contrasts to that taken by Freud who saw repression as a precursor to hysteria—no repression, no hysteria (Freud, 1896). According to Freud's theory, the effect of repressing emotion-laden thoughts was an increase in arousal leading possibly to hysterical symptoms if repression could not be maintained.

I examine this current conceptualization of repression in light of anatomical

evidence which shows that people classified as 'repressors' are, in fact, highly aroused despite verbal self-reports to the contrary (Weinberger, 1990; Weinberger & Schwartz, 1990; Shedlar, et al., 1993; Tomarken & Davidson, 1994). I show from studies on childhood conversion disorder that the most likely interpretation of the data is that people identified as repressors are consciously suppressing their negative emotions. Following therapeutic intervention, children tend to reveal their true feelings and thoughts previously concealed from parents (and initially from their therapists) for fear of angering an abusive caregiver. (See Appendix One for a rare, first person account of the experiences of one adolescent, 'Trish', recounted in Kozlowska, 2003).

From this brief overview it would appear that hysteria, as it involves the medical profession, might best be explained as abnormal (exaggerated), automatic, physiologic, and behavioural responses to threat. Would this stance help eradicate the stigma attached to the condition and the problem of why patients often insist they are suffering from an organic illness as Vuilleumier (2005) predicts? Is it possible to achieve the desired consonance between the patient's subjective experience and the doctor's objective professional opinion based on medical evidence? If not, why not?

1.7 The mechanistic versus organismic approach to understanding hysteria

One significant reason for thinking that a reconciliation is not likely to happen any time soon is that, as pointed out, most doctors continue to subscribe to the practice of classifying organically unexplained symptoms as 'psychological', consistent with the dualist foundations of medical science. As Jureidini & Taylor (2002:123) put it: 'Hysteria is one serious psychiatric disorder that might indeed be ... all in the mind ... The problem must therefore be psychological ...'. The implication is clear. If the symptom is not physical and thus 'real' it must be psychological and thus not 'real' (Chalder, 2001). Whether or not we acknowledge the merit of conceptualizing hysterical symptoms as animal defense reactions, it is obvious from contemporary neuroscientific and endocrinologic

findings that this dichotomy is false. Physico-chemical processes can actually modify or change mental representations and *vice versa,* resulting in illusions or false beliefs about the body states (Ramachandran & Blakeslee, 1999). In Chapter Seven I use the phenomenon of false pregnancy to illustrate how this interactive process might account for some cases of adult hysteria where the patient falsely believes that she is physically ill.

I also examine how the mechanistic paradigm and mind-body dualism has resulted in the acknowledged poor and often hostile relationship between patients diagnosed with somatoform disorders and their doctors. As things stand at present, the conceptual constraints characteristic of the organic versus functional/psychological view in medicine has created an impasse which cannot be resolved (Jureidini & Taylor, 2002).

Kozlowska (2007) suggests that the way forward lies in reconceptualizing conversion disorders as innate self-protective strategies elicited in the context of threat initially from hostile, inadequate or unavailable caregivers. In her recent paper entitled *The Developmental Origins of Conversion Disorders* (2007), she adopts an organismic paradigm—attachment theory, as a framework for understanding how conversion reactions develop and may persist into adulthood without appropriate clinical intervention. The remainder of Chapter Seven is devoted to examining attachment and interpersonal communication in the context of somatoform disorders.

1.8 Attachment theory and somatization

Attachment theory, as the name implies, seeks to describe and explain interpersonal relationships beginning with the relationship formed between primary adult caregivers and the infant in their care. A core belief of attachment theorists is that the human infant has a need for a secure relationship with her caregivers without which normal social and emotional development will not occur (Bowlby, 1973, 1980). Thus, so-called conversion symptoms in adults are likely

to have their origins in dysfunctional family relationships that threaten the child's safety and result in insecure attachment (Kozlowska, 2007; Noyes, et al., 2008; Stuart & Noyes, 1999).

Evidence for this view has been accumulating over recent years (Taylor, et al., 2000; Ciechanowski, et al., 2002; Schmidt, et al., 2002; Waldinger, et al., 2006; Stuart &Noyes, 1999; Noyes, et al., 2008; Kozlowska, 2007). Both clinical impression and investigation point to an association of distinctive attachment styles with different reactions to threat currently classified as separate disorders in the DSM-IV. However, according to recent neurobiological models of conversion disorders which define such reactions as defense strategies, the habitual expression of a particular strategy in the context of dysfunctional family-attachment relationships, is activated by one and the same defensive system discussed in Chapters Three and Four (Le Doux, 1998). What distinguishes these different reactions that currently constitute and define somatoform disorders and their sub-types, is the different kinds of attachment relationships formed during socialization.

The two self-protective responses to external threat that Kozlowska (2007) sees as being relevant to our understanding of conversion disorders is the freeze response, already discussed, and appeasement defense behaviours that signal submission to an angry conspecific. Unlike the freeze response, appeasement, particularly in humans, can take many forms depending on individual socialization experiences. Appeasement displays are shaped by operant conditioning and reflect those responses that the child has learned will be rewarded by parents or are instrumental in avoiding punishment. Because illness or injury is often a means of securing attention, comfort and protection or functions to mitigate anger in a caregiver, some anxious individuals use illness behaviours as a means to such ends.

Consistent with the above view, these two different responses are primed, and

come to be expressed as conversion symptoms in the context of different developmental experiences. The freeze response involving motor and sensory inhibition, including the inhibition of negative affect, is observed by Kozlowska (2007) to occur in children raised by parents who tend to use harsh punishment to enforce control. The attachment style developed here is referred to as 'compulsive compliance' and can lead to disorders of inhibition (Crittenden, 2006) where the individual overly relies on reward/punishment contingencies to guide her behaviour. Submission, evidenced by appeasement displays in the form of illness behaviour is more likely to be observed in the context of chaotic family environments where parents are unpredictable in their responses to the child's behaviour. Without the ability to predict aversive outcomes and thus avoid punishment, children can suffer high anxiety (Mischel, 1976). Using illness to regulate parental behaviour is a learned self-protective strategy .

Although the enactment of appeasement displays in the form of illness behaviour might appear to be deliberately manipulative and this may well be the case once the child has become consciously aware of his acquired ability to meet his needs in this way, such behaviour is initially implicitly learned. As Kozlowska (2007) says:

> In the context of significant threat, the behavioural and somatosensory components of emotional responses can be activated automatically, and children can present with unwanted and medically unexplained pain or neurological symptoms (either motor or sensory in character) ... Although increasing maturity allows older children and adolescents to reflect on and modify many aspects of their behaviour, the neural activity underpinning many behavioural repertoires remains implicit, and patterns of behaviour that were established before the availability of explicit memory systems may continue to be triggered automatically without being subjected to reflection (Kozlowska, 2007:488-493).

The organization of attachment behaviours (or strategies) involves the principles of associative learning identified by behaviourists. Thus, there is no need to appeal to higher cognitive systems such as those required for explicit deception or self-deception (if it exists) when explaining the etiology of hysteria. Feigning neurological symptoms or other illness behaviour does not technically fit under

the umbrella of somatoform disorders which on Kozlowska's view reflect involuntary disturbances of body function or 'implicit deception' respectively.

Conceptualizing hysteria as arising from two distinct implicit responses to threat has significant implications for paediatric and adult clinical practice. The most obvious implications of this distinction for intervention are: (a) in the case of children who evidence components of the freeze response the clinician has a duty of care to report children whom she believes are at risk from very hostile parents; (b) in the case of children who have learned to manage threat by using illness as a means of avoiding punishment or gaining attention, both child and parents will need to learn to modify their behaviour. For example, by increasing parental consistency and ensuring the child is not rewarded for displays of illness behaviours. At the same time, efforts should be made to decrease levels of anxiety and unpredictability in the family system (Kozlowska, 2007).

I conclude that converging opinion based on scientific evidence derived from many different sources, points to the fact that psychological formulations of hysteria disembodied from underlying neurobiological processes can no longer be sustained. An organismic paradigm such as attachment theory is considered heuristic in the case of somatoform disorders in particular, but may also serve as a useful explanatory tool for understanding other so-called 'psychiatric' problems as Crittenden (2006) suggests. I say 'so-called psychiatric problems' because once we understand how such disorders develop, and comprehend their adaptive significance, it becomes apparent that their amelioration can only be achieved by changing the dysfunctional family/social environment in which such complaints have their origins.

2 The theory of conversion hysteria

The first years of the 1890s saw the publication of Freud's major formulations of the neuroses mainly in collaboration with his mentor and colleague, Josef Breuer. It was during this time of intense thought on the etiology of nervous disorders and in particular hysteria, that Freud developed many of the constructs which formed the bases for his theory of mind and the therapeutic method of treating the troubled mind—psycho-analysis.

In this chapter I examine the theory of hysterical conversion put forward by Breuer and Freud in their seminal work *Studies in Hysteria* (1895). It is to the *Studies* that most people turn to find the theoretical origins of hysterical conversion, or, as it is commonly called today 'conversion disorder'. This work explicitly reveals the neurological assumptions underpinning the theory of conversion hysteria. Post-1895, these assumptions became increasingly implied in Freud's writings as he realized that his intention to 'furnish a psychology that shall be a natural science' (Freud, 1895/1954:295), would not be achievable during his life-time. However, Freud did not abandon his neurological theory as is commonly suggested (Merskey, 2001). Rather, it remained, as the editors of the Standard Edition state, an 'invisible ghost [which] haunts the whole series of Freud's theoretical writings to the very end' (Strachey, 1962:290).

Relative to Freud's later works, it can be said that the theory of conversion put forward in the *Studies* reveals the importance of 19th century neurology to Freud's reasoning on the etiology of symptoms observed in his patients. The idea that the neuroses in general, and hysteria in particular, have their origins in emotions such as 'fright, anxiety, shame or psychical pain', such as that suffered by an insult (Breuer & Freud, 1895/2004:9), is congruent with current neurobiological theories of hysteria. So, too, is the notion that abnormal arousal states are involved in hysteria. However, Freud's use of the language of

electrophysiology conveys the presumption of a simple, direct correspondence between neural and psychological events consistent with 19th century opinion.

With advances in neurology, the electrophysiological model of the brain and nervous system was shown to be incorrect, however, Freud did not modify or change his theory of conversion despite the fact that it was predicated on increasingly obsolete notions. As we shall see, the complexity of brain systems and processes interposed between input (stimuli) and output (responses) renders the idea of a direct correspondence between ideation and somatic phenomena, and thus the concept of conversion, false. I argue that the concept of conversion has no explanatory power and should therefore be removed from the psychiatric lexicon.

2.1 Conversion hysteria – an outline

The main theoretical position adopted by Breuer and Freud in the *Studies* seems, on the surface, a simple one. They hold that any experience which gives rise to distressing affects or emotions increases intracerebral 'excitations' or arousal. (p9) In the normal course of things, this 'quantity of nerve force' or 'psychical energy' is either 'abreacted' (discharged) in a variety of conscious reflex actions such as running away, crying, screaming, acts of physical retaliation and so forth. Even if the quanta of energy is not totally abreacted, the memory of say a hurtful statement is corrected in association with other thoughts, such as considering one's own worth, so that generally, the person succeeds in causing the affect to diminish or disappear. In the case of hysteria, none of these things happen because the memory of the emotional experience is cut-off or 'dissociated' from consciousness.

Two principal reasons for the dissociation of ideas are proposed by the authors. One is that the memory is incompatible with the person's self-concept and thus had to be 'defended' against by conscious suppression resulting in the dissociation of the mental entity from the stream of consciousness. This was

Freud's preferred hypothesis. The other, argued for by Breuer, was that the memory was formed and invested with affect while the person was in an altered state of mind he called 'hypnoid'. Both hypotheses assumed the clinical necessity of abreacting the quantity of energy attached to emotional memories. It follows, on this view, that because such dissociated memories have not been divested of their affect through conscious processing or in action, their full quota of nervous energy is 'converted' or transformed into an 'abnormal' (hysterical) reflex rather than a normal reflex. This pathogenic result is explained by the fact that a primary function of the nervous system is to keep the quantity of intercerebral excitation or level of arousal, 'constant' (the constancy principle) which, in turn, had its conceptual foundation in the more general observation that organisms tend to operate in accordance with homeostatic principles (Breuer & Freud, 1895/2004).

In a nutshell, we could say that according to Breuer and Freud, just as the memory of a traumatic learning experience no less than the experience itself, can elicit normal somatic and behavioural responses (e.g. crying) so, in some people, under certain circumstances, such experiences and memories can elicit abnormal (hysterical) responses. As it is famously stated in the *Studies*: '*hysterics suffer for the most part from reminiscences*' (p222, italics in original).

2.2. Sexual hysteria

However, even in the opening paragraph of their preliminary comments, the authors are open to the possibility that instead of the traumatic memory being dissociated, the patient may simply find the experience too unpleasant or painful to talk about. As Breuer writes, the 'fended off' mental entity may not be a memory of a traumatic experience at all, but an idea with sexual content (p7). As Freud was to later claim in a letter to his friend and colleague, Fleiss: '...the psychical structures which in hysteria are affected by repression are not in reality memories—but impulses...' (S.E. pp247/248). This view had already been made clear in the *Studies*.

Although Breuer thought it was 'self-evident... that the non-sexual affects of fright, anxiety or anger lead to the development of hysterical phenomena...the sexual factor is by far the most important and, in terms of pathology, the most fruitful...'(p246). The patient's concern with sexual issues was not seen by the authors as the result of any moral weakness in their patients as some practitioners had suggested. On the contrary, both Breuer and Freud observed that 'sexual' hysteria was to be found more in those with 'great moral purity [who] perceive everything sexual as irreconciliable with their moral substance, as soiling and sullying'.(p246) Breuer goes on to state: 'The sexual needs of hysterics vary just as much from person to person and are no more powerful than in healthy people. Hysterics, however, fall ill from them and, for the most part, precisely by fighting them, that is, by fending off sexuality'. (p247)

Subjects considered socially taboo for persons of refined sensibilities, such as masturbation, sexual promiscuity, and rape within marriage, were all freely discussed in the *Studies* and pursued by Freud in later works as he came to see the repression of such instincts to be the main cause of hyperarousal and hence hysteria. By placing such emphasis on sexual hysteria, Freud was to antagonize many of his more conservative colleagues. As Rivers (1920:164) wrote of Freud and those who adhered to his tenets:

> [They] have become so engrossed with the cruder side of sexual life that their works might often be taken for contributions to pornography rather than to medicine. In some of Freud's followers this absorption in the sexual has gone to such lengths that perverse tendencies and prurient ideas are sensed in every thought, waking or sleeping, of the patients who came under their care.

Yet the repression of female sexuality, particularly among the Victorian middle classes, was a historic fact and likely played a significant role in the etiology of hysteria among Breuer and Freud's predominantly female patients. As Breuer comments in the *Studies* (with a degree of insight and empathy not often found in the Victorian male mind), 'I do not think I am exaggerating when I assert that the great majority of severe neuroses in women have their origin in the marriage bed.'

(p246) And, as he adds in a footnote to this remark: 'This is certainly a subject in which the acquired knowledge of experienced physicians should be communicated to their juniors, who, as a rule, blindly overlook sexuality—at all events so far as their patients are concerned.' (p246) So while there might be many different causes of 'intracerebral excitation', sexual matters were considered by Breuer and Freud to be at the heart of the neuroses and therefore feminine hysteria.

Whilst the authors emphasized sexual repression as a probable cause of anxiety and hence hysteria in the female population, according to Rivers (1920), the mass of the medical profession reacted to the suggestion in ways typical of the prudery and timidity displayed by Victorian culture toward such delicate matters:

> The mistake which is now being made by many is to regard this excess as a necessary part of the Freudian scheme instead of an unfortunate excrescence, probably due in large measure to the social environment in which the theory had its origin. There are even those who are so obsessed by the sexual aspect of Freud's psychology [sic] that they regard sexuality as its basic principle and have fallen into a state of mind which wholly blinds them to its merits (Rivers, 1920:164).

As a medical officer serving in the army during World War I, Rivers had the opportunity to witness, first-hand, effects of fear-induced hyperarousal in enlisted soldiers producing, on an enormous scale, hysterical phenomena such as paralysis and blindness. Rivers suggested that although the Freudian theory of sexual hysteria might be applicable to female civilians, in cases arising out of the war experience, matters of a sexual nature did not appear to play a relevant role in the onset of symptoms:

> We now have abundant evidence that those forms of paralysis and contracture, phobia and obsession, which are regarded by Freud and his disciples as pre-eminently the result of suppressed sexual tendencies, occur freely in persons whose sexual life seems to be wholly normal and commonplace, who seem to have been unusually free from those sexual repressions which are so frequent in modern civilization, especially among the more leisured classes of the community... The point is that while we have over and over again abundant evidence that pathological nervous and mental states are due, it would seem, directly to the strains and shocks of

> warfare, there is, in my experience, singularly little evidence to show that, even indirectly...a part has been taken in the process of causation by conflicts arising out of the activity of suppressed sexual complexes (Rivers, 1920:166).

Rivers is obviously referring to Freud's theory as it evolved post-1895, where the connection between traumatic hysteria and ideogenic hysteria if not denied is noticeably absent and emphasis is placed on the theory of repression in the etiology of symptoms. As pointed out in section 1.1, Freud explicitly states in the *Studies* that any experience which gives rise to distressing affects can be considered a psychical trauma capable of resulting in hysterical symptoms. There was never any intention to deny that conversion disorders had pathophysiological correlates (Marshall, et al., 2001; Kitcher, 1995).

2.3 Hysteria and heredity

According to Breuer, while there may be many precipitating causes of hysteria, the basis or foundation of the condition is 'an idiosyncrasy of the entire nervous system'.(p245) However, while the nervous system is adversely affected in all cases of hysteria, on the view presented in the *Studies*, somatic phenomena which have their inception in the sexual instincts or emotion-laden ideas, should be distinguished from symptoms that are a consequence of an innate abnormality of the nervous system. (p245) The stigmata that often attend a general excitability of the nervous system are not caused by ideas but an innate predisposition.

What does it mean to be innately predisposed to hysteria? As we have seen in section 2.2, unlike many of their colleagues, Breuer and Freud believed that fundamentally, hysteria was a neurophysiological problem rather than a case of moral or intellectual inferiority. They observed that in some patients, there is an inherited disposition for the nervous system to be in a constant state of abnormal excitability, so that the condition was seen to affect several members of a family. Excessive excitability was observed to manifest in traits such as 'liveliness', 'creativity', and a certain restlessness that could lead to boredom if constrained or

to generalized anxiety depending on the person's situation. On the authors' view such individuals have a need to discharge excessive electrical activity in physical or mental activities in order to avoid developing symptoms. As Breuer puts it:

> Their liveliness and restlessness, their craving for sensations and mental activity, their intolerance of monotony and boredom, may be formulated thus: they are among those people whose nervous system while it is at rest liberates excess excitation which requires to be made use of (p240).

Another personality characteristic which the authors believed had its inception in this abnormal feature of the nervous system, was the tendency to become intensely focused on a part of the body that may have been previously injured or diseased and to become obsessed by the condition. The person's preoccupation with the affected part had the effect of magnifying the pain experienced and could produce great distress:

> The free floating excitation is, as it were, diverted into this path, and a local hyperalgia is produced. As a result, every pain, however caused, reaches maximum intensity, every ailment is 'fearful' and 'unbearable' (p241).

Unlike so-called 'normal' people, in whom the level of excitability returns to an optimum 'zone' following an emotional episode, in this type of patient:

> ...the quantity of excitation not only remains behind but is constantly increased by the influx of fresh excitations. A slight injury to a joint thus leads to arthralgia, and the painful sensations due to ovarian swelling lead to chronic ovarian neuralgia; and since the nervous apparatuses of the circulation are more accessible to cerebral influence than in normal people, we find nervous palpitations of the heart, a tendency to fainting, proneness to excessive blushing and turning pale, and so on. (p241)

On this view, such somatic phenomena are not caused by ideas but are a direct consequence of the excitability of the nervous system. Thus, despite the fact that the basis of all hysterical symptoms were seen by the authors as an idioscyncracy of the whole nervous system, it is argued that ideogenic phenomena 'are simply conversions of affective excitation' or 'an abnormal expression of the emotions'. (p245)

However, without any objective evidence short of an observed 'excitability', it is

difficult to see how Breuer and Freud could discern between patients whose symptoms were the result of a so-called 'innate disposition' and those whose symptoms were psychogenic. The distinction was later dropped by Freud in favour of a purely ideogenic or psychological causation. Freud's adoption of a unicausal hypothesis contradicted a central argument put forward in the *Studies*, namely, that hysteria has many different precipitants: 'it is impossible to place ideogenesis at the center of hysteria as it is sometimes done nowadays'. (p245) Freud, in particular, maintained that hysteria is always 'overdetermined' in the sense that 'several partial traumas' of a diverse nature rather that one major trauma, occurred prior to a hysterical episode.(p9)

Nevertheless, whatever the precipitating cause or causes of hysteria, on the view promoted in the *Studies*, the resulting excess intracerebral excitation is the basis of hysterical phenomena. This is a fundamental tenet assumed throughout Freud's later works. When cerebral excitation flows into systems which are normally accessible only to the peripheral or autonomic nervous system (ANS) they affect the viscera normally protected from central nervous system (CNS) projections by strong resistances, producing various symptoms in organ systems.

The important point to note is that in patients suffering from ideogenic hysteria there were deemed to be no changes in the nervous system, unlike patients whose symptoms derived from an innate, abnormally excitable, nervous system. Once this distinction was discarded by Freud, the term 'hysterical' became, for many practitioners, a synonym for 'functional' (as opposed to organic). Functional disorders, on this view, have their origins in the person's mind or imagination rather than biophysical processes and thus are not 'real' despite experiences to the contrary.

2.4 Innate versus ideogenic hysteria

With ideogenic hysteria the symptoms 'arise as the consequence of affects in people of a hysterical disposition and are at first simply an abnormal expression

of the emotions'. (p245) Through repetition, the apparently physical stigmata become perceived by the patient as real, purely somatic symptom. Yet as Breuer puzzled:

> If a physical symptom is caused by an idea and repeatedly released by it, one would expect that intelligent patients, capable of self-observation would be conscious of this connection, that they would know from experience that the somatic phenomenon occurs at the same time as the memory of a specific process...Sometimes patients really observe the connection and are conscious of it. A woman may say, for example, that her mild hysterical attack (shaking and palpitations, say) stems from some great emotional upset and recurs only when an event reminds her of it. But for very many hysterical symptoms, probably the majority of them in fact, this is not the case. Even intelligent patients do not know that their symptoms come on as a consequence of an idea and take them to be independent physical phenomena. If this were not so, the psychical theory of hysteria would already have reached a venerable old age (Breuer and Freud, 1895/2004:221-222).

Why were these clinicians so convinced that there was no organic reason for the patient's symptoms? Breuer reasoned that had there been any actual damage to the nervous system by the repeated activation of distressing memories or ideas, then such changes would have given rise to 'genuinely somatic symptoms' that were 'self-sufficient' or 'independent of the ideas which set them going'. (p221) However, he and Freud had found that, much to their surprise, all kinds of hysterical symptoms, some which had persisted for years:

> *...immediately and permanently disappeared when we had succeeded in bringing clearly to light the memory of the event by which it was provoked and in arousing its accompanying affect, and when the patient had described that event in the greatest possible detail and had put the affect into words.* (p6, italics in original).

The eradication of symptoms through the cathartic method described, convinced these investigators that in cases of psychogenic hysteria, the symptoms were not 'real' or 'organic'; a view that persists to this day among orthodox clinicians (Chalder, 2001).

Contemporary practitioners would sympathize with this observed conundrum. The organic-functional dichotomy remains a stumbling block to the understanding of medically unexplained symptoms. As we have seen (section 1.5), some have even equated the problem of hysteria with the patient's belief that their symptoms are caused by disease rather than the product of distressing memories or ideas. However, I shall argue that these are two different problems. If we are interested in explaining symptoms we need to examine what happens neurophysiologically when a person experiences distress. If we want to know why a person in distress erroneously believes she is suffering from an organic disease that requires a different examination—one that focuses on higher cognitive processes involved in inference and interpretation.

Breuer and Freud were more interested in explaining symptoms but were hampered in their interpretation by a lack of knowledge regarding the stress response and the role of endogenous chemical messengers (hormones and neurotransmitters) in symptom formation. When a person is under stress, these chemicals are produced in greater quantities than normal and can produce side effects which may be misinterpreted as symptoms of disease. I will expand on this point in section 2.8.

Through learning (by association) symptoms may take on the properties of habitual or conditioned behaviours, manifested when certain stimuli are present. This will be a recurring theme throughout the present thesis. There does not need to be any structural damage to the brain or nervous system for the experience of enduring physical symptoms to occur in hysteria nor in the organs, tissues or cells of the affected body part or parts (Henningsen, 2003; Damasio, 2003; Kozlowska, 2005). Medical research into the physiological effects of stress is yielding answers to the paradox of unexplained symptoms in terms of biological, especially neural and endocrine regulatory processes.

2.5 The cathartic method

If the somatic phenomena characteristic of hysteria are symptoms of distress which are either maintained by a person's environment or through the repetition of unwanted mental representations, it follows that anything which reduces a person's stress levels will not only reduce the level of neural excitation but also the level of stress hormones circulating in the blood-stream. As I shall show in the next section, these endogenous chemicals produced in response to threatening stimuli, bind to neuron receptors throughout the brain and body causing abnormal or so-called 'hysterical' symptoms if the stress is intense or chronic. The success of the cathartic method practiced by psychoanalysts may be simply due to a reduction in the patient's stress levels as suggested by Breuer in the *Studies*:

> There is a normal and appropriate reaction to the excitation engendered by very vivid and irreconcilable ideas – to communicate them by speaking. We find a comically exaggerated version of the urge to do this in the story of Midas's barber, who spoke his secret aloud to the reeds; we find it too, as one of the fundamental principles of a great historical institution – the Catholic confessional. Communication affords relief, it discharges tension even when it is not made to a priest or followed by absolution. If excitation finds that this way out is blocked, it will sometimes be converted, as is the excitation of traumatic affects, into a somatic phenomenon...(p212)

Perhaps we can understand why then one of Breuer's patients, the famous 'Anna O.' called this cathartic method, the 'talking cure'.

We are told by the authors that as cerebral excitations were 'leveled out' through the cathartic process, hysterical symptoms of the most various kinds 'immediately and permanently disappeared' (p221). These symptoms included:

> Neuralgias and anaesthesias of very various kinds, many of which had persisted for years, contractures and paralyses, hysterical attacks and epileptoid convulsions...petit mal and disorders in the nature of tic, chronic vomiting and anorexia...various forms of disturbance of vision, constantly recurrent visual hallucinations, etc. (p4)

In other words, symptoms disappear with the lowering of intracerebral arousal, which, in turn, is caused by a variety of emotionally distressing memories or ideas

that exist outside the patient's conscious awareness. It is the release of a quantum of energy corresponding to the repressed idea that effects the cure by restoring balance to an overburdened nervous system.

It is now possible to see how deeply reliant Freud was on the tenability of 19th century neurological theory and the notion of a quantitative conversion of affective ideas. Had he abandoned the quantitative theory of conversion hysteria as most commentators erroneously believe (see Merskey, 2001), there would have been no physiological explanation for the symptoms, as Kitcher (1995) points out:

> Although psychoanalysis was a complex theory with many detailed and intertwined claims, this hypothesis was a keystone...the theory of the neuroses assumed energy needing discharge. This energy was supposed to be the...explanation for the tics, paralyses, and other bodily symptoms of neuroses. If the nervous system did not strive to discharge energy, then psychoanalysis had no explanation for these phenomena and they were supposed to be it core explanada (Kitcher, 1995:156/157).

It is one thing to say that Freud abandoned any attempt to provide a scientific account of hysteria when he realized neurology would not, in his life-time at least, provide the sought after details and concepts necessary to achieve this end. It is quite another to suppose that he relinquished the belief that it should be possible to state the facts of psychology, including abnormal psychology, in neurological terms. Although Freud tended to emphasize the psychological or qualitative aspect of hysteria post-1896, the quantitative dimension was always assumed (Strachey, 1962; Marshall, et al., 2001).

For example, he explicitly states in *Beyond the Pleasure Principle*, published in 1920: 'The mental apparatus endeavours to keep the quantity of excitation present in it as low as possible or at least to keep it constant' (SD 18:9). And, in *An Outline of Psycho-Analysis* (1938) Freud obviously presupposes that his readers are familiar with his quantitative theory of conversion (see S.E. XVIII:26-27, XXIII:146).

Just as Breuer and Freud reasoned that hysteria has its inception in the emotions

and emotional arousal, 21st century researchers are also turning to the neurobiology of emotions in order to understand symptom formation in hysteria (Henningsen, 2003; Halligan & David, 2001; Halligan, et al 2000; Damasio, 2003. For reviews see Vuilleumier, 2004; Broom, 2004; Kozlowska, 2005). In the following section I will show that many of the basic physiological assumptions informing Breuer and Freud's theory are currently maintained but explained within the framework of modern neuroscience.

2.6 Hysteria as a problem of self-regulation

It is evident to anyone who reads the *Studies* that, for the authors, the problem of hysteria is fundamentally linked to the concept of self-regulation or homeostasis. As they saw it, the somatic phenomena characteristic of this condition can be attributed to the operation of the nervous system as it attempts to maintain a certain equilibrium of energy conducive to good mental health. Like many of their colleagues, the authors held the view that all organisms contained a finite amount of energy which is kept constant by means of the same homeostatic controls responsible for maintaining other internal conditions, like temperature and the concentration of bodily fluids. It was a small conceptual step from this view to the postulation that the nervous system functioned according to the 'constancy' principle. This assumption, couched in the language of electrophysiology, is revealed in the following passage excerpted from the *Studies*:

> ...there is an optimum for the height of the intracerebral tonic excitation. At that level of tonic excitation the brain is accessible to all external stimuli, the reflexes are facilitated, though only to the extent of normal reflex activity, and the store of ideas is capable of being aroused and open to association in the mutual relation between individual ideas which corresponds to a clear and reasonable state of mind. It is in this state that the organism is best prepared for work (Breuer and Freud 1895:198).

Breuer argued that just as there is an optimum for bodily functions outside of which physical processes deteriorate, so too cognitive performance is adversely affected when the level of cerebral excitation exceeds an optimal level. For example, a person may become verbally incoherent when overwhelmed by emotions indicating, according to the above view, that cerebral excitations are

interfering with the normal ability to order one's thoughts. It is hypothetically such excessive excitations which lead to hysterical phenomena or abnormal reflexes. Hyperarousal detrimentally affect both body and mind.

What evidence is there for the idea that the psyche is a homeostatic-seeking device and that excessive excitation or arousal not only affects cognitive performance but may result in abnormal somatic states? Cortical arousal can easily be demonstrated by placing electrodes on the scalp of a human or infrahuman. These electrodes pick up electrical activity of cortical cells through the skull and are printed out on an electroencephalogram or EEG monitor. When the cortex is at rest patterns of cortical activity are slow and rhythmic but fast and out of synchronization during arousal. (Electroencephalogram power is simply the amount of electrical energy being produced per unit of time—Springer and Deutsch, 1993).

Nearly one hundred years ago psychologists Yerkes and Dodson (1908) suggested that the relationship between arousal and performance could be depicted as an inverted 'U'. When arousal is extremely high due to fear or some other intense emotion, performance deteriorates because the individual becomes overwhelmed and disorganized. Thus performance was believed to be of the highest quality when arousal was at a moderate level. Under these conditions the person is attentive and alert but not swamped (Mischel, 1976). As Le Doux (1998) states, although arousal is important in all mental functions, contributing significantly to cognitive functions such as attention, perception, memory, and problem-solving, we need to have just the right level of activation to perform optimally.

While there are many stimuli, both positive and negative, capable of inducing a state of hyperarousal, Breuer and Freud focused on threatening events or the memory of such threat. Whilst in this state the nervous system is 'hyperaesthetic' or overly sensitive toward sensory stimuli which 'quickly become distressing'. Under normal circumstances, the quanta of electrical energy is discharged or

abreacted through direct reflex actions such as fighting the enemy or fleeing from the source of threat. However, such primitive release is not always possible nor is it encouraged by civilized society. We are constrained by law from acts of physical retaliation and often accused of cowardice if we flee from our enemies.

Contemporary neuroscience confirms these observations. When arousal occurs, cells in the cortex, and in the thalamic regions which supply the cortex with its major inputs, become more sensitive (Le Doux, 1998:289). While much of the cortex is potentially hypersensitive to inputs during arousal, the systems that are processing information are able to make the most use of this effect. This means that non-specific arousal systems can have very specific effects. For example, if the amygdala, known to be involved in the processing of emotional stimuli, is triggered by the sight of a snake say, then the neurons in this region are going to be especially affected by arousal. Other neurons that are inactive at this point do not reap the benefit (Le Doux, 1998:288).

The non-uniform effects of arousal were noted by Breuer and Freud, who also acknowledged the benefits of arousal 'for some situations and purposes' (such as confronting harmful stimuli). However, they were more concerned with the detrimental effects of chronically high levels of arousal on the nervous system and how the systems responsible for regulation might reduce such levels, or what might happen in the event that they fail. Hysterical symptoms, on their view, were the result of an overburdened nervous system seeking to regain a level of equilibrium or constancy. This hypothesis was consistent with 19th century neurology which assumed that the central nervous system functioned as a modified reflex, expelling energy when possible and storing it only to permit the more efficient discharge of incoming energy (Kitcher, 1995).

2.7 The reflex theory of cerebral function

The use of the language of electrophysiology throughout the *Studies* conveys the presumption of simple, direct, correspondences between neural and psychological

events. As we have seen, this position was consistent with the more general view held by 19th century neurology. In the old paradigm the body was seen in terms of energy and matter. Hardwired reflexes caused by electrical stimulation across the synapse ran the body in a more or less mechanical reactive fashion with little room for flexibility, change or intelligence.

In 1833, Marshall Hall had introduced the term 'reflex' to describe a basic principle of action in animals that connected sensory impressions to motor activity. However, whereas Hall had limited such reflex actions to the spinal cord system, a number of German investigators argued that the reflex concept should be extended to include cerebral reflexes, thus opening the way for a unified concept of the reflex as the basic unit of neural action (Kitcher, 1995).

It was plausible then, for Breuer and Freud to hypothesize that excessive intracerebral excitation might be the direct cause of abnormal reflexes. For example, as we have seen, they believed that a case of 'mild hysteria' in the form of heart palpitations, was caused by excessively strong cerebral excitation that had 'by-passed' or 'broken-through' the normal resistances that exist between 'the organ of ideation' (the CNS), and (ANS), affecting the heart. This simplistic notion of how stimuli, whether external sensory stimuli or internal 'ideas', influence or change bodily processes is shown to be incorrect by modern neuroscience.

From the time Freud collaborated with Breuer to write the *Studies* until the end of his career as a psychologist, he accepted the veracity of the reflex theory of nervous function rather than treating the embryonic model as a working hypothesis. He seemed to assume that the basic overall picture was correct and did not concern himself when the paradigm changed irrevocably with the finding that self-regulation was not accomplished directly through electrophysiological processes but indirectly through complex cycles of feedback and electrochemical adjustment.

Freud would have been in his fifties when the first peptide 'secretin' was found during the early years of the 20th century. Secretin was shown to act on an animal's small intestine to stimulate the secretion of pancreatic juices. This astounded physiologists who up until that time had assumed that all physiological functions were controlled by electrical impulses from the nerves (Pert, 2003). Freud was hardly too old or ill, at this point in time, to explore the implications of this finding for his theory of hysteria, but as Kitcher says: '...once he had built nineteenth-century neurophysiology into the foundations of his theory, he eschewed further physiological investigations that might have permitted midcourse corrections' (Kitcher, 1995:163).

2.8 The neuroendocrinology of hysterical disorders

While many physiological processes and behaviours are automatically controlled and may thus appear to be reflexive, in complex organisms such processes and behaviours are more than just spontaneous or reflexive. Importantly, for our purposes, the nervous system does not simply function in a way analogous to an electrical system as Freud and his contemporaries envisaged and it does not become 'overexcited' by stimuli necessitating discharge of electrical energy.

Instead a stimulus or change in the external environment may be of sufficient strength to initiate an impulse. Again, contrary to 19th century thought on the subject, an impulse is not an electrical current and unlike an electrical current the strength of the impulse is always the same (Rolls, 1999). At the synaptic cleft or synapse, chemicals known as neurotransmitters are released and flow across the synaptic gap. Neurotransmitters are used by one neuron to signal another. The molecules of the transmitter diffuse across the gap and attach themselves to dedicated receptors on the membrane of the neuron receiving the impulse and another impulse begins in the second cell (Le Doux, 1998). Thus, the transmission of information within the brain and nervous system and any resulting physiological and behavioural change is accomplished electrochemically not

simply electrically.

Some endogenous chemicals or peptides such as adrenaline and noradrenaline play a dual role in the brain's communication with the body and *vice versa*. That is, they act not only as neurotransmitters causing the next cell in line (typically another neuron) to do something different, but instead may percolate into the bloodstream affecting events far and wide. When adrenaline enters the bloodstream it is acting in the role of a hormone (Sapolsky, 1998). (Adrenaline would have been responsible for the heart palpitations experienced by the patient diagnosed with conversion hysteria referred to by Breuer and Freud (section 2.3). Adrenaline and other neurotransmitters secreted by the hypothalamus during a stressful experience also act on the brain and can lead to states of hyperarousal which in turn affects learning, memory and other cognitive processes (section 2.5).

On this revised view, any aversive stimulus that activates affective systems in the brain, including negatively valenced memories and ideas, is potentially capable of eliciting the stress response, flooding the body and brain with chemicals that prepare the organism for 'fight or flight'. Chronic stress can thus lead to a state of chronic hyperarousal whereby the brain and nervous system is rendered more sensitive to incoming stimuli causing the person to be more easily shocked or startled.

2.9 Stress, hypoarousal and passive defense

So far, we have only considered how an external event, the memory of that event or an unwanted idea, might result in the stress response mediated by peptides such as adrenaline and noradrenaline which, in turn, could be responsible for some somatic states identified with hysteria. With the discovery of the opiate receptor in the late 1970s many other hitherto puzzling somatic phenomena associated with hysteria also began to make sense. Hysteria was, and still is, predominantly characterized by sensory losses, for example, analgesia ("Sometimes my body, or

part of it, is insensitive to pain"); kinesthetic anesthesias ("Sometimes it is as if my body or part of it has disappeared"); and motor inhibitions ("Sometimes I am paralyzed for a while") (Nijenhuis, 2000).

It is well documented that during times of extreme physical pain, endorphins, the organism's natural opioids, are released from the pituitary gland under the regulatory influence of the hypothalamus. These endogenous opioid peptides are structurally very similar to synthetic analgesics such as morphine and heroin. Importantly, this class of peptides has also been shown to be released during psychological or emotional stress (Sapolsky, 1998) and may have a profound influence on the way a person perceives not only their body states but also their mental states (Damasio, 2003; Perry, 2001). In Chapter Four we will examine in more detail how this process might occur.

When produced in response to chronic or acute stress endorphins can have a 'numbing' effect that may be experienced physically by the distressed individual as a sensory disturbance or loss of feeling in a limb or mentally as an 'indifference' to the stressful events occurring around him. Chronic patterns of submission, helplessness, inability to set boundaries, feelings of inadequacy, automatic obedience, and repetition of the victim role are just some of the psychological manifestations identified with such states of hypoarousal or what some clinicians refer to as 'passive defence' in which a person does not actively defend against danger (Van der Kolk, et al., 1989; Perry, 2001). As we shall see, these states are most often observed in people who perceive they cannot escape a stressful situation and may be envisaged as an alternative stress response to that of the more familiar 'fight or flight' syndrome. As Taylor (1989) sees it, hysterical somatic phenomena that mimic symptoms of neurological disease are characteristic of the animal defense behaviour 'playing possum' or the 'freeze' response.

Although Freud emphasized the effects of hyperarousal elicited by anxiety-

producing thoughts, according to Trupp (2000), Freud was to speculate that under extreme and sustained psychological distress, the brain may produce its own anaesthetizing chemicals and did not rule out the possible significance of neurochemicals playing a role in the symptoms of the hysteria. He also thought that future chemical manipulations of human emotion might prove as efficacious in a manner quite different from both hypnotic suggestion and his own (later) psychoanalytic interventions (Trupp, 2000). In these predictions Trupp (2000) thinks it likely that Freud's early experimentation with cocaine played some part.

Traumatized or distressed people may exhibit a propensity to alternate between states characteristic of hyperarousal and those associated with hypoarousal, or reside primarily above or below the parameters of the optimal arousal zone (Van der Kolk, 1989) displaying symptoms of anxiety disorders or disorders of inhibition respectively (Crittenden, 2006). On my view, many of the various symptoms currently classified as 'psychological' can be attributed to states of hyperarousal and hypoarousal caused by stressful life events on the one hand, and, on the other, an inability of the hypothalamus to restore equilibrium. By introducing the concept of hypoarousal and identifying the systems responsible for mediating abnormal arousal states in general, I show that it is possible to gain a more comprehensive view of the processes Breuer and Freud believed underpinned hysterical or abnormal 'expressions of the emotions'.

2.10 Implicit information processing

With advancements in neurology, the reflex theory has been discarded as an explanation for cerebral function above that of simple stimulus-response behaviours. Damasio (1994) tells us that as organisms acquired greater complexity, brain-caused actions required more intermediate processing between stimulus and response. In between the brain's five main sensory input sectors and three main output sectors, lie the association cortex, the basal ganglia, the thalamus, the limbic system cortices and limbic nuclei, the brain stem and cerebellum. Together this 'organ of information and governance' holds both

innate and acquired knowledge about the body proper, the outside world, and the brain itself as it interacts with these internal and external environments. Damasio describes this knowledge as 'dispositions':

> All our memory inherited from evolution and available at birth, or acquired through learning thereafter, in short, all our memory of things, of properties of things, of persons, places and events and relationships, of skills, of biological regulations, you name it, exists in dispositional form (a synonym for implicit, covert, nonconscious) waiting to become an explicit image or action (Damasio, 1999:332).

Dispositional representations exist as potential patterns of neuron activity, which Damasio likens to the fictional town of Brigadoon. In other words, they exist in a potential state subject to activation in response to a thought or perception (Damasio, 1994:104). Their activation sets into motion a complicated concatenate of neurochemical responses which reliably control reflexes, drives and instincts. As Damasio (1994:119) puts it:

> ...neural signals give rise to chemical signals, which give rise to other chemical signals, which can alter the function of many cells and tissues (including those in the brain), and alter the regulatory circuits that initiated the cycle itself. These many nested regulatory mechanisms manage body conditions locally and globally so that the organism's constituents, from molecules to organs, operate within the parameters required for survival.

Damasio (1994) understands the workings of these subcortical processes to be the essence of the feelings we call emotions, motivations and drives. Although Breuer and Freud did not have the benefit of knowledge regarding the chemical nature of emotional feelings or qualia, they did put forward in the *Studies* the idea that hysterical symptoms may have their origins in the emotions. For example, Freud thought that it was not unlikely that the expression 'to swallow something', applied to an insult that had gone unanswered might really come from 'the sensations of innervation that appear in the throat if one stops oneself from speaking and prevents oneself from reacting to the insult'. (p184) Instead of hysteria using symbolization to create a somatic expression for the emotional idea as Freud initially argued, he thought it might be more the case that hysteria has

'not taken linguistic usage as its model at all, but that both hysteria and linguistic usage draw from a common source' (namely, the emotions). Taking an evolutionary perspective Freud states:

> They [the emotions] may now for the most part have been so much weakened that their verbal expression seems to be a figurative translation, yet it is likely that all this was at one time literally meant, and hysteria is right to express its stronger innervations by restoring the original meaning of the words. (p184)

Thus, when the patient reports that she feels as if something were 'stuck in her throat', the sensation and the figurative expression might be envisaged as having their inception in primordial systems that, at one stage in our evolution, would have expressed anger through screaming or some other vocal means. As Freud says, 'I maintain that it is not as singular and arbitrary as one would think for the hysteric to use symbolization to create a somatic expression for the affectively marked idea'. (p183) Thus, rather than the psyche always creating the symptom by symbolization, Freud conceded in the *Studies* that symptoms derived from the emotions and as he remarks, come with a 'psychical interpretation'. (p183) This latter interpretation, I suggest, is the more plausible and finds support from modern conceptualizations of implicit and explicit memory systems discussed in Chapter Three.

Implicit or unconscious information processing occurs in associative systems that are not only responsible for emotion, motivation and drives but also for more cognitive processes such as perception, procedural learning and memory, and the association of ideas. We are aware of the end result of such unconscious information processing, for example, the emotion, motivation or perception represented in consciousness as a feeling or an image. However, consciousness has little direct control over such processing, that is, what we feel or see (Le Doux, 1998; Damasio, 1994). According to Le Doux (1998:9):

> States of consciousness occur when the system responsible for awareness becomes privy to the activity occurring in unconscious processing systems. What differs between the state of being afraid and the state of perceiving red is not the system that represents the conscious content (fear

or redness) but the systems that provide the inputs to the system of awareness.

Damasio (1994) has also argued persuasively from scientific evidence that emotion and emotion feelings are subserved by separate neural systems.

If hysterical symptoms are the expression of abnormal or exaggerated fear emotions, examining the operation of the systems involved in conscious cognition will not help us to understand the phenomena characteristic of this condition. For example, when a person experiences a traumatic event not only do they encode memories that can be recalled at a later time and related to others or mentally processed, they encode memories of the experience. Retrieval results in the expression of bodily responses that prepare for danger (Le Doux, 1998). These responses may include a partial or total opioid-mediated freeze response affecting the subjective perception of the person's body and an inhibition of motor responses e.g., the inability to move or speak, or an enhanced perception of sensory phenomena perceived as threatening resulting over time in disorders of inhibition (Crittenden, 2006).

On this view, there would be no need to evoke the presence of an unconscious (potentially explicit) memory and by implication the concept of a quantitative conversion of affect in order to explain the symptom. All we need is knowledge about how fear conditioning operates within the amygdala and associated systems, which I will examine in the following chapters.

3 Learning to fear

Most theorists from Freud on have assumed that clinically debilitating anxiety is the result of traumatic learning experiences. While the current DSM outlines symptoms and situational factors that allow skilled clinicians to distinguish between the various anxiety disorders, leaders in the study of human fear have argued that when comparing the physiological responses seen in people diagnosed with phobias, panic disorders, PTSD, and so on, (disorders Freud referred to collectively as neuroses), what they have in common is far greater than their differences (Le Doux, 1998). On this view, such disorders reflect the activation of one and the same underlying response system to various fear conditioned stimuli whether in the form of external or internal stimuli, such as thoughts, memories and so on (Le Doux, 1998; Rolls, 1994; Damasio, 1994).

It was argued in the previous chapter that the sensory and motor disturbances characteristic of hysteria might be viewed as the operation of this innate 'general-purpose defense response control network' (Le Doux, 1998:158). Identification of this system will be undertaken in Chapters Four and Five. In the present chapter I am going to examine how, and under what circumstances, normal defense responses become pathological resulting in abnormal behaviour which often persists throughout the person's life-span. The main question I will be asking is this: Can the basic laws and principles of learning theory explain the dynamics underlying hysteria in a more parsimonious, scientific way than Freudian psychoanalytic theory?

3.1 The basic principles of learning in animals

First, what is meant by 'learning'? For the traditional behaviourist, learning refers to a change in observable behaviour that occurs as the result of experience (Yussen & Santrock, 1978). The behaviour should be relatively permanent, lasting for more than a few seconds or minutes and may last for several hours,

days, months or years. Learning occurs through association of a stimulus and a reflexive response (classical conditioning) or through association of a stimulus and a free or random response (operant conditioning).

It is important to recognize that the behavioural and physiological changes we associate with fear, are not considered by the behaviourist to be learned. Rather, they are thought to be reflexive. Just as all infants blink when a bright light is shone into their eyes, so babies cry, or make abrupt involuntary movements when they hear a loud sound. These reflexive behaviours are understood to be 'hard-wired' into the infant's nervous system and occur spontaneously. Whilst we do not actually learn the fear response, under certain circumstances such reflexive behaviours can be conditioned or learned in response to not only harmful stimuli but otherwise neutral or harmless stimuli which just happen to be present when fear conditioning initially occurs. In this way a person can learn to fear a wide range of stimuli not the least of which are other people.

3.2 Classical conditioning and affective behaviour

John B. Watson was responsible for popularizing the concept of classical conditioning in the United States during the 1920s. In his famous experimental work with an infant named Albert (often referred to in the literature as 'Little Albert'), he showed how this mechanism operates in learning to fear events or stimuli in the environment, so that many objects, smells, sights and sounds that are often in themselves harmless develop the capacity to evoke the fear response (Watson, 1926).

Albert allegedly had no observable fears when Watson first began his experiment. However, it could be demonstrated that when a loud sound was made next to his cot, he was startled and tried to escape from the situation. In the language of learning theory, the loud sound had served as an unconditioned stimulus (UCS) and Albert's startled response and attempts to escape were the unconditioned responses (UCR) we identify with fear behaviour. Watson (1926) then paired a

neutral stimulus, a small white rat that Albert had been happily playing with prior to the experiment, with the UCS. After several pairings of the UCS and the white rat, Albert showed a startle and an avoidance response pattern to the neutral object as well as to the loud noise so that presentation of the white rat alone—the conditioned stimulus (CS)—elicited a fear response (CR) (Watson, 1926).

From these findings Watson concluded that certain aversive stimuli (e.g., loud noises, pain, or sudden loss of physical support) are innately capable of eliciting fear responses. When these UCS occur, other stimuli that happen to be present acquire the capacity to elicit conditioned fear. Neuroses arise as the result of such traumatic learning experiences and then persist and influence behaviour throughout the person's life (Watson & Rayner, 1920).

Research has since shown that the more shocking or terrifying the event, the greater the rate of learning. Whereas Watson's subject, Little Albert, had required several presentations of the UCS with the potential CS (the rat) before he responded with fear to the animal, in extremely traumatic situations it often takes only one pairing of an UCS with a neutral stimulus for the association to be learned. This makes sense from an adaptive view as an animal that is a slow learner may not survive for long if, on the first exposure to a life-threatening stimulus, it fails to be wary of, say, the water hole where it barely escaped from a predator.

A particularly dramatic demonstration of single exposure fear conditioning using human adult volunteers, was carried out by Campbell, Sanderson & Laverty (1964). Here, the experimental conditions were extraordinarily traumatic: a temporary interruption of breathing induced by the drug Scoline, which causes momentary motor paralysis without impairing consciousness. While subjects experienced no permanent physical harm, the drug temporarily generated, in most volunteers, a horrible terror and the feeling that they were dying. The conditioned stimulus was a neutral tone. On the basis of a single pairing (of the tone with the

drug effects), a long-lasting conditioned fear response to the neutral tone was generated in these subjects. The fear reaction was evidenced by marked changes in such emotional indicators as heart rate, muscle tension, and respiration. Moreover, this traumatic, conditioned, emotional reaction was highly resistant to extinction: it persisted intensely despite many repeated extinction trials.

As behavioural approaches to understanding behaviour grew in popularity, many psychologists reinterpreted Freud's theories in terms of the basic principles of learning identified in infrahumans and humans alike.

3.3 Psychoanalytic theory versus behaviourism

From a behavioural perspective, Freud's complex constructs and theories were considered unnecessary obscurations which only served to confuse our understanding of neurotic behaviour (e.g., Wolpe, 1988; Ringness, 1975). Comparisons were often made between interpretations based on learning theory and those made by psychoanalysts. For example, using classical learning principles, Wolpe (1988) reinterpreted Freud's well-known phobic case concerning a small boy, 'Hans'. Hans was seen to have a phobic reaction to horses after witnessing a traumatic event in which a horse fell down, (most probably from lack of food, exhaustion or old age). Freud's view was that the horse phobia was an unresolved Oedipal conflict: a fear of being castrated by his father for desiring his mother had been displaced to horses. The trauma of witnessing the horse falling was the event that allowed the repressed unacceptable wish to emerge in the form of a phobia.

Wolpe, like any good behaviourist, saw it differently. He argued that a neutral stimulus (the horse) had become a CS capable of eliciting the fear response. Furthermore, Wolpe criticized Freud's selective use of patient information. Hans had allegedly reported that he 'got the nonsense' when he saw the horse fall down and his father confirmed that the anxiety broke out in his son immediately following the incident. Freud dismissed these surface explanations but Wolpe

took them at face value. Like Watson, neurotic behaviours, on his view, were nothing more than fear responses that had been conditioned to an aversive event and maintained by the presence of CS, that is, stimuli that were present during the initial traumatic event.

Another case where classical learning theory seems to offer a more parsimonious explanation for an observed neurosis involved an American airman named Pearson Brack who served in World War II. Brack developed traumatic hysteria following injuries received during an enemy attack on his aircraft (Mischel, 1976).

Brack was a bombadier in the Tunisian theatre of operations and flying his ninth mission when his airplane was severely damaged by flak at an altitude of about 10,000 feet. Suddenly the plane jolted and rolled and then began to dive. Brack regained control of the plane just in time to avoid crashing. During the plane's fall, however, he was hurled violently against the bombsight and seriously injured.

After his return from this mission he was hospitalized for a month and then, seemingly recovered, was returned to flight duty. On his next two missions, the tenth and eleventh, he fainted. Direct observation revealed that Brack's tendency to faint seemed specifically linked to being at an altitude of about 10,000 feet.

His problem was brought to the attention of a psycho-dynamically oriented psychiatrist. After intensive interviews, the psychiatrist concluded that his patient's fainting attacks were connected to deep, underlying anxieties rooted in childhood experiences. Brack was described as a basically immature person with long-standing insecurities who had inadequately identified with his father. The near-fatal plane incident was seen as essentially trivial, except in so far as it precipitated anxiety in an already insecure and immature individual. Consequently, the psychiatrist attempted to locate the hypothesized roots of the

problem in the form of repressed underlying anxieties. We can observe in this interpretation of Brack's predicament the same tendency to trivialize the precipitating event criticized by Wolpe in Freud's analysis of Little Hans.

In contrast, classical conditioning theorists saw the relevant causes of Brack's neurotic behaviour as the current conditions that seemed to control the patient's fainting spells (Mischel, 1976). We have seen that Brack's injury occurred when the plane was at an altitude of roughly 10,000 feet. On their view, altitude cues acted as CS eliciting a traumatic reaction (CR)—fainting—rather than any repressed childhood experiences hypothesized by the psychiatrist.

Which interpretation was right? Was Brack's fainting predisposed by an inadequate identification, or a conditioned traumatic reaction, or a little bit of both, or neither? At this juncture there is no way of answering these questions definitively. Regardless of who is correct, we should be clear about one thing: the psychodynamic and behaviourist perspective on the causes of neurotic behaviour are fundamentally different.

Yet despite these theoretical differences their treatment methodologies and goals are similar in that clinicians of both theoretical persuasions advocate the patient confronts and comes to terms with his fear.

3.4 Behaviour therapy: basic strategies

One of the main techniques of behaviour therapy was and still is, to some extent, 'systematic desensitization' developed by Wolpe during the 1960s (Wolpe, 1963). This treatment modality was based on the principles of classical conditioning in the belief that neurotic behaviours were conditioned responses to stimuli that other people did not necessarily find anxiety provoking.

For learned physiological and behavioural effects of classically conditioned fears to be extinguished, Wolpe reasoned that it would require counter-conditioning the

individual to make a competing (antagonistic) response to an anxiety-eliciting stimulus. If a response antagonistic to anxiety (such as relaxation) could be made to occur in the presence of an anxiety producing stimulus so that it is accompanied by a complete or partial suppression of the anxiety responses, the association between these stimuli (CS) and the anxiety response (CR) would be weakened. This hypothesis was based on a belief that no-one can be relaxed and anxious simultaneously.

To test this hypothesis, Wolpe taught his patients relaxation techniques that they could use later to inhibit anxiety. For example, Wolpe treated an adolescent boy who had a severe hand-washing compulsion. In addition to the compulsive hand-washing behaviour, the boy often spent up to three-quarters of an hour in an elaborate ritual of cleaning his genitals following urination before devoting several more hours to washing his hands.

Wolpe noted that his patient's washing rituals were always precipitated by urination so he taught him to relax deeply while viewing, or vividly imagining, a situation involving urine or the act of urinating. These stimuli were graded according to their potential to elicit anxiety. For example, imagining urine in a container might be considered a low anxiety stimulus, while being presented with a picture of a boy in the act of urinating would be considered to have the potential to arouse severe anxiety. In this way a stimulus hierarchy was constructed.

Beginning with the least anxiety-eliciting image, the boy advanced to successive items until the entire hierarchy was completed. If, at any point in the procedure, he experienced a decrease in his relaxed state (becoming anxious) while presented with an anxiety stimulus, he signalled Wolpe who promptly told him to discontinue his image of the stimulus until he felt calm. In this way, the boy was desensitized to stimulus hierarchies that dealt with urine and urination. When the images no longer provoked anxiety, the patient abandoned his cleanliness ritual. Using this regime, Wolpe reported excellent and rapid progress with a number of

patients suffering various neurotic behaviours.

The methodology used by Wolpe was, in some ways, reminiscent of that utilized by Freud and particularly Breuer (Breuer & Freud, 1895). For example, the use of relaxation techniques to help the patient achieve an almost hypnotic state; encouraging the patient to confront anxiety-producing stimuli; and the supportive assistance of the therapist in the reenactment or imaging process. To a degree, the goal of their treatment modality was also similar in that these clinicians attempted to eradicate the abnormal learned fear or anxiety associated with traumatic events.

However, whilst exponents of both interventions claimed their respective techniques were successful in attaining their goal with the majority of patients, only systematic desensitization has been subjected to carefully controlled experiments. Findings from these studies indicate that desensitization is a valuable method for treating not only phobias, but also a number of anxiety disorders that historically have been labeled 'neurotic' (Mischel, 1976).

Over the years, psychologists have demonstrated that many fears in older children and adults that cannot be readily explained by reference to known events may arise from traumatic learning processes (Le Doux, 1998). Often these events have occurred before the age of three or four years. It is well acknowledged by neuroscientists that due to slow development of the explicit (hippocampal) memory system, explicit memories of such emotional experiences are either not laid down in neural circuits, or memory fragments are not sufficiently integrated for a whole memory to arise in consciousness. However, emotional memories formed by the amygdala, which is part of the implicit system, may be elicited by current stimuli associated with reward or punishment. If Albert's experience had been of a more traumatic nature, or had Watson not had the good sense to undo (extinguish) the learned fear of rats, the child may have developed a rat phobia yet never known why unless he was told by a third party who witnessed the event, such as his mother.

Thus, if a phobia, for example, is developed toward a certain object during early childhood and we were to ask the afflicted person to explain his abnormal fear response, he might well give us a rationalization or confabulation, as Freud observed, rather than the truth, yet this common occurrence should not necessarily be interpreted as an instance of repression. A considerable number of studies have shown that as we frequently do not know enough about the real reason for our fears, we call upon our intellect to produce an explanation that conforms to the most likely scenario (e.g., Ringness, 1975; Gazzaniga & Le Doux, 1978; Rolls, 1999).

3.5 Hysteria and classical conditioning

Classical conditioning is a form of learning by experience which occurs outside of conscious awareness and, as behaviourists hypothesized many years ago, involves an organized but relatively independent response system that is evoked and maintained by its own set of regulating conditions (Mischel, 1976). I will say more about this system in the following chapters.

The question we need to now ask is can classical conditioning theory help us to understand the somatic phenomena characteristic of hysteria'? In some instances it obviously can. For example, when Breuer (Breuer & Freud, 1895) discusses how one patient knew that a certain situation always evoked heart palpitations (section 2.2), we can now see, with the aid of classical conditioning theory, that her symptoms were most probably the abnormal or exaggerated physiological effects of fear conditioning. Although Breuer does not describe the circumstances in which the (fear) response first took place, we can deduce that each attack was triggered by a CS. Breuer diagnosed this patient as suffering from hysteria so why not attribute all cases of hysteria to fear conditioning?

What other symptoms might yield to this explanation? The physiological signs that attend the fear response are not limited to heart palpitations. They can include

fainting as in the Brack example, 'freezing' or muscular contractions when terrified, and vomiting due to shock, just to state some of the more well-known reactions to a CS. As we have seen, according to classical conditioning theory, these so-called 'symptoms' are UCR to UCS but can become conditioned to neutral stimuli such as places, people, objects—even symbolic representations such as words may act as CS.

For human beings physical punishment evoking pain might be accompanied by verbal admonishments such as 'naughty boy', which then act as CS eliciting the fear response. As Jureidini & Taylor (2002:124) state, 'For the human child, terror need not arise from physical threat. Words will elicit it, or merely the implicit threat in some forseen future situation.' Although these clinicians do not refer directly to classical conditioning theory, their example lends itself to such an interpretation.

Hysterical or abnormally strong fear reactions have been observed in infants as young as two months while in the presence of abusive caregivers (CS). Components of the opioid-mediated freeze response may include: an inhibition of the blink response, facial immobility, motor stillness, lack of vocalization (e.g. crying), startle responses, and rigid immobility (where children may hold an awkward body or limb position unnecessarily for an excessive period of time) (Kozlowska, 2007). A particularly graphic illustration of the freeze response in an infant at-risk, is cited by Kozlowska (2007). The case involved a two-month-old boy, 'Harry', who was admitted to hospital with an unexplained fracture of the right leg. A video-taped play interaction between mother and child yielded the following observations:

> On being placed in the arms of his mother, Harry averted his gaze from his mother's face; his eyes widened in fear; and his body froze in sitting position. Harry's mother tried to engage him by showing him toys, patting him gently on the head, and talking to him in a low voice. Harry's gaze remained averted; he failed to respond to his mother's cues and maintained a silent, frozen posture. Harry's mother became increasingly distressed at his lack of response. She kissed Harry and roughly examined

> his head, neck, ears, and genitals, rotating the frozen, immobile Harry on his side and back, and lifting him up by his arms. Throughout the interaction, Harry remained silent, his limbs and torso retaining their frozen posture (Crittenden in Kozlowska, 2007).

Children who present with conversion symptoms that have their phylogenetic origins in the freeze response may continue to present with symptoms of the freeze response throughout their lives because the classically conditioned association was established implicitly by systems operating outside conscious control. As Breuer and Freud (1895) noted, patients often believe (erroneously), they could have controlled or even prevented their hysterical behaviour, such is the strength of most peoples' convictions regarding the ability to control bodily movements in accordance with their goals.

It seems plausible to hypothesize, in light of the above discussion, that hysterical symptoms are abnormal or exaggerated fear responses, as Breuer and Freud (1895) argued. In the language of behaviourism, such somatic phenomena are unconditioned (unlearned or innate) responses to an unconditioned stimulus such as pain which by association has become linked to an external stimulus. Thereafter, the external stimulus automatically triggers the response. In the previous chapter we called this unconditioned response, the stress response, which involves a concatenate of somatic events people experience and refer to as 'fear' or other negative emotions, but in reality represent the organism's attempt to meet the demands of threat.

As a number of clinicians and researchers have noted, the freeze response is most relevant to an understanding of hysterical symptoms, currently limited to reactions that mimic symptoms of neurological disease (e.g., pseudo-paralyses, and sensory disturbances). In civilian life, the majority of cases have their roots in early socialization experiences where as a child the person could not escape or avoid an abusive caregiver (see section 1.3). As a consequence of repeated interactions with a caregiver who inflicts pain and suffering on a child, molecular, structural and functional changes occur (Brown, 1994; McCallister, et al., 1999)

that increase the likelihood of long-term symptoms (Perry, 2001; Courschesne, et al., 1994; Perry & Pollard, 1998).

According to this view, the more any neural system is activated, the more it will modify and 'build in' the functional capacities with that activation, just as the more someone practices the piano, the more the motor-vestibular neural systems involved in that behaviour become 'engrained'. Similarly, the more someone is exposed to a second language, the more the neurobiological networks allowing that language to be perceived and spoken will modify. And, the more threat-related neural systems are activated during development, the more they will become 'built in' (Perry, 2001).

We have dealt with the formation of symptoms through classical or Pavlovian conditioning to threat and the ability for conditioned stimuli to evoke such innate defense reactions. Although the evidence for this neurobiological interpretation of hysteria is not conclusive at this stage, the findings are compelling. The opioid-mediated freeze response provides us with a more than plausible hypothesis for the motor and sensory disturbances characteristic of hysteria, and is also thought by some commentators to explain the paradoxical phenomenon traditionally referred to as 'la belle indifference' that often accompanies this condition (Kozlowska, 2007). On this interpretation, opioids do not simply filter out physical pain but also emotional pain.

However, we have yet to deal with the role of operant conditioning in maintaining a symptom that has proved successful in avoiding a threatening stimulus or has been socially rewarded by attention.

3.6 Operant conditioning and sickness behaviour

It is important to clarify at the outset of this section, the difference between the individual who has learned (through operant conditioning) that illness or disability is associated with rewards or helps avoid punishment, and the person who

deliberately uses illness behaviour in order to manipulate significant others. The difference between implicit and explicit deception (Kozlowska, 2007) from a social learning view has its basis in implicit and explicit learning of the consequences of a person's actions. Implicit social learning through experience or vicariously, does not require the engagement of brain systems engaged in conscious rule-learning, although, as we shall see, once a person becomes aware of the association between a behaviour and the reward or punishment that follows, the opportunity for manipulation exists. While this form of learning does not seem to be involved in hysteria understood as conditioned responses to threatening stimuli, it may help us to grasp the etiology of what is perhaps best conceived of as illness behaviour. Implicitly learned illness behaviour may become the basis for feigning or pretending.

Operant conditioning is a form of learning described by Skinner (1971) in which the response initially occurs freely, that is, not in reaction to a specific stimulus. Recall that in classical conditioning the initial unconditioned response is reflexive, or spontaneous, to a specific stimulus. While Skinner (1971) accepted that classical conditioning occurred in humans, he believed that our behaviour was not simply the result of reflexive mechanisms but also involved more flexible responses learned through reward and punishment. According to this view, if a response is followed by a reward it is likely to be repeated. In Skinner's terminology, this effect on the response is called *reinforcement*. Conversely, a response followed by an unpleasant experience is less likely to recur. Skinner referred to this effect as *punishment*. He was not concerned with whether the event was experienced by the organism as pleasant or unpleasant, rather, Skinner's focus was on the functional value of an event measured in terms of behaviour. Therefore, if an event follows a response and the response is repeated, the event is a *reinforcer* whether it is pleasant or unpleasant. If an event follows a response and decreases the likelihood that the response will recur, the event is a *punisher*.

It was Skinner's belief that psychology is the science of behaviour: inferences about unobservable inner states and motives were not considered by him or his followers to constitute adequate explanations and added nothing to a scientific account of the conditions controlling behaviour (Skinner, 1953). As he saw it, a 'drive' such as 'hunger' is just a convenient way of referring to the observable effects of deprivation or satiation of some substance, such as food, for a given period of time. If an event (reward) follows a response and the response is repeated, the event is a reinforcer: we do not need to refer to the organism's internal state to explain normal or abnormal behaviour.

In this respect Skinner's position on learning was quite different to that held by early behaviourists who took a more Freudian view of the unconscious dynamics involved in learning. Theorists such as Mowrer (1939) and later Dollard and Miller (1950) thought that if a behaviour served to reduce tension or arousal evoked by either a strong internal or external stimulus (drive), then that behaviour is reinforced. Think of an animal in the psychologist's laboratory. Motivated by the drive of hunger, the animal engages in various food-seeking activities. At one point, it happens to see a lever (a cue). The animal's behaviour (response), at first accidental, is to press the lever, and this action releases food into its cup. It eats the food thereby reducing the tension caused by hunger. Now, in the future, when the animal is hungry, it is more likely to press the lever again. The association between the cue stimulus (the lever) and the response (pressing it) has been strengthened.

This concept of learning, put forward by Dollard & Miller (1950), with its emphasis on drives was very similar to Freud's emphasis on motives and impulses as the forces underlying behaviour. However, whereas Freud emphasized instinctual drives, Dollard & Miller made room for many learned (secondary) drives, the roots of which lie in primary drives such as the need for food and shelter. Most human behaviours involve goals and incentives whose relations to innate biological needs seem extremely remote. For example, people strive for

power, love, self-realization and so forth. However, for Dollard & Miller these researchers, such motives could be traced back to basic survival needs. Money, status and power ensure a person's survival and position within society while a lack of these benefits may have the reverse effect. Social inclusion is necessary for individual survival and bonding with a mate ensures the survival of the species. Even human creativity may have its phylogenetic roots in exploratory behaviour evidenced by the willingness of an animal to venture into novel situations. On this view, such behaviour might be considered a precursor to creativity.

As we shall see in Chapter Seven this view is consistent with that put forward by Kozlowska (2007) and other 'evolutionary psychiatrists' (Nesse, 2000), namely that somatoform disorders, have their phylogentic roots in animal defense behaviours.

While Skinner eschewed all talk of internal states in an attempt to explain behaviour in terms of changes in external conditions only, we can perhaps understand his position as a reaction to the then current trend among personality theorists to attribute human behaviours to obscure motivational states such as 'wishes' and 'desires' rather than to basic biological needs shared by all animals. Skinner and his like-minded colleagues saw this less scientifically rigorous application of motivational theory as an obstacle to understanding human behaviour. For example, a child's spending an unusual amount of time cleaning his room or grooming himself might be explained by postulating that the child had 'strong cleanliness needs', or because he had a compulsive desire for order.

For Skinner such hypotheses about motives might sound like explanations but tell us little unless the motive is defined objectively and the causes of the motive itself is established. For example, what makes the child have 'cleanliness needs'? What determines his 'compulsive desires'? Why does he wish to be clean? Radical behaviourism criticized many concepts regarding human needs as being no more

than motivational labels attached to human activities. Thus, orderly behaviour may be attributed to a motive for orderliness, submissive behaviour to submissive needs and so on. To avoid this circularity and to untangle explaining from naming, behaviourally oriented psychologists like Skinner preferred to analyse behaviours in terms of the observable events and conditions that seemed to vary with them. Rather than hypothesize the needs that may propel a particular activity, they tried to discover the events that strengthen its future likelihood and that maintain it or change it. In other words, they searched for the conditions that regulate the observed behaviour rather than hypothesized need states inside the person as motives that drive him (Mischel, 1976).

Modern behavioural psychologists do not need to be so cautious. References to emotion and motivational behaviour in humans are based on animal studies that show we have far more in common with other species than personality theorists conceded in the mid-20th century. As many researchers tell us, when it comes to such behaviour reward and punishment systems operating outside of conscious awareness compute the value of sensory stimuli based on past learning, and then use the selection between different rewards and avoidance of punishments to produce appropriate behaviour (Rolls, 1999; Damasio, 1994; Le Doux, 1998). The behaviour can be thought of as appropriate in the sense that it is based on the sensory systems and reward decoding that our genes specify (through the process of natural selection) in order to maximize their fitness (reproductive potential). Thus, as both Freud and Skinner implied using different theoretical frameworks, humans often do not have insight into the emotional and motivational causes of their behaviour and their explanations often take the form of confabulations or rationalizations based on common sense inferences.

An amusing anecdote which serves to illustrate the nonconscious nature of operant conditioning is provided by McCrone (1993:5). The story is set in the 1960s during the high tide of behaviourism and is about a psychology professor whose students collude to play a prank on the die-hard behaviourist. For most of

his lectures, the professor scribbled away on the black board pausing only now and then to make a particular point, at which time he would often toss a chalk lightly in one hand. By prior agreement, the students paid special attention to these moments of chalk-tossing, leaning forward in eager anticipation of the professor's every word. Whenever chalk was not being tossed, the students put on a great display of yawning, fidgeting and generally acting terminally bored. Of course, what they were attempting to do was reinforce the chalk-tossing behaviour by offering flattering attention.

Gradually and unsuspectingly, the professor spent more and more time tossing chalk. Then, during the following weeks, as the students responded only to ever more dramatic displays from him, the chalk started to rise higher and higher until the poor man was regularly hitting the ceiling of the lecture room. Without realizing it, the professor had become as controlled as one of his caged rats.

It was amusing for the students to observe the professor's behaviour being controlled by the same principles of learning he had used to condition bizarre behaviour in laboratory rats, however, it would be tragic if people inadvertently develop neurotic behaviours as a result of accidental reinforcement contingencies. Skinner (1953) claimed to demonstrate how this might happen using pigeons for his subjects. He observed that if a pigeon was given food at regular intervals, say, every 15 seconds, regardless of what it was doing, irrational or inappropriate behaviour developed. Conspicuous operant responses conditioned in this way included turning sharply to one side, hopping from one foot to the other and back, bowing and scraping, and strutting. The particular piece of behaviour became a permanent part of the bird's repertoire even though the food had been given according to an automatically regulated feeding program and was unrelated to the bird's behaviour.

What Skinner doesn't mention is the fact that had the pigeons in his experiment not been hungry, operant conditioning or learning would not have occurred. In

other words, as early behaviourists argued, an innate stimulus such as hunger or pain motivates defensive behaviour and if that behaviour is successful in reducing such unpleasant stimuli, it is reinforced. The inclusion of primary internal stimuli in operant conditioning rather than just secondary external stimuli helps us to understand how a reflexive response might become subject to external reinforcement leading to illness behaviour. The idea that some internal stimuli, such as the taste of food if the animal is hungry, or pain, are unlearned (primary) instrumental reinforcers, while others become reinforcing by learning due to their association with such primary reinforcers and are thus secondary or contingent, is congruent with contemporary behavioural theory (Rolls, 1999:4).

On this view any behaviour that is successful in avoiding an unpleasant event thereby reducing threat-elicited arousal would be reinforced and thus maintained.

Skinner called this process whereby habitual behaviours are formed, *negative reinforcement*. Again, Skinner explained this type of behaviour without resorting to the role of primary (internal) reinforcers. The concept of negative reinforcement is often confused with punishment, however it is quite different. For example, supposing a child is constantly reprimanded by her teacher for looking around the class-room when she should be reading. The child buries her head in her reader and the teacher stops berating her. If removal of the teacher's scolding occurs each time the child hides her head in a book, the child's response is said to have been negatively reinforced—she may even become a habitual reader (Yussen & Santrock, 1978). Negative reinforcement involves a far more subtle process than punishment as a negative reinforcer encourages responses by removing the aversive stimulus. Punishment simply produces compliant behaviour by ensuring that the act is less likely to recur. Negative reinforcement may explain how innate physiological responses to threat or components of those responses can become strategies used by the individual as a means of avoiding or removing an aversive stimulus.

Consider the following abstract from a case of hysterical vomiting reported by Taylor (1989) which shows how a classically conditioned response can become operantly learned through negative reinforcement :

> A girl of nine vomited her lunch when her sister and boyfriend started to punch one another. The fighting stopped and she was 'rushed to hospital'. Later a recurrence of the vomiting led to another admission and then one lasting six weeks, needing intravenous fluids. The psychiatrist met her gently mewing into the steel bowl provided. Her trick of deliberate vomiting continued inexorably ... No negotiation with the patient about her facility to vomit proved possible over 18 months in psychiatric care nor during a subsequent stay at another hospital. There, she suffered a rupture of the esophagus from the stomach and, following a heroic repair, she requested orange juice by mouth. When refused this she made to vomit, reopened her wound, and bled to death (Taylor, 1989:391).

Although the psychiatrist reporting this case understands the patient's vomiting behaviour to be a 'deliberate trick', it is clear that the child's vomiting attack was initially caused by witnessing domestic violence. In the terms of classical conditioning, an UCS, the violent dispute, elicited an UCR—fear induced vomiting. In addition to the people responsible, any number of stimuli that had been present at the time this violent fracas occurred may have later acted as CS capable of triggering vomiting (the CR) on future similar occasions.

However, as we can see from this documented case, the child continued to vomit in hospital where it was unlikely that the CS were present. It is true that we could explain the persistence of vomiting as due to the human propensity for thinking or using internal symbolic images and words as Jureidini and Taylor (2002) suggest. For example, thinking about going home might have been sufficient to elicit vomiting if 'home' was a strong conditioned stimulus, just as thinking about an impending visit to the dentist might cause heart palpitations.

Yet, we have reason to suppose that although classical conditioning was initially involved in this case, the child appeared to have learned that her vomiting was a successful strategy inasmuch as it caused the fear-inducing event to cease (and thereby reduced arousal). In the terms of operant conditioning theory, the

classically conditioned symptom was negatively reinforced and only later still, was the behaviour deliberately used to manipulate others. A more trivial example might be the child who cries when her mother leaves her at the school gate so mother takes her home again thus reinforcing an emotional, unconditioned response triggered by a perceived absence of physical support by a caregiver (a primary unconditioned stimulus). The next morning the child cries again and if this pattern continues she may deliberately use crying behaviour in order to avoid going to school.

Classical and operant conditioning can occur not only through direct experience but also through identifying with others (Bandura, 1969). Thus, some cases of hysteria may be explained by appealing to observational learning. As clinicians have observed many dysfunctional families appear to communicate through the language of illness and there can be 'contagion' due to modeling so that certain 'sicknesses' seem to run in these families (Jureidini & Taylor 2002; Kozlowska, 2007; Stuart & Noyes, 1999). Early exposure to illness increases the likelihood that distress will be manifested somatically as I shall show in Chapter Seven.

3.7 Observational learning

Observational learning, or learning without direct reinforcement, is also referred to in the literature as 'cognitive', at other times, 'vicarious', 'modeling', 'imitation, or 'identification'. All these terms refer to the tendency for a person to produce the actions, attitudes, or emotional responses exhibited by real-life models such as one's parents or symbolized models, for example, the comic-book hero Superman. Consequently, social learning theorists have argued that for the sake of clarity and parsimony, these terms may be used interchangeably to refer to behavioural modification produced by exposure to modeling stimuli (Bandura, 1969).

For our purposes what is important about observational learning is there is evidence to show that vicarious experiences may also lead to strong (classically)

conditioned emotional reactions (Berger, 1962) and, under extreme circumstances, may result in hysterical phenomena. For example, in one experiment, adults repeatedly observed the sounding of a buzzer paired with fear responses feigned by another adult. Gradually, the observers themselves developed conditioned fear responses to the sound of the buzzer alone (Bandura and Rosenthal, 1966). Consider also the development of strong fears, even phobias, to snakes and spiders in people who have never been harmed directly by these creatures. Often observing the emotional upset of other people when confronted by a snake or spider is sufficient to elicit strong feelings of fear.

Hysteria may, in some cases, occur as the result of identification with a significant other. As Showalter (1997) observes, mass hysteria is more likely to occur among people with whom one identifies such as friends or family members. Consider for example, a case recounted by Breuer and Freud (1895:8):

> A highly intelligent man is present when his brother has an ankylosed hip joint stretched under anaesthetic. Just as the joint gives way with a crack he feels a violent pain in his own hip joint which then lasts for almost a year....

An interesting, albeit extreme example of this phenomenon, is the condition known as 'couvade syndrome' or 'sympathetic pregnancy' which occurs in some men who over-identify with their pregnant spouses. The whole gamut of changes associated with pregnancy, including abdominal swelling, lactation, craving for strange foods, nausea, even 'labor pains', can occur (Ramachandran, 1999:218).

Considerable research has been carried out by social psychologists over the years to assess the effects of modeling and observed consequences on behaviour. For example, in one study, children watched a film of an adult who displayed novel aggressive responses, such as hitting and kicking an inflatable doll (Bandura, 1965). The consequences elicited by the adult's aggressive behaviour were deliberately varied, so that in one condition, the film sequence showed the adult's aggressive behaviour being punished; in a second it was rewarded; and, in a third, it had no consequences. When tested after viewing the film, the children who had

seen aggressiveness rewarded showed the highest incidence of the modeled behaviour while the children who observed the adult being punished showed the lowest incidence of aggressive behaviour. In other words, operant conditioning appeared to occur in these children indirectly rather than directly.

These kinds of observations have naturally influenced clinicians treating patients with hysterical symptoms. Often such symptoms are considered by the practitioner to be the result of something like observational learning, particularly if there is a family history of functional illness. This view dates back to the well-documented work of Babinski, who like Freud and Janet, studied under the famous French physician Jean-Martin Charcot (1825-1893). Babinski convinced Charcot that the symptoms presented by his patients were actually artifacts of the neurological examination itself which encouraged patients to manifest suggested symptoms by rewarding their compliant behaviour with shelter and food. Most of Charcot's subjects or so-called 'patients' were prostitutes—distressed, vulnerable, women who, as Babinski pointed out to his mentor, were not only highly suggestible to Charcot's ideas, but encouraged by his interest in them. On Babinski's view, these patients were not afflicted by a disease but by an idea (Babinski in McHugh & Slavney, 1998).

In light of the above discussion, we can detect several processes at work in Charcot's patients. First, people in a state of abnormal arousal due to anxiety are highly suggestible and thus more likely to accept an interpretation of their condition by an authority figure (Le Doux, 1998). However, the importance of this situation recorded by Babinski is that it nicely illustrates how observational learning of operant responses can occur that mimic those genuinely elicited in traumatized patients. As new patients were admitted to the Paris hospital 'La Salpetriere' where Charcot ran his clinic, they were brought into contact with others suffering from various manifestation of hysteria. They saw that these patients were well fed and attended to by medical staff and 'the most celebrated doctor of his time' (Showalter, 1997:30) showed that he was greatly interested in

their problems. In the terms of observational learning theory, the new patients modeled behaviour that they observed was reinforced or rewarded in others on the same ward.

Interesting too, is the treatment that Babinski encouraged Charcot to put into action in order to modify their behaviour as it is similar to that proposed by behavioural therapists. One practical application of operant conditioning is behaviour modification. For decades clinical psychologists have used techniques based on simple reward and punishment contingencies to change unacceptable behaviours learned through the same implicit process. Often parents unwittingly reinforce acts of aggression, pretending to be sick, tantrums, and so on, through attention, just as Charcot and his medical staff innocently reinforced the modeling of mental illness. Children are also most vulnerable to vicarious operant conditioning because they generally tend to imitate the behaviour of the same sex parent with whom they identify (Mischel, 1976).

Long before learning theories of behaviour and behaviour modification were formulated, Babinski proposed a two-step plan aimed at changing the learned hysterical behaviour of patients. The program was based on isolation and counter-suggestion. First, the patient was transferred to a general ward away from the influence of other patients manifesting hysterical behaviour and also from staff members who, by their concern and attention, were responsible for unwittingly reinforcing and thus maintaining the so-called 'symptoms'. The second step, counter-suggestion, was aimed at changing the patient's view of themselves and their predicament. They were discouraged from casting themselves in the 'sick' role and helped to address the psycho-social problems underpinning their distress. Staff members expressed their withdrawal of interest in hysterical behaviour subtly, for example, patients were told that they were now in recovery and although they would receive some physiotherapy to assist them gain mobility, the main task was to concentrate on how to remedy the main cause of their problem, their lifestyle.

These face-saving counter-suggestions reduced a patient's need to go on producing hysterical symptoms in order to certify that her problem was real. The symptoms then gradually withered from lack of nourishing attention. Patients began to take a more coherent and disciplined approach to their problems and found a resolution more appropriate than hysterical displays.

History shows that others have also been aware of the processes operating to maintain neurotic behaviour. For example, during World War I, the infamous Dr. Yealland (1918) chose to treat soldiers suffering with hysterical symptoms not with Babinski's benign indifference, but with cruel and punishing methods. It was Yealland's task as a medical officer to return his patients to active duty as soon as possible in order to ease a critical shortage of men. To this end, he carried out 'shock' treatments and autosuggestion on his patients. For example, he treated a soldier suffering from hysterical mutism by tying him in a chair, applying electric shocks to his throat while, at the same time, encouraging him to 'remember, you must behave as the hero I expect you to be... A man who has gone through so many battles should have better control of himself'.

Another war-time example offered by Taylor (1989) concerns a documented case of mass hysterical phenomena observed by a medical officer which occurred among survivors of a war-ship torpedoed during World War I. A number of men presented with quadriplegia, paraplegia, mutism, snarling, weeping, barking, shaking amounting to spasmodic movements of the upper limbs. These symptoms all yielded to treatment aimed at restoring normal physiological and psychological function. As the medical officer recorded in his notes:

> The treatment was simple. They were stripped naked in an overheated room and energetically rubbed by two vigorous sailors with a hair glove soaked in alcohol. As soon as they had been warmed externally and internally with rum, I took each one separately and smacked him harder and harder until the disturbances disappeared, all the time speaking kindly to them and expressing my delight at the rapidity of their recovery. No-one resisted more than ten minutes: many were cured of contagion on

> witnessing the treatment of others. The majority expressed to me their gratitude on witnessing the treatment of others...I was able to see my patients for a week and there were no relapses (Ross, quoted in Taylor, 1989).

According to behavioural psychologists, as all modeling or imitative behaviour is essentially under the control of reinforcement, we might explain the instant 'cure' of soldiers watching such extreme treatment carried out by the medical officer as the result of observational learning. It is doubtful however, that in such cases, where fear conditioning has occurred, the patient is actually cured. One would think that it would be to an animal's advantage not to forget aversive stimuli and the strategies learned to avoid them even if the threat was no longer present. To a victim of rape, the knowledge that the perpetrator is incarcerated often does not cause her anxieties to cease or prevent sometimes, extreme, evasive behaviours from being taken to avoid a similar act of abuse.

It is apparent that in cases of hysteria, tentatively defined as abnormal reflexive or automatic defense responses, the victim is not simply suffering from an 'idea'. The symptoms, at least to begin with, are real physiological responses that can be measured and observed. However, if, and when, operant conditioning occurs, what would otherwise be transitory phenomena may become a 'strategy' or learned habit and thus appear resistant to all remedial attempts unless recognized by an astute, experienced physician. As Taylor says, in the case of clinically presented hysteria, 'hysteria, [can] take a more chronic hold ...[if] these physiological defense mechanisms do not 'unlock' instantly the situation improves' (Taylor, 1989:394). The following case cited by him, shows the importance of recognizing and dealing with the symptom as soon as possible:

> A courageous GP refers a boy aged eight who has 'gone of his legs' that morning. The GP says he knows it is hysterical but that he cannot stop it. The boy is brought by car. A kind psychiatrist negotiates through the window the need for the boy to mount the steps to come and talk. The boy comes. He talks about how his estranged father has promised first a trip to the cup final and then to the replay and had twice let him down. The anger, love, disappointment, and the humiliation in front of his friends took his legs from under him, turned him weak at the knees, turned his

legs to jelly. He walked out of the consultation and remained well.

Although there is no evidence to suggest that the child did not respond to similar future challenges with distress, Taylor's point that if such defense mechanisms are not encouraged or rewarded by the social environment the problem does not become chronic, or habitually produced, is valid in light of anecdotal evidence involving cases of transient threatening situations. However, as I have argued, if the threat is prolonged, particularly in children, specific changes in neurodevelopment and function may occur resulting in long-term symptoms.

We have examined the possibility that in humans as well as in infrahumans, emotional/motivational learning, that is, learning through reward and punishment, is greatly enhanced by observational learning. Such learning can proceed without awareness and this appears to be the rule throughout the animal kingdom including the human kingdom as the amusing story of the chalk-tossing professor suggests. However, some social learning theorists such as Rotter, Anderson, Bower, Bandura and DeNike, showed that we are not just the victims of habitual or conditioned responses as traditional behaviourists claimed. On their view, a person's private beliefs about what is happening to her and her expectations may also affect the person's actions. On their view, this requires an *awareness* of the contingencies and rules governing the sequences to which the person's responses will lead.

3.8 Learning and awareness

Consider, for example, an operant conditioning situation in which the experimenter gave social reinforcement (saying "Mmm-hmm") to subjects when they said human nouns (eg. 'girl') in a word naming-task. Not surprisingly, subjects who became aware of the contingency for reinforcement (i.e., guessed correctly that social reinforcement from the experimenter depended on their saying human nouns) greatly increased their output of nouns (De Nike, 1964). Those who remained unaware of the contingency did as poorly as subjects in a control group who were reinforced randomly for ten percent of their responses.

Most interesting was the finding that students showed no appreciable improvement in their performance until they correctly discerned the contingency for reinforcement; as soon as they became aware of the contingency they gave dramatically more noun responses.

Whenever a symptom is reinforced the propensity for that symptom to become a learned strategy for avoiding a negative event or as a means of gaining care and attention is present. Caregivers who neglect their child's basic need for security and stability or fail to pay them any attention unless they are ill or injured, may inadvertently reinforce such behaviours. The enactment of illness behaviour may occur in these children without awareness as the associative systems responsible for operant conditioning operate outside conscious awareness. How to discriminate between the patient whose symptom is the result of operant conditioning from the person who is aware of the association between her response and a reinforcing event is a constant concern for clinicians. However, we should not conflate learning the contingency for reinforcement (operant conditioning) with the awareness of that contingency which is considered by some psychologists to be one of the highest possible forms of learning (Gagne, 1977).

Technically, patients who deliberately induce their symptoms for secondary gain are not diagnosed with somatoform disorders. Rather they are seen as 'malingerers' or as Breuer & Freud (1895:244) put it, the deception is not based on the hysterical disposition, but on 'the complication of this disposition with other types of degeneration, of innate moral inferiority'. They go on to state that:

> The 'malicious hysteric' develops in exactly the same way, when an innately excitable but emotionally impoverished person falls prey to the egoistic stunting of character that is so easily engendered by chronic infirmity.

This situation is perhaps analogous to that of a person who has heart disease or back problems and uses his condition as an excuse to avoid unpleasant task or social duties. The condition is genuine in the sense that we can offer a

physiological explanation for the symptom (the stress response), just as we can for heart disease or back pain. However, the deliberate use of the complaint or symptom as an excuse for 'escaping' duties or controlling the behaviour of others is a psychosocial problem that requires different remedial methods to those used to treat the actual condition.

From a developmental perspective, it is entirely feasible that once a child is aware of the contingencies and rules governing the 'sick' role he may choose to enact appropriate sick behaviour in order to escape or avoid an unpleasant situation or just to gain attention. Every parent is familiar with the pretended ache or pain that can suddenly precede an exam, an inoculation or some other unpleasant event scheduled to occur at school that day. In other words, the child has become aware of the 'if...then' rule which substantially facilitates learning irrespective of what is being learned. Once this happens, a person is able to manipulate (gain mastery over) situations and other people (Mischel, 1976).

In the majority of cases when hysteria or its contemporary appellations, are diagnosed, the patient has become, as Taylor (1989) suggested, 'locked' into a pattern of behaviour. What the physician is mainly seeing in older children and adult patients is not an abnormal response to an immediate or ongoing threat but at best a habitual enactment or at worst an act of deception. As we have seen, Breuer thought that such deceit does occur in hysteria, despite what was then a current trend among his colleagues to deny simulation, but in his experience such cases were not common. The standard medical view has erred in the opposite direction as the literature shows. This is unfortunate as many victims of hysteria are entrenched in domestic situations where defense mechanisms do not 'unlock' because the person cannot escape their stressful circumstances. In the next chapter, we will attempt to understand the predicament of these patients from a neurobiological perspective. What happens inside the person that results in him experiencing disturbing symptoms?

4 The neurobiology of fear

It is clear from the previous chapter that a scientific understanding of hysteria must include the well-researched principles of classical and operant conditioning. In turn, these principles have their origins in the function of the animal nervous system. As Rolls (1999:4) states:

> A fundamental operation of most nervous systems is to learn to associate a first stimulus with a second which occurs at about the same time, and to retrieve from memory the second stimulus (or its consequences) when the first is presented.

For instance, if an infant touches a hot radiator and burns his fingers, an association is made between a primary stimulus (pain) and the radiator (a secondary stimulus). Following the event the radiator becomes a CS capable of eliciting avoidance behaviour an UCR. The UCR is subjectively experienced as the aversive feeling we call 'fear' but in actual fact fear feelings are the result of the nervous system preparing the body for action. The emotion fear entails autonomic reactions to threat or perceived threat, resulting in familiar physiological changes such as accelerated heart rate, rapid pulse, sinking feeling in the stomach, tremors, mobility problems, and so on.

Thus, an emotional experience and emotional behaviour is the result of a nonconscious 'bottom-up' process. If this view of emotions is correct, it follows that we do not need to appeal to conscious emotion feelings or an understanding of consciousness in order to explain hysterical reactions understood as abnormal fear reactions. It is often the case in humans that 'top-down' processes in the form of explicit memories and thoughts have the capacity to trigger emotion systems causing anxiety and anxiety-related symptoms, but the reaction itself is initiated by certain sub-systems in the brain common to many different species (Damasio, 1994; Le Doux, 1998; Rolls, 1999).

4.1 Fear as biological function of the nervous system in response to threat

Fortunately, neuroscience has accumulated a considerable body of research findings on the structure and function of the brain systems responsible for reacting to threatening stimuli. In his book *The Emotional Brain* (1998), Le Doux presents this information and attempts to correct many of our misconceptions on the subject of emotions resulting, for the most part, from our common-sense intuitions. Like Damasio (1994), Le Doux (1998:14) wants to make a distinction between the automatic processes we call emotions and emotion feelings:

> Feelings of fear, for example, occur as part of the overall reaction to danger and are no more or less central to the reaction than the behavioural and physiological responses that also occur, such as trembling, running away, sweating, and heart palpitations...Fear feelings and pounding hearts are both effects caused by the activity of this system, which does its job unconsciously—literally before we actually know we are in danger. The system that detects danger is the fundamental mechanism of fear, and the behavioural, physiological, and conscious manifestations are the surface response it orchestrates.

It is the case, that although different species have their special ways of responding to danger, commonality of function patterns is the rule at the physiological level and there are only a limited number of response strategies available to an animal: withdrawal (avoiding the danger or escaping from it); immobility (freezing); aggression (appearing to be dangerous and/or fighting back), and submission (appeasement). Hysterical symptoms may be seen, on this view, as derivative upon such responses. (Recall, for example, that conversion paralysis has been thought of by some theorists as a partial or incomplete freezing response.) Again, the extent to which these strategies apply across the various species is striking. Consider the following description of human defence by Blanchard and Blanchard (1972) quoted in Le Doux (1998:131):

> If something unexpected occurs, a loud noise or sudden movement, people tend to respond immediately...stop what they are doing...orient toward the stimulus, and try to identify its potentiality for actual danger. This happens very quickly, in a reflex-like sequence in which action precedes any voluntary or consciously intentioned behaviour. A poorly localizable or identifiable threat source, such as a sound in the night, may elicit an active immobility so profound that the frightened person can hardly speak

> or even breathe, i.e. freezing. However, if the danger source has been localized and an avenue for flight and concealment is plausible, the person will probably try to flee or hide...Actual contact, particularly painful contact, with the threat source is also likely to elicit thrashing, biting, scratching, and other potentially damaging activities by the terrified person.

Although the above description is anecdotal in the case of humans, the strategies do accord with our experience and observations of the fear response in others, and have been observed to occur routinely in other animals under controlled laboratory conditions. This uniformity suggests to Le Doux and his colleagues that either we all learn to be fearful in the same way or, more likely, the patterns of fear reactivity are genetically programmed into the human brain.

4.2 The emotional and cognitive unconscious

It is now widely accepted in the human sciences that operating outside of conscious awareness are neural systems capable of comprehending external events and regulating organized interactions with the world (e.g. Milner, 1963, 1982; Le Doux, 1985; Smolensky, 1988; Reber, 1993; Damasio, 1994; Karni, 1996; Rumelhart, Hinton et al., 1996). These systems are thought to be direct descendents of phylogenetically older parts of the brain and, more than the mechanisms of language and consciousness, reflect our biological heritage as vertebrates (Le Doux, 1985).

Scientific acceptance of the role of unconscious or implicit information processing in human behaviour has occurred only slowly over the years, due, for the most part, to a widespread reticence to promote Freud's concept of the unconscious as the abode of repressed mental entities (Reber, 1993; Le Doux, 1998). With the rise of cognitive science, the association of the unconscious with psychopathology has largely been replaced by an adaptive model which promotes the cognitive unconscious as the foundation for perception, practical learning, problem-solving, and a host of various processes once attributed to higher cognitive systems (Wilson, 2002).

Evidence for implicit learning taken from the annals of neuroscience is vast. Most comes from studies with patients who have suffered injury or disease to some part of the brain. Two documented cases that graphically illustrate the existence of implicit learning are used by Le Doux (1998) to support his argument for distinguishing between the emotional and cognitive unconscious.

In the early part of the 20th century, a French physician named Edouard Claparede examined a patient who, after suffering brain damage to a certain region of her brain, lost the ability to form what we would now call explicit memories (episodic and semantic). Her memory problem was so severe that if the doctor left the room and returned several minutes later, she could not recall having met him. One day, Claparede concealed a pin in his right hand so that when the patient shook his hand, as she was accustomed to doing, the pin pricked the palm of her hand and she pulled it back reflexively. The next time he returned to the room she still did not recognize him but refused to shake his hand even though she could not give him a reason (Le Doux, 1998). In terms of classical conditioning theory, Claparede's patient had formed an association between a painful stimulus (the pin prick) and her doctor which elicited a fear response in the form of avoidance.

Another patient, H.M., who suffered debilitating epileptic seizures underwent extensive surgery to remove large areas of the temporal lobes identified as the sites responsible for his condition (Milner in Le Doux, 1998). However, following the procedure H.M. lost what most people would think of as his memory. Specifically, he lost his capacity to form explicit, declarative or conscious long-term memories of the type so important in the maintenance of the autobiographical self. Forty years after his surgery, H.M. still had no knowledge of how old he was, where he was living, or the current status of his parents (who were long deceased). Later, after considerable research, the cause of his amnesia was shown to be directly related to the amount of hippocampal tissue removed.

This region of the brain has since emerged as a leading candidate for the encoding of new memories (Le Doux, 1998).

Although H.M. could not learn the names of his carers, nor remember their faces or recognize a recent photo of himself, like Claparede's patient, he was capable of emotional learning. H.M. learned to avoid 'stooges' who had been instructed to act consistently 'cool' toward him yet could not give a reason for his behaviour. In addition to demonstrating emotional intelligence, H.M. was able to learn both manual and cognitive skills and solve problems that required learning quite complicated, rule-based strategies and even improved upon these skills with practice, all without having any recollection of having the learning experiences that lead to his improved performance. This and other similar cases show that the type of knowledge we refer to as 'tacit', 'procedural', or 'know how', is not dependent on conscious learning. For example, a person may know how to back a trailer into a driveway but not be able to relate all the implicit rules that have emerged from experience and gradually led to his expertise.

It is evident that the systems spared in these patients learn through association. However, whereas in classical conditioning learning occurs through association of a stimulus and a reflexive or innate response, giving rise to affective behaviour, operant conditioning involves an association between a response and negative or positive feedback from the environment producing more flexible behaviours. Through operant conditioning we can learn social skills and other practical skills that are adaptive and useful to survival within our particular family, social group, and wider social sphere.

Since both classical and operant conditioning (including observational or imitative learning) occur unconsciously, some psychologists have expressed the view that these behaviours are not 'mental' or 'cognitive' in nature (e.g., Gagne, 1977; Yussen & Santrock, 1978). For example some behavioural changes occur very slowly and have a profound import on many areas of a child's psychological

life such as the logical skills required to understand the laws of physics and principles of higher mathematics. They are viewed by Yussen & Santrock (1978:156) as 'structures of thought' or cognition: 'These structures develop more slowly than learned behaviours, they are mental in nature, and they underlie a broad range of behaviour.' Since these authors suggested we distinguish between the different kinds of learning (classical, operant and observational) and cognition (equated with mental or conscious processing), psychologists have increasingly come to view all learned behaviour as the result of cognitive processes and the structures underlying such behaviours are generally referred to as 'the cognitive unconscious'.

Le Doux (1998) objects to this conflation as it either requires us to conceptualize emotions and emotional behaviour as 'cognitive' or leaves emotions out of the study of the human mind altogether. Is emotion a kind of cognition? Le Doux doesn't believe so. On his view emotion and cognition are best thought of as separate but interacting processes mediated by separate but interacting brain systems. To obviate any heated debate over this claim based on semantics, Le Doux points out that scientific evidence shows that when the hippocampal system is damaged the person loses the ability to remember a traumatic experience, as we have seen in the above cases. However, emotional memories are left intact as evidenced in the expression of bodily responses that prepare for danger (e.g. avoidance behaviour). Conversely, when a certain region of the brain, the amygdala, is damaged, humans (and other animals) lose the capacity to respond to danger. In the normal undamaged brain:

> These two systems operate in parallel and store different kinds of information relevant to the experience. And when stimuli that were present during the initial trauma are later encountered, each system can potentially retrieve its memories. In the case of the amygdala system, retrieval results in expression of bodily responses that prepare for danger, and in the case of the hippocampal system, conscious remembrances occur. It is very helpful to keep the workings of the declarative system separate from other memory systems when considering how anxiety disorders might arise and be maintained (Le Doux, 1998:239).

Thus, as Zajonc (1984) argued, the study of emotion and cognition should be kept separate.

If we want to understand the neurobiology of hysteria, it seems we need to know something about the amygdala system and how, through classical conditioning a threatening stimulus, such as an hostile caregiver, activates this system unconsciously, triggering a freeze response or submission—the two strategies identified as being relevant to conversion reactions.

4.3 The neuroanatomy of fear

The amygdala is a small, almond-shaped region in the forebrain which has long been considered important for various forms of emotional behaviour. It is here that information about innate and conditioned stimuli are encoded in the form of associative memories (Le Doux, 2000; Corcoran and Quirk, 2007). In support of this claim, lesions made to the amygdala have been found to eliminate the ability of rats to learn fear associations, and lesions made after conditioning abolish previously learned fear responses, as well as fear responses to a predator (Corcoran and Quick, 2007). In other words, amygdala lesions are found to interfere with fear conditioning. As Le Doux states:

> In the presence of a fear-arousing stimulus, and the absence of amygdala activation (for example, if your amygdala were damaged) you might use your cognitive powers to conclude that in situations like this you usually feel 'fearful' but the fearful feelings would be lacking because of the importance of amygdala inputs to working memory, of amygdala-triggered arousal, and of amygdala-mediated responses that produce feedback (Le Doux, 1998:298).

In the early days of neuroscience, the amygdala was considered part of the limbic system and the term is still in use today to denote regions of the brain dedicated to the processing of emotional stimuli and the execution of defense behaviours. Structurally, the limbic system was conceptualized as distinct from the more recently evolved cortex, home to reason and higher thought processes, as the associated term 'sub-cortical' implies. It was thought that the structures below the

cortex were 'slaves' to their cortical 'master' (Le Doux, 1998). Congruent with this view, neuroanatomy textbooks depicted the flow of information from the sensory processing structures as being directly to the cortex where all the interesting things were done to the stimulus, such as assembling data into perceptions of the external world that we experience (Le Doux, 1998).

However, it turned out that this traditional view was wrong. First and foremost, sensory signals are projected directly to the amygdala, and cortical processing only contributes indirectly to fear conditioning by providing more detailed representations of the sensory array. Le Doux (1996, 1998) refers to these two routes as the 'direct' and 'indirect' routes respectively. The dual route model of fear conditioning has been the subject of considerable study and as it is relevant to the case for understanding conversion reactions, will now be considered.

4.4 The dual route model of fear conditioning

In a series of animal experiments conducted during the late 1980s and extending through to the 1990s, Le Doux and his colleagues identified the passage taken by an auditory signal in the nervous system of rats that had been fear conditioned to the sound of a buzzer (Le Doux, 1998). They had hypothesized that if the traditional view was correct the flow of auditory information would be directly to the cortex. Instead, it was found that the target was subcortical regions including the region now identified with fear conditioning—the amygdala. Disconnection of the auditory thalamus from the amygdala prevented conditioning from taking place. However, lesions made to the auditory cortex did not interfere with the process.

Le Doux concluded from this series of experiments that the auditory stimulus had to rise through the auditory pathway from the ear to the thalamus and then proceed to the amygdala and associated structures, but does not have to travel the distance to the auditory cortex in order to generate a fear response. Thus an amygdala-initiated response to a stimulus can occur before the cortex has begun

to process such information.

The dual route theory has served as inspiration for modeling fear conditioning using neural networks (e.g., Armony, et al., 1995; den Dulk, et al., 1998; Balkenius & Moren, 1988).

4.4.1 Connectionist simulations with a dual route model of fear conditioning

After many years of being ignored, computational approaches to understanding fear conditioning and emotions in general have recently been taken by cognitive scientists. This has largely been due to the rise of connectionism, a branch of cognitive science that is particularly suited to studying the neural dynamics of adaptive behaviour.

Although many different models have been generated, it is generally assumed by connectionists that neural network architecture is either of a feedforward or feedback type, and which is implemented depends on the problem the researcher is investigating.

'Feedforward' is a term used by connectionists to describe a system which reacts to changes in its environment in a predefined way, usually, in order to maintain some desired state of the system. In a feedforward network, a pattern of activation, representing a stimulus, flows unidirectionally from input to output. Connectionists use this type of model to train a network to associate a set of arbitrary patterns presented on the input with a corresponding set of patterns presented at the output.

By contrast, in interactive, or feedback networks, nodes have feedback relations among each other. That is, the flow of activation is not one way as in feedforward networks, rather a node 'a', sends activation to node 'b', and node 'b', in turn, sends activation to node 'a' (Read & Miller, 1998). The activation of nodes is updated many times as the system moves towards an equilibrium state which

constitutes an 'if...then' rule. Many of our modern domestic appliances are operated by such systems. For example, an air-conditioner might use five rules which act on the motor in order to maintain a specified temperature, e.g., 'if cold then stop'; 'if cool then slow', etc. Another example is the 'cruise control' system fitted on some late model motor vehicles. If in use, this mechanism enables the vehicle to maintain a steady road speed. When an uphill stretch of road is encountered, the car slows down below the set speed; this speed error causes the engine throttle to be opened further, bringing the car back to its original speed.

A feedforward system, on the other hand, would in some way 'predict' the slowing down of the car based on past and existing data. For example, it could measure the slope of the road and, upon encountering a hill, open up the throttle by a certain amount in anticipation of the extra load. The car does not have to slow down at all for the correction to be made. However, other factors than the slope of the hill and the throttle setting influence the speed of the car: air temperature, pressure, fuel composition, wind speed, and so on. Just setting the throttle based on a function of the slope may not result in constant speed being maintained. Since there is no comparison between the output variable, speed, and the input variable, it is not possible to resolve this problem with purely feedforward control.

The two types of control are not mutually exclusive; the feedforward system just described could be combined with the feedback system correcting for any error in the predetermined adjustment made by the feed-forward system.

Is something like this happening in our brains? A feedforward architecture would provide an organism with what Le Doux (1998) calls a 'quick and dirty' form of information processing analogous perhaps to the direct thalamo-amygdala pathway. This direct pathway allows the organism to begin to respond to potentially dangerous stimuli before it fully knows exactly what the stimulus is which can be very useful in dangerous situations.

A feedback architecture would correct for errors made by the feedforward system, just as the cortex appears to correct (in normal brains) an inappropriate response initiated by the amygdala. For example, a person might jump out of the way of a coiled rope thinking it was a snake, but a more detailed representation projected from the visual cortex to the amygdala would constitute the person realizing that what he thought was a snake was, in fact, a coiled rope.

Some researchers hypothesize that feedforward and feedback systems interact in the cerebellum to help co-ordinate our movements. The cerebellum is thought to generate a prediction of what is going to happen given a motor command to say, pick up a carton of milk. The prediction is based on information about the current global position of the body *vis-à-vis* the carton, and other salient variables. The model or simulation can be updated and improved upon when feedback finally arrives to tell the system what actually happened on the basis of the prediction (Wolpert, Miall, & Kawato, 1998:338).

Consistent with the above rationale, computational models have been built by researchers that explore the interaction between converging thalamic and cortical inputs onto neurons in the lateral nucleus of the amygdala (Armony & Le Doux, 1997), as well as the role of local feedforward and feedback inhibition in stimulus processing (Li, et al., 1996).

Generally speaking, these models confirm the results from animal studies, namely that the direct route between the thalamus and amygdala seems more important for learning relatively crude associations between an unconditioned and conditioned stimulus. Furthermore, simulated lesions to the indirect path after conditioning revealed that almost the entire conditioning effect remained, whereas it almost completely disappeared after lesioning the direct path. As suggested by animal research, the indirect route (from thalamus through the cortex to the amygdala) seems necessary for learning more detailed and specific associations.

It may well be the case that subjective, consciously experienced fear, is a mental state that occurs when the defense system of the brain (the system that detects threats and organizes appropriate physiological responses) is activated, but only if that brain also has the capacity for consciousness (Le Doux, 1998). On this view, also shared by Damasio (1994), feelings (in this example, fear feelings) occur when the system responsible for awareness becomes privy to the activity occurring in unconscious processing systems. As Le Doux (1998:19) puts it:

> What differs between the state of being afraid and the state of perceiving red is not the system that represents the conscious content (fear or redness) but the systems that provide the inputs to the system of awareness. There is but one mechanism of consciousness and it can be occupied by mundane facts or highly charged emotions.

4.4.2 The philosophical implications of the dual route model of fear conditioning

Continuing research on the neural substrate of fear conditioning confirms and expands on the pioneering work of Le Doux and his colleagues. We can say, with reasonable confidence, that emotional learning and behaviour can take place without the assistance or intervention of higher cognitive processes responsible for reasoning, reflection and conscious thought. In other words, contrary to the idea that emotions such as fear are contingent upon beliefs or conscious judgments, the opposite appears to be true; fear is primarily a specific response to the perception of danger in systems that function outside of conscious awareness.

The operation of implicit and explicit memory systems means that a person can be afraid of riding an escalator but simultaneously judge that escalators are not fear eliciting mechanisms without there being any contradiction or inconsistency. As Hume (1740) observed long ago, a man suspended in a cage over a precipice may be very afraid, although he knows at another level that he is perfectly safe.

In his critique of propositional attitude theory Griffiths (1997) points out that research in cognitive psychology and neuroscience requires us to postulate

information-processing mechanisms that operate parallel to those responsible for the formation of reportable (explicit) beliefs. However, on the view we have been examining it is the emotional unconscious that is responsible for fear feelings, rather than the cognitive unconscious.

From a philosophical perspective, these neurobiological findings render inadequate the view of emotions as beliefs or normative judgments promoted by propositional attitude theorists (Griffiths, 1997). Any theory of emotion that is based solely or primarily on introspectively accessible aspects of the mind is necessarily incomplete given the fact that much emotional processing occurs unconsciously. It is also likely to lead to false premises regarding the causes of emotions and emotional behaviour. Systems involved in the processing of responses to an emotional stimulus are likely to include historical data not necessarily available to the introspective mind. For example, early socialization experiences with caregivers and significant others that occurred before explicit memories of emotional events became accessible to higher cognitive systems responsible for the ability to introspect, are factored into an emotional response. A father who often has angry feelings towards his children and frequently abuses them, may not recall his own mistreatment as a child and rationalize such behaviour by saying that the children were misbehaving.

According to Bargh, a goal of social psychology should be to make people aware of such unconscious influences that affect our thoughts, feelings and behaviours. However, he admits that this is an uphill battle: 'Inasmuch as people check such a proposition against their own phenomenal experience to test its validity, we will never be persuasive, because by definition one can never have a phenomenal experience of perception without awareness' (Bargh quoted in Le Doux, 1998).

Other factors such as a person's self-concept may also prevent him from considering information that is inconsistent with the way he perceives himself. This notion was at the heart of Freud's theory of defense mechanisms such as

rationalization. Scientific evidence for the existence of an emotional unconscious which influences our thoughts, feelings and behaviour often in ways that are threatening to the conscious self, corrects the Freudian concept of unconscious ideation as a cause for this type of confabulation.

4.5 Emotions and homeostatic regulation

The dual route model of fear conditioning explains why we humans seem to have so little control over our emotion feelings, thoughts and behaviour. As we saw in Chapter Two, the processes we call emotions are intimately connected to homeostatic regulation. As Damasio (1994) says, it would not be advantageous to the biological integrity of the organism if conscious systems had the ability to directly interfere with these finely tuned physiological processes:

> At their most basic, emotions are part of homeostatic regulation and are poised to avoid the loss of integrity that is the harbinger of death or death itself, as well as to endorse a source of energy, shelter or sex. And as a result of powerful learning mechanisms such as conditioning, emotions of all shades eventually help connect homeostatic regulation and survival "values" to numerous events and objects in our autobiographical experience. Emotions are inseparable from the idea of reward and punishment, of pleasure and pain, or approach and withdrawal, of personal advantage and disadvantage, inevitably emotions are inseparable from the idea of good and evil (Damasio, 1999:54).

This may be why synaptic connections from the emotional systems to the cognitive systems are stronger than those from the cognitive systems to the emotional systems (Le Doux, 1998). Although humans can deliberately set up situations in order to modulate their emotions such as viewing scary movies, riding a roller coaster, eating tasty foods, consuming alcohol and other drugs, it is extremely difficult to convincingly induce an emotion without participating either directly or vicariously in an emotion-eliciting event. Emotions are things that happen to us rather than things we will to occur (Le Doux, 1998).

If the workings of these phylogenetically older systems in the brain are integral to survival it may also help explain why extinguishing fear memories is notoriously

difficult.

4.6 Extinguishing learned fears

Extinguishing learned fears is the bread and butter of psychotherapists who use various techniques to accomplish this end, as previously discussed. However, extinction, despite the claims of some therapists, does not mean the neurotic fear has been erased. The retention of the original association can be uncovered by a variety of maneuvers including changing the context, stressing the animal, or simply allowing time to pass (spontaneous recovery). As Le Doux, 1998:252 points out: 'Unconscious fear memories established through the amygdala appear to be indelibly burned into the brain. They are probably with us for life'.

The idea that extinction does not involve the erasure of emotional memories is consistent with a number of findings about conditioned responses in general. For example, Pavlov found that extinguished responses would simply return with the passage of time. Freud and other 19th century clinicians similarly noted that unbidden memories of traumatic events might return as physical sensations, horrific images or nightmares, behavioural re-enactments, or a combination of these. Modern theorists would explain these phenomena as manifestations of dedicated systems in the brain responsible for reproducing emotional memories and memories of emotional experiences.

In animal research it has been repeatedly found that the CR to a CS can be restored after extinction by lesioning the indirect route, or by inducing a dysfunction of the system (Le Doux, 1996). This suggests that cortical systems are involved in extinction where extinction simply entails inhibiting the output initiated by the amygdala rather than the complete eradication of the memory. One area of the brain that seems to be involved in the extinction or inhibition of a conditioned response to fear stimuli is the orbitofrontal cortex (OFC) implicated in hysterical paralysis (see section 1.5). (The descriptions 'ventral' and 'medial' are also used in the literature when referring to the OFC (e.g., Damasio, 1994;

Rolls, 1999) to distinguish this area of the frontal lobes from the more dorso-lateral parts identified with the perception of conscious voluntary control of actions. When discussing experiments relating to this region of the frontal lobes, I will use these terms interchangeably).

The amygdala-OFC system is strategically placed close to the higher cortical areas that receive sensory, including somatosensory information, and is also near to the various regions constituting the basal ganglia that are involved in the reinforcement of motor actions (Gray, 1995; Rolls, 1995). Thus, it is in an ideal position to mediate behaviour according to changes in the environmental stimuli (internal and external) associated with reward and punishment.

While both the amygdala and the OFC are involved in operant conditioning, the amygdala does not appear to provide such rapid relearning of reward-related emotional responses to stimuli as does the OFC (Rolls, 1999). As we shall see in the next chapter, the OFC has been shown to be involved in the maintenance of conversion reactions (CS) that have been operantly conditioned.

Whilst identifying the neural correlates of fear conditioning and defense behaviours is an important step in bridging the mind-body gap currently obscuring our understanding of hysteria, it tells us nothing about the neurochemical changes that are responsible for the distorted body representations which cause the individual to believe she is suffering from a neurological disease. For this we require knowledge of the effects of peptides such as hormones and neurotransmitters as they go about their work of preparing the body for defense.

4.7 The neuroendocrinology of fear

Output from the amygdala and OFC is directed not only to facilitation or inhibition of motor responses to CS but is also involved in producing autonomic and endocrine responses (Rolls, 1999; Damasio, 1994; Le Doux, 1998). A growing body of current evidence shows that repeated, prolonged, chronic stress

often precedes affect dysregulation characterized by somatoform disorders and other co-morbid conditions (Perry, 2001; Schore, 2003; Sapolsky, 1998). Thus, in addition to familiarizing ourselves with the relevant brain structures and functions implicated in conversion reactions, we need to know something about the structure and function of the autonomic nervous system (ANS) and the chemical messengers involved in the various physiological changes to threat.

4.7.1 The ANS and arousal states

The ANS is divided into two branches, the sympathetic and parasympathetic. These two components of the ANS are known to be distinct modular circuits that control arousal expressions, with the catabolic sympathetic branch being responsible for energy-mobilizing excitatory activity, and the anabolic parasympathetic branch involved in energy-conserving inhibitory activity. The sympathetic branch is activated by any stimulus above a species-specific arousal threshold, and functions to increase arousal, trigger an immediate anticipatory state, and rapidly mobilize resources in response to appraised stressors (Shore, 2003).

During the lifespan of the organism an autonomic mode of reciprocal sympathetic-parasympathetic control would be expressed in an organism that responds alertly and adaptively to a personally meaningful stressor. Specifically, the stress response occurs when conditioned fear stimuli are processed by the amygdala and associated systems. The activation of these systems triggers the hypothalamus which produces corticotrophin-releasing factor (CRF). CRF is sent to the pituitary gland causing the release of adrenocorticotropic hormone (ACTH) which, in turn, results in the release of a steroid hormone from the adrenal gland. The adrenal hormone then travels back to the brain. This cycle of activity that occurs routinely in response to threatening stimuli helps the body to deal with the stressor (Le Doux, 1998).

In the normal course of events, when a stressful episode is over, the defense

response is extinguished because, as Le Doux (1998) says, such behaviour is no longer appropriate (rewarded). As noted, the right hemisphere OFC (RHOFC) is responsible for extinction in response to changing external circumstances. Indeed, in the processing of emotional stimuli (rewarding or punishing) the RHOFC acts as a major centre of CNS control over these two branches of the ANS (Shore, 2003).

As soon as the context is appraised as safe, the organism immediately returns to the relaxed state of autonomic balance expressed in a slowing of the heart rate, relaxation of muscles, lowered blood pressure, and capillary constriction. These operations also allow for breathing to return to normal rates, increases in digestion, onset of bowel and bladder activities, and re-establishment of immune functions suspended during the stressful event (Shore, 2003).

4.7.2 Chronic stress and affect dysregulation

Unfortunately, for many individuals who are raised in dysfunctional family environments, the presence of constant threat does not allow the ANS to achieve a balanced state of arousal and symptoms of hyperarousal and hypoarousal may manifest in anxiety-related disorders or disorders of inhibition respectively (Perry, 2001; Crittenden, 2006). As we shall see in Chapter Seven, according to Kozlowska (2007) these different extreme responses to threat have their phylogenetic origins in innate defense strategies common to many animal species—immobility and submission. I shall argue that both responses predominantly involve a sustained opioid-mediated inhibition of behaviour.

While it is not difficult to envisage how immobilization (freezing) might be displayed in disorders of inhibition, submission appears to lead to different behaviours depending on the circumstances, particularly the pattern of interaction developed between caregiver and child. For example, submission may be expressed in appeasement displays if such behaviour has been reinforced. In other instances, submission may result in displays of 'learned helplessness' if attempts

at appeasement are unsuccessful (Miczek, et al., 1990). Here the individual just 'gives up' and may show signs of depression and other co-morbid conditions associated with hypoarousal states (Perry, 2001). Although such states are currently classified in the DSM-IV as various disorders, on this view, they should perhaps be understood as having their origins in submission characterized by defeat.

For example, a chronically traumatized child who cannot escape from the constant threat of physical, emotional, or psychological pain might enter a state of chronic hypoarousal characterized by the signs and symptoms physicians have come to associate with dissociative disorders such as hysteria. As Perry (2002) says, if a child stays in a state of hypoarousal for a sufficient period of time, she will alter the homeostasis of the systems mediating the response. A sensitized neurobiology of dissociation will result and she may develop prominent dissociative-related symptoms (e.g., withdrawal, somatic complaints, helplessness, dependence) and related disorders (e.g., somatoform disorder, major depression).

In the neonate, hypoarousal always follows a hyperarousal response to an unconditioned fear stimulus which Perry (2001) calls the 'alarm reaction'. If this response is not rewarded, that is, if, for example, the infant's caregiver does not respond to his signals of distress or responds with anger or physical abuse, 'the converse of use-dependent development occurs, namely, there is a disuse related extinction of the hyperarousal response and an initiation of a parasympathetic response' (Perry, 2001). This hypoarousal response is well characterized in models of animal stress reactivity. For example, studies show that the defeat response is often observed in subordinate primate males—those who have fought and lost to an alpha male and are forced to live a life of subjugation and constant threat from which they cannot readily escape given their dependence on the group for survival. These animals can be negatively differentiated from dominants on all biophysical levels: behavioural, emotional, physiologic, and neuroendocrinologic (van der Kolk, 1989).

4.7.3 The opioid theory of conversion disorder

In section 1.3 we saw that Damasio (2003) also hypothesizes that some 'so-called' hysterical conversion reactions that allow patients not to feel or move parts of their body and several 'somatoform' disorders can be explained by the endogenous opiate theory, although he offers little in the way of evidence. Damasio believes that these molecules modify the body maps that portray pain and fear (Damasio, 2003:115).

We can envisage these body maps as a set of correspondences from all parts of the body toward the body-sensing regions in the brain. As far as the conscious mind is concerned there is only one source of such information, namely, the pattern of activity present at any given moment in these body-sensing regions of the brain. Consequently, any interference with this mechanism by high levels of opiates can create a 'false' map of what is transpiring in the body at a given moment. The brain effectively eliminates from the central body maps the pattern of activity that would permit the experience of pain or aversive emotions (Damasio, 2003).

On this view, the patient suffering from dissociation disorder and/or some hysterical symptoms that involve the inhibition of emotional reactions, would actually hold the false belief that they are physically incapacitated, when, in fact, their condition is due to the brain filtering out body signals and/or negative affective states through the production of these morphine-like chemicals (refer to section 3.5). Although these individuals are not suffering from a neurological disease, their symptoms are 'real' in the sense that an organic explanation can be provided which justifies their perception of incapacity but not their interpretation or the interpretation of others based on the medical model.

4.7.4 Evidence for the opioid theory of conversion disorder

Further evidence that endogenous opioids might be producing some dissociative

type symptoms and sensory disturbances observed in cases of hysteria derives from the use of opioid receptor blocking agents. Dissociation and stress-induced analgesia is readily reversible in animals by use of the opioid receptor blocker naloxone (van der Kolk, 1989; Pert, 2003). Bohus et al., (1999) also found evidence of a role for this drug in treating dissociative symptoms in the context of borderline personality disorder; a condition often observed to co-occur with hysteria (Noyes, et al., 2008). A highly significant reduction in the duration and intensity of dissociative phenomena and tonic immobility as well as a marked reduction in analgesia was observed during treatment with naloxone (see also Nuller, et al., 2001).

More recently, Simeon & Knutelska (2005) found there was an average 30% reduction in dissociation and depersonalization symptoms among 14 sufferers of depersonalization disorder after being treated with this drug. Conversely, it is of note that the selective kappa opioid receptor agonist enadoline has been found to induce depersonalization in addition to numerous other physical and psychological effects (Walsh, et al., 2001).

To date, the hypothesis that the opioid system may play an important role in the pathogenesis of dissociation and, by implication, some hysterical symptoms, has not been explored by many researchers (Medford, et al., 2005; Simeon & Knutelska, 2005). This is surprising given that there is no recognized pharmacotherapeutical treatment for such disorders.

4.7.5 Endogenous opioid addiction and maladaptive behaviour

Chronic high levels of stress-induced opioids may also lead to opioid addiction in some individuals, in the same way as individuals using exogenous opiates like heroin can become addicted to such substances. Synthetic drugs such as cocaine and heroin exert extreme control over behaviour, over-riding the organism's natural reward systems so that the person's actions become maladaptive. In his paper *The Compulsion to Repeat the Trauma*, van der Kolk, (1989), sees the

endogenous opioid system as being involved in the perpetuation of a wide range of self-destructive behaviours:

> War veterans may enlist as mercenaries, victims of incest may become prostitutes, and victims of childhood physical abuse seemingly provoke subsequent abuse in foster families, or become self-mutilators. Still others identify with the aggressor and do to others what was done to them. Clinically, these people are observed to have a vague sense of apprehension, emptiness, boredom and anxiety when not involved in activities reminiscent of the trauma (van der Kolk, 1989).

The observed tendency for the victim of domestic violence to repeatedly return to her abuser is often a source of frustration, anger and despair for her family, friends and concerned professionals. Yet, seen from the perspective of the opioid theory, the abused and abuser can be compared to the heroin addict and her supplier. Leading a 'normal' moderately stressful life would be, for the person so addicted, tantamount to drug withdrawal, possibly leading them to seek out the very conditions that caused their pathology in the first place. Thus, a vicious cycle might be established which locks the victim into his pathological behaviour (van der Kolk, 1989).

Recall that in the process of constructing a clinical profile on the hysteric, Breuer also observed that his patients not only suffered feelings of apprehension and anxiety as one might expect, but paradoxically they reported a sense of boredom and restlessness. According to Breuer, this made them 'incapable of bearing a monotonous life and causes their need for sensation...' (Breuer & Freud, 1895:243). The above considerations might provide us with insight into another more insidious reason why hysterical symptoms are maintained. It is possible that people who respond to trauma by entering into hypoarousal states become 'addicted' to high levels of endogenous opioids.

The extreme inhibition of behaviour by implicit systems involved in reward, exemplified by a deterioration in the ability of normal rewards to govern behaviour, is referred to as 'motivational toxicity' (Bozarth, 1994) and is a distinguishing feature of addiction. Does hysteria constitute a pathological control

of behaviour? Might it be the case that endogenous stress-induced opioids quickly and uniformly exert extreme control over behaviour in a similar way to cocaine and heroin?

Although the neural mechanisms responsible for this disruption are slowly being identified, the dynamics are not well understood. However, put simply for the moment, endogenous opioids appear to gain their addictive qualities by acting on the brain's reward systems normally used to guide the organism toward adaptive behaviour. In other words, stress can, in some circumstances, cause the disruption of operant conditioning by rewarding behaviours that lead to aversive outcomes such as self-mutilation or death by hysterical vomiting. Stress-induced opioids seem to have this effect because they activate a brain mechanism involved in appetitive motivation and reward. The opioid theory offers a plausible explanation for maladaptive behaviours, that is, actions which seem to have predominantly unfavourable consequences yet may persist despite conscious intentions to the contrary.

In the laboratory brain stimulation experiments have been conducted on rats with similar effect. For example, laboratory animals will lever press at high rates (greater than 6,000 times per hour) in order to obtain brief stimulation pulses to certain brain regions. The reinforcement from direct electrical activation of this reward substrate is more potent than other rewards such as food or water. The potency of this electrical stimulation is most dramatically illustrated in a classic experiment where animals suffered self-imposed starvation when forced to make a choice between obtaining food and water or electrical brain stimulation (Routtenberg and Lindy, 1965). Electrical brain stimulation reward, psychomotor stimulant reward and opiate reward all appear to involve activation of the same system.

A more trivial example of the effects of opioid addiction might be the love of potentially life-threatening, extreme sports which seems analogous to the self-

stimulating lab rat that ignores internal and external punishers normally avoided, in order to obtain a stress-induced release of opioids. As Bozarth (1994) observes, the stress-induced release of these peptides offers an explanation for such risk-taking behaviour.

4.8 The neurobiology of social pain

Our discussion may help to solve another paradox raised in the literature on the neurobiology of social pain.

In both infrahumans and humans, relational devaluation and rejection from conspecifics has been found to induce varying degrees of analgesia (or reduced pain sensitivity), which has been attributed to increases in endogenous opioids (MacDonald & Leary, 2005; Panksepp, 2003; Eisenberger, et al., 2003). In humans the increased release of endorphins under the influence of the OFC has been observed to decrease the pain of perceived social rejection (Eisenberger, et al., 2003). Recall that in section 1.3 it was noted that opioids have not only been shown to block physical pain signals but are also believed to be instrumental in ameliorating socially induced emotional and psychological pain (Panksepp, 2003; MacDonald & Leary, 2005). Their release in response to signals emanating from damaged tissue is obviously adaptive. If an animal is wounded but has no sensation of pain, its chances of escaping from a predator and hence for survival are enhanced.

Social pain theorists have shown that there is strong evidence from animal research that opioids are involved in signaling the adequacy of social conditions, with low levels of opioids signaling an unsatisfactory social environment and motivating the pursuit of social interaction. For example in primates, low opioid levels motivate social behaviours such as grooming of conspecifics (Martel et al., 1995) and increase the crying behaviour of isolated rat pups (Carden, et al., 1996; Carden & Hofer, 1990).

At first, this finding may seem to contradict the thesis that conversion reactions indicate a high level of opiate production. MacDonald & Leary (2005) also report this type of conflict in the social pain data, with some researchers showing that perceptions of social support are associated with reduced levels of social/physical pain. Other findings suggest that isolation leads to analgesia in non-human animals and that exclusion can lead to decreased pain sensitivity for hurt-prone humans. These authors ask: 'Why would both inclusion and exclusion lead to decreased pain sensitivity?'

One answer might be that like all endogenous chemicals, the production of opioids is kept in balance through the process of homeostasis. In normal quantities, opioids play an important role in facilitating and maintaining social attachment behaviour. The brain circuits involved in the maintenance of affiliative behaviour are precisely those most richly endowed with opioid receptors (Pert, 2003; MacDonald & Leary, 2005). An animal may be 'rewarded' by a release of 'feel-good' opioids if it engages in social behaviour, and 'punished' by a drop in opioid levels if it withdraws from social interaction. Therefore, in the normal course of things, opioids, along with other endogenous chemicals such as oxytocin, regulate social behaviour (Damasio, 1994). However, just as extreme pain leads to analgesia and concomitant diverse psychological and physical effects so, too, extreme social pain induced by exclusion, seems to have a similar result. In other words, we need to avoid the tendency to think that 'more is better' and focus on balance when it comes to examining self-regulatory processes. On this interpretation of the data, there is no conflict.

4.9 Neuroimaging techniques and psychopathology

Just as there appears to be a general lack of interest among medical professionals regarding the neuroendocrinologic aspects of conversion reactions, a number of researchers have observed this same disinterest regarding findings from neuroimaging studies carried out with patients diagnosed with conversion disorder (Anthwal, et al., 2001; Spence, 2000).

Brain imaging has yielded a plethora of neural correlates of affective states such as human sympathy, the ecstasy of peak musical experiences, the response to breathlessness, and feelings of social pain which we have discussed in the context of the defeat response. Recent developments have also contributed valuable insights into the brain areas activated in functional conditions such as depression, auditory hallucinations, post-traumatic stress disorder, schizophrenia, and obsessive-compulsive disorder (Anthwal, et al., 2001). A detailed discussion of neuroimaging techniques such as PET (positron emission tomography) and fMRI (functional magnetic resonance imaging) is beyond the scope of this present work but useful reviews of this technology may be found in Carson et al., (1998) and Frackowiak et al., (1997).

Thus, the advent of functional brain imaging has made it possible, in principle, to localize symptoms, or task-dependent psychological processes of human cognition and affect in the living brain. The observed reticence of medical professionals to explore these avenues of enquiry is difficult for those outside of the profession to understand given the lack of a biomedical explanation for conversion reactions has limited progress in its understanding (Jureidini & Taylor, 2002).

Another reason why neuroimaging studies should be of interest to clinicians is the possibility they offer for discriminating between patients who are genuinely experiencing symptoms and those individuals who are feigning. At present, the standard diagnostic criteria for hysteria as laid down in the DSM-IV requires that the physician infer whether the symptom presented by the patient is intentionally produced or feigned. Conscious simulation of motor or sensory loss is possible and often very difficult to distinguish from symptoms of hysterical conversion. As Spence (2001) states: "physicians can be poor judges of human deception". As mentioned, physicians often feel the need to warn their colleagues not to be 'caught out' by malingerers whose intention it is to 'fool' the doctor in order to

achieve some secondary gain (e.g., financial compensation; an excuse for not fulfilling one's responsibilities, etc.).

Although there is much excitement among theoreticians about preliminary findings as they relate to hysteria or conversion disorder, skeptics of this methodology have correctly observed that the results are often conflicting or findings are open to misinterpretation because of the complexity of neuroanatomy. Nevertheless, in general, investigators are gaining interesting insights into just how little higher cognitive systems, hypothesized to subserve reasoned decision-making, feature in our everyday actions, particularly when those actions involve systems that use associative learning and memory to guide emotional and motivational behaviour. In the following chapter I will show that the OFC, particularly in humans and other primates, plays an important role in the maintenance of innate and learned defense strategies that are rewarded.

In the present chapter I have begun to identify the brain systems and neurobiological processes underlying fear conditioning and the extinction of fear responses. Knowledge of these systems and their function is imperative if we want to understand the pathological or hysterical fear responses that appear to present as symptoms of physical disease. Many of the more traditional physical and psychological symptoms identified with hysteria, such as various analgesias and sensory/somatosensory disturbances, feelings of detachment or dissociation from the world and others, and a generally flat emotional landscape have been sourced to a stress-induced over-production of endogenous opioids.

These hypoarousal states identified with immobility, or submission, evidenced by the defeat response, have been the subject of much study in infrahumans and more recently, in humans. Preliminary investigations indicate that, as with other animals, these responses are often adopted by humans when flight or fight is not possible, that is, when a person cannot escape from threatening external stimuli or, internal mental stimuli such as thoughts and impulses associated with

punishment.

5 The functional anatomy of hysteria

So far, we have discussed hysteria and hysterical symptoms in terms of the conditioning of unlearned fairly stereotypical responses to threat. External (secondary) stimuli gain their ability to trigger such hard-wired responses by their learned association with internal (primary) aversive stimuli such as pain, or withdrawal of physical and emotional support; stimuli that threaten the organism's survival. We have seen that the system responsible for initiating such fear responses is principally the amygdala. Activation of this region of the brain by the actual presence of a CS or the rememberance of a CS triggers neuroendocrine, autonomic, behavioural and in humans (and possibly other animals to a more or less degree) experiential changes.

However, it has also been noted that although the amygdala is necessary for fear conditioning to occur there is evidence from both animal and human studies to show that the OFC is instrumental in the execution of conditioned or learned behaviours (Corcoran & Quick, 2007; Rolls, 1999). Rolls (1999) argues that this is because the OFC is capable of more rapid learning and relearning of reward-related emotional responses to stimuli than the amygdala. In primates this ability is probably very important during social interaction when reinforcing stimuli are constantly being exchanged and the reinforcement value of these must be continually updated (relearned), based on the actual reinforcers received and given (Rolls, 1999).

Corcoran & Quick (2007) would agree with Rolls but as their recent findings show, the OFC is not necessary for the execution of innate fear responses to unconditioned stimuli. For example, when the OFC is lesioned in a rat it still freezes in the presence of a natural predator. However, a CR to a CS does appear to be under the control of the OFC which regulates fear behaviour in accordance with environmental cues as Le Doux (1998) also observes in his discussion on the

extinction of a CR.

Since clinical hysteria is acknowledged to develop in the context of the social environment as a result of interactions between family members and one's social group, behaviours characteristic of this disorder could be hypothesized to involve the OFC following fear conditioning. This hypothesis appears to be supported by findings from neuroimaging studies carried out with patients diagnosed with hysterical disorders which show an activation of the OFC (Marshall, et al., 1997; Vuilleumier et al., 2001; Tiihonen, et al., 1995; Devinsky, et al., 2001; Ward, et al., 2003).

The purpose of the present chapter is to ascertain how best to interpret this finding in light of what is known about the structure and function of this region of the prefrontal lobes and the principles of learning theory. At first glance, hysterical reactions appear to be over-learned or habitual responses that were once adaptive inasmuch as such strategies protected the individual from harm inflicted upon them by aggressive caregivers. Without clinical intervention aimed at changing the behaviour of both caregiver and child, these individuals may continue to present with chronic symptoms that within a dualistic medical framework cannot be explained.

The inappropriate perpetuation of such pathological or abnormal behaviours may be no more difficult to understand than the familiar phenomenon of addictive behaviour as suggested previously. People often habitually engage in harmful behaviours such as excessive alcohol and food consumption and, despite their best intentions, fail in their efforts to moderate such habits. We often attribute addictions to a lack of 'will-power' or a weakness of character, believing the person could change his or her behaviour if they really tried. So too, hysterics have been accused of lacking moral fortitude. However, in both instances, we could hypothesize that the unwanted behaviour is dominated by what Bozarth (1994) calls 'super-potent reward', a phenomenon that has been attributed to the

reinforcing properties of endogenous opioids (section 4.9). Thus, when a person tries to move her 'paralyzed' limb, her intention may be over-ridden by the OFC acting under the influence of opioids.

Early on in his career, Freud described this intrapsychic conflict as a 'clash' between the 'anti-will' and the 'will' and later as conflict between instinctual impulses (the id) and inhibiting external and internal constraints (the superego and ego respectively). Contemporary cognitive scientists propose that there is often conflict between implicit (automatic) and explicit (controlled) processes. Whereas the former is able to guide behaviour particularly to obtain the greatest *immediate* reinforcement, the explicit system can potentially enable immediate rewards to be deferred (Rolls, 1999). As explained, the person who continues to manifest a CR (hysterical symptom) in the absence of the CS appears to do so under the influence of potent endogenous chemical substances. Thus, understanding the neurobiology of hysterical disorders may further our understanding of the more general problem of addictive behaviours. This rationale has already been applied to other self-destructive behaviours of an interpersonal nature in section 4.9.

Conscious control over entrenched fear behaviours is not simply a matter of exerting self-control. It is a complex and often unsuccessful process undertaken by the prefrontal lobes.

As with most of our knowledge about the function of different brain regions, information regarding the type of processing likely to occur in the OFC and the frontal lobes (called prefrontal in the literature) in general, derives mainly from studies carried out on infrahumans and humans who have suffered damage to these areas. As we have seen in the case of H.M., from time to time neurologists have deliberately created lesions in the human brain in order to ameliorate the effects of various neurological disorders. Unfortunately for the patient these procedures are not without side effects and these effects tell us something about the importance of the lesioned area for normal brain function.

5.1 The prefrontal lobes and executive functions

Much of what is known about the prefrontal lobes, which are particularly enlarged in humans, began to emerge in the early part of the 20th century following surgery aimed at treating overwhelming anxiety, irrational fears, and emotional hyperexcitability (Springer & Deutsch, 1993). Although the evidence is acknowledged as being difficult to interpret, damaging or disconnecting parts of the prefrontal cortex, referred to as a *lobotomy*, provided a number of insights into the importance of this region for emotion and cognition. Moniz (1939), whose name is most often linked with this experimental surgery, operated on twenty patients and published an enthusiastic report on his findings which rapidly led to widespread use of the procedure. More than twenty thousand patients were subjected to prefrontal lobotomies of varying extent during the next fifteen years. Although irrational anxiety or emotional outbursts were sometimes controlled, intellectual deficits and other side effects were often apparent (Rylander, 1948; Valenstein, 1941).

For example, although patients still seemed capable of performing many different tasks, deficits in executing sequences of operations or solving complex problems were evident. A patient would have difficulty shifting 'set' and become stuck on a task. Having completed one step properly, the patient might continue to use the same strategy in totally inappropriate contexts. This tendency is commonly referred to as *perseveration*. Such deficits suggested to psycholgists that the frontal lobes are involved in the planning and organization of actions (Springer & Deutsch, 1993).

Investigators also noted an inability to inhibit prepotent or habitual responses elicited by a problem. The personality and emotional changes associated with damage to the prefrontal lobes were even more elusive than the intellectual deficits but generally it appeared that these brain regions might also control or inhibit emotional response tendencies as anticipated from early animal studies. It

was Jacobsen (1936) who first noted that lesions made to parts of the ventral prefrontal cortex in animals resulted in them becoming calmer and less frustrated when a reward was not given than before the procedure. It was Jacobsen's findings that inspired Moniz to carry out the procedure on humans, however the lesions made were more extensive and led to the intellectual deficits mentioned above. As we shall see, damage to the dorsolateral prefrontal cortex (DLPFC), particularly the left hemisphere (LH) DLPFC can lead to *cognitive perseveration* but when it occurs in the RHOFC it may lead to what Le Doux (1998) calls *emotional perseveration* because of the inability to respond rapidly and appropriately to reinforcement changes as they occur in the animal's environment.

Le Doux (1998) believes that in neurotic disorders the OFC may be malfunctioning. Like the rat with lesions to this area of the brain, the amygdala of the neurotic human stubbornly expresses its fear memories in the face of information that the stimulus is no longer associated with danger. However, as the evidence suggests, the explanation may be more complicated. The OFC may well be malfunctioning under the influence of a habitual parasympathetic response but the amygdala does not appear to be involved according to neuroimaging studies.

Perhaps this anomaly can be explained by the fact that Le Doux (1998) based his observations on the behaviour of lesioned rats in which the OFC and the prefrontal lobes in general, are not well developed. As Squire (1992) pointed out in his paper on implicit learning in rats, these animals will readily adopt a simple associational strategy while humans tend to use higher associative systems when processing emotional or cognitive information. If this is the case, we should be wary of extrapolating findings from animal studies to explain human behaviour.

Since these early findings, experimental studies have continued to show that damage to the OFC produces a wide range of emotional and motivational changes in primates. These changes appear to be related to the organism's inability to respond rapidly and appropriately to non-reward stimuli as Rolls (1999) suggests.

Various studies carried out with humans suffering damage to the OFC tend to support findings from primate experiments where the OFC is deliberately lesioned. For example, Rolls and his colleagues gave patients a visual task directed at assessing their ability to perform rapid alterations of stimulus-reinforcement associations (Rolls, et al., 1994). Patients could learn to obtain points (rewards) by touching one stimulus when it appeared on a monitor, but had to withhold a response when a different visual stimulus appeared otherwise a point was lost. That is, the subjects were 'rewarded' for making the correct response and 'punished' for making an incorrect or inappropriate response. After subjects acquired the visual discrimination (were conditioned), the experimenters unexpectedly reversed the reinforcement contingencies. The subjects with ventral frontal lesions made more errors in the reversal (or in a similar extinction) task and completed fewer reversals than control subjects with damage elsewhere in the frontal lobes or in other brain areas (Rolls, et al., 1994). In other words, when the stimulus-reinforcement contingency was changed, patients had difficulty extinguishing their conditioned behaviour.

Similarly, Bechara and colleagues found that damage to a patient's ventromedial (VM) region of the frontal lobes impaired their ability to successfully learn a gambling task (Bechara et al., 1994; see also Damasio, 1994). The patient was allowed to choose from two different piles of cards. One pile gave rewards with a reasonable probability but also had occasional very heavy penalties resulting in lower net gains than choices from the other pile. In contrast to control subjects suffering damage to other brain regions, patients with damage to the ventromedial region did not switch from the pile of cards providing significant rewards even when the large penalties were incurred. In this sense, they did not learn from the negative consequences of their actions.

Yet, according to these researchers, although VM patients could not demonstrate that learning had occurred, they were often able to verbalize the correct response:

patients seemed to know what they should have done but could not carry it through (Rolls, 1999; Damasio, 1994). According to Rolls, this dissociation is consistent with the theory that the OFC is normally involved in executing behaviour when the behaviour is performed by evaluating the reinforcement associations of environmental stimuli (Rolls, 1999).

For example, one of Damasio's patients ('Elliot') reported that he knew which deck of cards was the losing one but could not influence his actions (Damasio, 1994). We should note however, that his actions were queried by Damasio and his colleagues at the end of the trial and thus may have constituted a *post hoc* deduction based on the outcome of the game rather than an inability to affect the course of the game while in progress. As previously mentioned, psychologists and philosophers agree that humans are notorious for confabulating reasons to explain their actions after the event (see also section 3.4).

This phenomenon is most transparent in patients who have had their corpus callosum surgically severed in an attempt to ameliorate the effects of epileptic seizures. It is now well known that when the left hemisphere (LH) of the brain, dominant for language, is disconnected from the right hemisphere (RH), information provided to the RH only cannot be transferred to the LH. Yet, in a number of instances, when investigators asked a patient to provide reasons for what was actually a RH response to such information, the LH confabulated an explanation constructed from whatever external cues were available or feeling states communicated to the LH at a sub-cortical level, that is below the level of the lesion (Gazzaniga & Le Doux, 1978).

In the normal course of events people with intact brains often confabulate reasons for actions that are the direct result of implicit stimulus-reinforcer processing yet claim that the invented explanation results from direct knowledge of their own mental processes. It is suggested by a number of commentators that a substantial amount of self-report may be the product of confabulation (Griffiths, 1997). Thus,

we might anticipate that Elliot would claim to know what he should have done during the trials.

An idea of the degree to which operant conditioning mediated by the OFC plays an important role in normal behaviour and behavioural change is apparent from the behavioural ratings given by the carers of VM patients. These individuals were rated high on at least some of the following: disinhibition or socially inappropriate behaviour; misinterpretation of other people's behaviour; anger or irritability; and lack of concern for their own condition and that of others (Rolls, et al., 1994; Damasio, 1994). These observations of behavioural change in humans suffering lesions to the OFC should cause us to think that the malfunctions that occur in hysteria are the not due to *disinhibition* caused by a lack of OFC control over the amygdala. Quite the opposite—neuroticism and, in particular hysteria, is most often associated with fears about acting *inappropriately* leading to disorders of *inhibition* rather than *disinhibition.*

Neurotics, as Breuer & Freud (1895) suggested, are overly concerned about improprieties and are anxious to please significant others, behaviours that are consistent with a family history of over-control and often harsh punishment. As Kozlowska (2003, 2007) has similarly observed, children presenting with pseudo-neurological complaints are 'good children' who attempt to please their overly demanding parents.

One way of explaining what is going on in these individual's brains is to appeal to the influence of cognition in contributing to OFC responses to rewarding or punishing stimuli. Although Damasio and his colleagues do not consider the possibility that at least some of the behaviour displayed by OFC patients may be due to the inability of the LHDLPFC to influence emotional behaviour, we should examine this prospect. For example, if patients had received their brain damage in a road traffic accident and financial compensation had been awarded, they often tended to spend the money without appropriate concern for their future needs. The

blatant disregard for their future financial security may be interpreted as a failure of the DLPFC to influence prepotent responses aimed at obtaining the most immediate rewarding outcome (Rolls, 1999). While Damasio (1994) claims that VM frontal lesions are responsible for the inability of patients to be guided by future prospects rather than by immediate outcomes, Rolls (1999) argues that the DLPFC can potentially enable deferment of immediate rewards by influencing the OFC in ways we will discuss in the following two sections.

Another way the LHDLPFC can influence the OFC, which is pertinent to our understanding of the role of conscious cognition in contributing to abnormal fear and therefore hysteria, is through imagining the possible future repercussion of a proposed behaviour. If the imagining is represented in the OFC it can cause what emotion regulation theorists call 'up-regulation' resulting in an intensification of the fear response (Rolls, 1999; Oschner, et al., 2005). The effect of the LHDLPFC on emotional processing is explained by Rolls (1999).

5.1.1 Dorsolateral Prefrontal Cortex

Although higher cognitive systems responsible for long-term planning, imagination, and conscious reasoning, may not be able to control behaviour directly, they have the capacity to influence implicit direct reward-based systems such as the amygdala and OFC. The language based explicit system involves a computation with many 'if...then' statements to implement a plan to obtain a reward (Rolls, 1999:258). In this case, the immediate reward may actually be deferred if a second more highly valued reward was computed to be, in the overall scheme of things, an optimal strategy in terms of resource usage (e.g. time, energy, etc.).

Rolls (1999) argues that syntax is required here because the many symbols (words) that are part of the plan must be correctly linked or bound. Such linking may take the form: 'If I do A, then B is likely to occur, and then I should obtain C'. For example, after completing an ordinary degree, a university student may

refuse a position (immediate reward) with low pay and few prospects for advancement in order to work for a higher degree which she envisages will improve her chances of obtaining a more lucrative job with greater career opportunities (more highly valued delayed reward). However, the actual computation of a particular stimulus or situation would, on this view, still depend on activity in the OFC as the reward value of stimuli is computed and represented in this region of the brain (Rolls, 1999).

An essential building-block for such planning and problem-solving tasks is short-term or working memory which enables multiple pieces of information to be held in the correct spatial and temporal sequence (Rolls, 1999). Borrowing a term from computer technology, memory researchers sometimes refer to temporary storage mechanisms in the brain as 'buffers'. It is now believed that a number of specialized buffers exist and some would be associated with aspects of language use. These buffers may help keep the first part of a plan in mind until the person works through the whole plan (Le Doux, 1998). Such short-term memories are implemented in the DLPFC. So, whereas the OFC, in conjunction with the amygdala, is directly involved in executing behaviour in response to rewarding and punishing stimuli, the DLPFC is part of a cognitive system in which explicit, declarative processing occurs but can only affect behaviour indirectly.

If this were not the case the conscious mind, or on this view the explicit system, would be able to directly act on its computation even when the OFC is damaged. Yet, as we have seen, patients with lesions to the OFC cannot execute the correct action even though they may be able to articulate what they should do. It appears that this explicit learning and memory system must direct the contents of its processing to motivational and affective systems in order to influence behaviour. As Rolls states:

> .... the explicit system may decide on a plan of action or strategy, and exert an influence on the implicit system which will alter the reinforcement evaluations made by and the signals produced by the implicit system (Rolls, 1999:259).

It is interesting to note that when modeling implicit processing in a connectionist network, Smith and DeCoster (1998) also schematically show that the explicit system has no direct access to the motor systems but does project to motivational and affective systems. Thus, connectionist modeling also suggests that the explicit system can only indirectly influence emotional behaviour.

Important for our purposes, is the finding that there are outputs from the OFC to structures such as the basal ganglia (including the striatum and ventral striatum) to enable implicit, direct behavioural responses based on the reward/punishment value of a stimulus; these outputs do not pass through the language system. Behaviour produced in this way is produced by implicit systems and verbal declarations cannot be made about decisions resulting from such nonconscious computations (Rolls, 1999).

Evidence is gathering for the view that hysterical symptoms of the type traditionally associated with hysteria may represent the motor component of an innate response to threat that is learned and executed through the amygdala and following classical conditioning the OFC. The unconscious learning and memory of an emotional response in the form of 'symptoms' would explain why the affected individual experiences such disturbances as something that happens to them and why she vehemently denies the problem is 'all in the mind' or, alternatively, that she is faking her symptoms.

A similar view is put forward by Vuilleumier and his colleagues (2001, 2005) who propose that conversion symptoms demonstrate a selective inhibition of action resulting from inputs from the amygdala and OFC to the basal ganglia (see section 1.6). They suggest that these connections appear to provide a potential pathway by which strong emotions in the context of adversity could affect sensory and particularly somatosensory processing or result in the selective inhibition of action.

5.1.2 Expanding the dual route model of emotion and cognition

In section 4.3, the dual route model of fear conditioning was discussed with respect to the subcortical and cortical processes. We can expand on this theory with the finding that executive systems also appear to be divided according to whether the problem to be solved and action generated concerns emotional or cognitive stimuli. Conscious control over prepotent responses is not simply a matter of exerting will-power as pointed out. The question arises as to how decisions are made in animals such as humans that have both the implicit, direct reward-based system and the explicit rational, planning system which allows rewards to be manipulated thus changing behaviour. For possible answers we can again turn to the work of Rolls (1999) who has given this question considerable thought informed by his comprehensive knowledge regarding the operation of these systems. As he sees it, there are a number of situations in which the operation of one system is of more benefit to the organism than the other.

First, the implicit system may be especially important when rapid reactions to stimuli with reward or punishment value must be made. Then the direct connections from structures such as the OFC to the basal ganglia would allow for immediate action. In contrast, when the implicit system continually makes errors, Rolls thinks it would be beneficial for the organism to switch from automatic, direct action, based on obtaining what these systems decode as being the most positively reinforcing choice currently available, to the explicit, conscious control system which can evaluate with its long-term planning algorithms, what action should be performed next. By 'errors', Rolls means habitual behaviours that were once adaptive but are no longer so due to changing environmental circumstances.

For example, suppose that for many years taking route A may have been the most direct and shortest way for me to drive to work. Even though I know about a new route (B) and am told that it could cut down my traveling time, I continue to take

route A. To overcome my habitual behaviour, I consciously decide to try out route B. I might plan to get ready for work earlier than usual in order to compensate for any unforeseen time delays due to unfamiliarity, set the odometer to gauge whether the route is actually shorter, or time how long it takes me to reach my destination. If I find that route B is shorter and quicker, then, as part of my behavioural repertoire, theoretically, the OFC will select this behaviour in the future. In other words, driving to work on route B will become a new habit which no longer requires the intervention of the rational, planning system.

What this analysis suggests is that changing habitual behaviour which is no longer adaptive, is a function of both the OFC *and* DLPFC. The DLPFC, as part of the explicit system, is responsible for generating new behaviours and if these turn out to be adaptive (rewarded) then the OFC extinguishes the existing (habitual) behaviour because it is no longer appropriate. As Rolls states, there may also be a flow of influence from the explicit, verbal system to the implicit system in that the explicit system may decide on a plan of action or strategy and exert an influence on the implicit system which will alter the reinforcement evaluations made by, and the signals produced by, the implicit system (Rolls, 1999:259).

The above example of taking route B would be one possible scenario. Another more Freudian example might be a bored, sexually frustrated, housewife who feels she would like to escape her present circumstances but is also aware that she could not earn a sufficient wage to maintain her present life-style. She may cope with her problem by repressing her true feelings so as not to arouse suspicion in her partner. As we have seen, Freud suggested that this type of deception has an unconscious element in that the person's desires may not be made explicit following repression. In the literature on self-deception, this proposal is also put forward on the basis that if the person were conscious of her desires it would compromise the explicit system in what it produces (see for example, Alexander, 1975, 1979; Trivers, 1976, 1985, and a review by Nesse and Lloyd, 1992). However, I have argued against the idea that a thought, once conscious, can be

rendered unconscious (section 2.9) and continue to take this stance in Chapter Seven when I discuss the hypothesis that hysteria is best understood as an example of self-deception.

Another factor responsible for influencing the balance between control by implicit and explicit systems is, according to Rolls, the presence of pharmacological agents such as alcohol, which may alter the balance towards control by the implicit system. That is, it may allow the implicit system to influence the computations made by the explicit system and in so doing, alter the relative value it places on caution and restraint versus commitment to a risky plan or action. Most people are aware that excessive alcohol consumption can lead to over-estimating their ability to correctly assess their skills and judgment.

Other drugs besides alcohol might have this effect on automatic and controlled processing. I have shown that endogenous neuropeptides and hormones initially produced in response to stressors can, under some circumstances, lead to addiction initiating a cycle of self-abuse and possibly account for some behaviours traditionally categorized as 'hysterical'. Much misunderstood, or unexplained, human pathological behaviour may be a result of implicit, nonconscious processes which have their own 'reasons' and the explicit system can only infer these reasons from the limits of scientific knowledge.

Equipped with such knowledge we can begin to 'know ourselves' and to exert greater influence (as opposed to 'control') over such primordial systems when their actions are not in our best interests. As we shall see in the following chapter, emotion regulation theorists are involved in the task of assessing which emotion regulation strategies commonly practiced by humans are most effective in altering 'bottom-up' processing in response to fear conditioned stimuli. Their results show that suppression is not only the least effective strategy but also has negative implication for health and well-being.

5.2 Laterality effects in human emotional processing

An important observation in the literature on emotional processing that has not been adequately addressed so far is that in humans there is some lateralization of function in emotional processing.

The right hemisphere (RH) of the brain has long been associated with the processing of emotional and motivational stimuli and the execution of related behaviour based on classical and instrumental learning (Springer & Deutsch, 1993). This makes sense given that the left hemisphere (LH) in humans has become dominant during evolution for the processing of language and language-related problem solving.

A growing body of evidence shows that the neural circuitry of the stress system is located in the early developing RH (Schore, 2003). Because stress coping strategies are deeply connected into essential organismic functions, they begin their maturation (pre-and-post-natal) and all major fibre tracts can be identified by age three. Thus, implicit memory for emotionally significant events guides our behaviour long before explicit, language-mediated thought is capable of influencing our learned responses.

Early learning experiences with a traumatizing care-giver can negatively impact on stress coping strategies rendering them almost immune to modification, as learning theorists observed long ago. Affect dysregulation is now seen to be a fundamental mechanism of all psychiatric disorders and by implication is identified primarily as a dysfunction of the RHOFC (Shore, 2003).

Thus, with the development of language and writing, the more ancient specializations associated with the primitive mind of our ancestors became increasingly centred in the RH (Joseph, 1992; Springer & Deutsch, 1993; Damasio, 2003). The evolutionary history of this functional asymmetry could alone be the subject of a thesis, however, what is relevant to the present enquiry is

the fact that the processing of emotions, particularly negative emotions, is thought to be carried out, in large part, by this hemisphere (Springer and Deutsch, 1993). If we understand emotions to be primarily physiological responses to rewarding and punishing stimuli (Rolls, 1999), then we can see why this is the case.

Neuroscientists tell us that the highest level of integrated body-mapping occurs in the RH using the parietal lobes (e.g., Rolls, 1998; Damasio, 2003; Joseph, 1992). As we have two parietal lobes, one in the RH and one in the LH, sensory body images are maintained in both. However, whereas the LH maintains a sensory image of only the right side of the body, the RH receives sensations from both sides of the body and thus maintains a sensory image of the entire body (Joseph, 1992). According to Damasio (2003), comparable regions of the LH do not have the same function due, probably, to the committed participation of LH somatosensory cortices in language and speech. The LH is therefore significantly dependent upon the RH for its current knowledge about the state of the organism.

For example, after suffering a RH stroke, a patient might be blissfully unconcerned about their predicament, even mildly euphoric, because, on this view, without input from this hemisphere in the way of feelings, they simply do not comprehend the enormity of their plight. They may understand logically what the physician is telling them abut the future consequences of their condition, even the fact that they may not have long to live, yet in the absence of negative affect, the patient does not react as one might expect in the circumstances (Ramachandran, 1999). This inappropriate response contrasts with that of the patient suffering a LH stroke where the person is not only aware of her state but is generally overwhelmed by negative emotions and if the speech centers are intact, constantly talks about her concerns. Other evidence shows that patients are more likely to be depressed by a stroke if it is to the LH. This finding is consistent with the more general finding that the RH is implicated in feelings of depression (Starkstein and Robinson in Springer & Deutsch, 1993).

A number of researchers have observed that relative LH hypoactivation in the prefrontal lobes is linked to increased negative affect, decreased positive affect, or both. Perhaps, most important, is the finding that resting frontal asymmetry may be a biological marker of differential risk for affective or anxiety disorders (Tomarken & Davidson, 1994).

In previous chapters it was pointed out that excessive stress-induced production of opioids can result in feelings of analgesia which may present as somatosensory disturbances or dissociative states such as detachment, or a lack of emotion, depression and so forth. It has been reasoned that these effects are caused directly by opioids such as endorphins binding to opiate receptors in the brain. As the RH contains the most comprehensive representation of the body and the constant changes that occur in response to environmental stimuli, it is plausible that these stress-induced sensory changes have their greatest impact on the RH. It may also explain why the majority of hysterias are found to occur on the left side of the body (Devinsky et al., 2001).

5.3 The right hemisphere and hysterical symptoms

As far back as 1859 Paul Briquet documented that hysterical phenomena occurred disproportionately on the left side of the body. Of the 430 patients observed, 'hyperaesthesia, anaesthesia, chronic convulsions and paralyses were more frequent on the left side of the body.' In more modern times, Magee (1962), reported that only 3 out of 50 cases of hysterical hemianaesthesia or hemiplegia were right-sided. Similarly, Fallik and Sigal (quoted in Min & Lee, 1997) found that 76% of 33 hysterical patients with unilateral somatic symptoms had left-sided symptoms. Regan and LaBerbera (quoted in Min & Lee, 1997) reported that of 11 children and adolescents with unilateral conversion symptoms, 10 shared symptoms on the left side of the body.

These findings are reflected cross-culturally which is not surprising given that cerebral asymmetry is believed to be innate in humans and thus not influenced by

culture. For example, Min and Lee (1997) found that out of 61 Korean patients diagnosed with depressive disorders, anxiety disorders, and somatization disorders, somatic symptoms presented significantly more on the left side than the right side of the body. Many other researchers have commented on this predominance of left-sided symptoms in their hysterical patients but it has only been with the advent of functional neuroimaging that investigators could actually confirm the dominance of the right hemisphere in mediating hysterical symptoms (e.g., Devinsky et al., 2001; Marshall, et al., 1997; Vuilleumier, et al., 2001; Spence et al., 2000). We could hypothesize on the basis of these findings that in humans, the lateralization of hysterical symptoms follows on the lateralization of emotional processing.

This brief report on the functional asymmetry of emotion processing and hysterical symptoms, together with previous considerations regarding the brain regions that appear to be involved in maintaining conversion symptoms, point in the direction of such symptoms being the result of a dysfunctional affective system. It is dysfunctional in the sense that normal or adaptive neurophysiological functions aimed at coping with threat and the avoidance of threat, have come to dominate the afflicted person's somatosensory perceptions and behaviour. More specifically, the evidence would suggest that in the case of hysterical paralysis, motor systems, normally influenced by intentional goals, (associated with the function of left DLPFC) are inhibited by goals and subsequent behaviour automatically calculated by the right OFC to yield the current, most net reward gain.

Following their study with 78 patients diagnosed with conversion nonepileptic seizures (C-NES) Devinsky and his colleagues (Devinsky et al., 2001), came to a similar conclusion:

> Our findings suggest that right hemisphere dysfunction may facilitate the development of conversion symptoms, consistent with prior evidence of right hemisphere dominance in emotional regulation and supporting other findings that right hemisphere dysfunction may contribute to the

pathogenesis of conversion disorder (Devinsky, et al., 2001:370).

While there appears to be a consensus emerging on the psychoneurobiology of hysteria which locates the problem in RH dysfunction, not all investigators are convinced that such disorders arise in the way suggested. For example, Marshall et al., (1997) and Spence (2001), believe the evidence from neuroimaging studies shows that hysteria is a disorder of 'willed' action because there is typically a hypoactivation of the DLPFC uniquely associated with the 'subjective experience of deciding when to act and which action to perform' (Spence and Frith, 1999).
As this alternative view should be considered, I will now do so in the context of a neuroimaging study carried out by Marshall and his associates on a patient diagnosed with hysterical paralysis (Marshall, et al., 1997).

5.4 Hysteria as a disorder of 'willed' action

The patient investigated, B.L., was a 45 year-old right-handed widow with three children. Her relevant medical history included intermittent depression and reports of somatic symptoms such as mutism and lower-limb weakness extending over a period of 17 years. In the 3 years prior to her diagnosis of conversion hysteria, at least two documented psychologically traumatic events had occurred within the context of more general, chronic, familial and financial problems. Her current 'paralysis' began with a perceived weakness of the left leg and arm. The possibility that she had suffered a stroke was thoroughly investigated and ruled out.

Although, as Marshall and his colleagues state, it was difficult to categorically report that the patient was not feigning her symptoms, a revealing incident occurred prior to the study that led them to believe the diagnosis was correct. During the period of B.L.'s admission to hospital for investigation, a small fire had broken out on her ward and patients were evacuated. However, the nursing staff on duty at the time observed that B.L. either could not, or would not, attempt to escape like other patients, rather, she became 'seriously withdrawn and

unresponsive' (Anthwal, et al., 2001:225).

In fact, her emotional tone throughout was consistent with that noted in individuals presenting with what Perry (2001) and others have called the defeat response (section 4.8). If the patient was in a state of hypoarousal this might explain why Marshall and his colleagues reported that she did not appear anxious or upset during the study; an observation they believed was confirmed by the absence of amygdala activation. However, it should be noted that according to our previous analyses, amygdala activation is not a definitive sign of distress in humans (Rolls, 1999).

The role of the OFC in ameliorating feelings of social distress is confirmed by Eisenberger and her colleagues (Eisenberger, et al., 2003). Both the amygdala and OFC have outputs directed to producing autonomic and endocrine responses involved in opioid production (Rolls, 1999). As we shall see, some evidence for this interpretation of the patient's affective state is provided by these researchers' observations.

In an attempt to reveal the brain regions involved in B.L.'s condition, the investigators recorded brain activity when the patient prepared to move and attempted to move her paralysed left leg and when she prepared to move and did move her normal (right) leg. There were no control subjects in this experiment as the investigators considered the patient to act as her own control due to the clear-cut distinction between the motor capacity of her left (bad) leg and right (good) leg.

It was found that preparing to move or moving her normal leg, and also preparing to move her paralyzed leg, activated motor and/or premotor areas previously associated with movement preparation and execution. However, the attempt to move the paralyzed leg failed to activate the right (contralateral) primary motor cortex. Instead the right OFC was significantly activated. Marshall and his

colleagues suggest that this region inhibited prefrontal (willed) action normally executed through the (DLPFC) when the patient attempts to move her leg.

This argument was advanced and expanded upon by Spence (2001). Contrary to Rolls, Spence begins by arguing that any behaviour produced by unconscious (or implicit) systems cannot be considered an 'action'. Action, on his view, requires a deliberate choice (in consciousness) by an agent, that is, action requires conscious intention. For Spence, action without conscious thought is a self-contradictory conception (Spence, 2001). There are no involuntary actions merely involuntary movements.

Commenting on the above study, he hypothesizes that in patients with hysterical motor phenomena, 'the DLPFC is one brain region where abnormal 'willed' action might have a neurophysiological correlate' (Spence, 2001:243). This view is congruent with some past descriptions of hysteria. For example, as Brodie (1837) says:

> It is not the muscles which refuse to obey the will, but the will itself which has ceased to work (quoted in Merskey, 1999).

Here it appears to be assumed that so-called 'voluntary' actions in response to rewarding or punishing stimuli are normally a result of conscious intention acting *directly* rather than *indirectly* on motor systems. Thus, if a person cannot move her leg, according to Spence (2001:235) it follows that the disorder is the result of either a dysfunction of the DLPFC or 'an intention to act to deceive (the 'self' or the 'other')'. While our analysis does not rule out self-deception or deception as a complication of hysteria, on the evidence presented, the more likely interpretation is that hysterical symptoms do not have their inception in DLPFC dysfunction.

The first notable difference between the interpretation of the data by Marshall and his team and the one considered in the present thesis is that the psychological and emotional state of the patient is not considered to be a variable in B.L.'s capacity to move or not to move her 'paralyzed' leg. While these important factors in the

etiology of her condition are acknowledged in the patient's medical history, the results of the study are discussed more in relation to the functional organization of the normal motor system rather than within an emotional/motivational framework.

The idea that the pattern of activation observed may indicate that the patient's response could have signified an unconscious, emotionally motivated phenomena never enters the discussion following the rejection of this possibility based on the opinion that the patient showed no signs of emotional distress either before or during the study.

A second difference is the interpretation that the overall pattern of activation observed in this patient is 'abnormal' or 'pathological'. This does not appear to be the case when considered in light of other neuroimaging studies carried out with 'normals'. To explain, we need to know something about a region of the brain that alerts us to intercerebral conflict, namely, the anterior cingulate cortex (ACC) which has connections to both the OFC and DLPFC.

In various neuroimaging studies designed to identify the neural correlates of conflict states and conflict management, the activation of the ACC in conjunction with the OFC signals to the researcher that emotional conflict is being automatically resolved. For example, ACC activation has been associated with emotional distress in social exclusion studies and a combination of right ACC/OFC activation indicates that the distressed state is being regulated by the OFC (Eisenberger, et al., 2003). Conversely a combination of ACC/DLPFC activation is more typical seen in subjects attempting to resolve cognitive conflict, although as we shall see it is often the case that both frontal regions are recruited in real-life situations.

5.5 Anterior cingulate cortex (ACC)

The role of the ACC in cognitive and emotion regulation mediated through the DLPFC and OFC respectively is currently under intense investigation by research

psychologists. As it stands at the moment, the ACC appears to be involved in alerting the prefrontal regions to emotionally distressing or conflicting information that requires further processing.

Both the OFC and DLPFC have connections to the ACC and although the amygdala has relatively meager connections to the DLPFC it sends strong signals to the ACC once activated by an emotional stimulus. So through the ACC the amygdala is able to influence the cortical areas that are processing the stimuli activating it. This might be very important in directing attention to emotionally relevant stimuli by keeping the short-term object buffer focused on the stimuli to which the amygdala is assigning significance (Le Doux, 1998). For example, a person may be deeply engrossed in a cognitive or physical activity when his attention is suddenly diverted by the smell of smoke and the activity is temporarily forgotten while he seeks out the cause. Subjectively, the amygdala's response to aversive stimuli (in this case) is felt as arousal as discussed in Chapter Two.

As with most studies on brain function cognitive processes have tended to receive far more attention from researchers than emotion processes. Thus, studies aimed at understanding the function of the cingulate cortex have generally been designed to evaluate the role of this brain region in cognitive tasks such as inhibiting inappropriate cognitive responses, rather than emotion responses. For example, in the Stroop colour-word task the subject is instructed to name the colour of the ink of an incongruent word-colour stimulus (e.g., RED printed in green ink). Because word reading is a more automatic cognitive process than colour naming, subjects must resolve cognitive interference and inhibit the correct but more facile response in order to give the appropriate answer (Adleman, et al., 2001).

As we might predict, neuroimagining studies show that both the ACC and DLPFC are activated during studies of this type, a finding that is consistent with the standard model of cognitive control. When the ACC detects conflict between

implicit and explicit goals for action, the DLPFC is alerted and subsequently engaged in reducing conflict by biasing information processing towards the criteria most relevant to successful task completion. On the basis of such findings, the ACC, in conjunction with the DLPFC, is now recognized as part of what has been called the frontal lobe attentional network, a cognitive system involved in selective attention, mental resource allocation, decision-making processes and voluntary movement control (Adleman, et al., 2001).

However, considerable evidence has accrued which shows that this view is in need of modification (Le Doux, 1998; Rolls 1999; Greene, 2004; Egner and Hirsch, 2005). As Egner and Hirsch (2005) state, 'control' is implemented by both lateral and medial prefrontal cortices. On this view, the adaptations that allow us to respond flexibly to novel or challenging cognitive tasks are separate to those that allow for the inhibition, and if necessary, over-riding of responses that may have previously been associated with successful task performance but are no longer appropriate (rewarded). This executive function is not, according to Rolls, primarily carried out by the DLPFC as some researchers argue. As we have seen, this appears to be a function of the OFC particularly when it comes to decisions and actions that lead to personal punishment or reward. Greene et al., (2004:397) have also come to a similar view:

> ...one might render the emotional/cognition distinction in terms of contrast between, on the one hand, representations that have direct motivational force and, on the other representations that have no direct motivational force of their own, but that can be contingently connected to affective/emotional states that do have such force, thus producing behaviour that is both flexible and goal directed.

In other words, our emotions and emotional behaviour are not under direct voluntary control.

Thus, it is not unusual or indicative of a pathological state to observe a pattern of neural activation similar to that of patients diagnosed with hysteria, that is, an ACC/OFC combination. As mentioned this pattern of neural activation has also

been observed in 'normal' subjects during social exclusion studies (Eisenberger et al., 2003); moral judgment (Greene et al., 2004), and in a later study carried out by the above researchers, it was found to occur in hypnotic paralysis (Halligan et al., 2000).

Upon closer scrutiny what appears common to these studies is that the task set by the investigators induced negative emotions in subjects engendered, for the most part, by self-relevant discrepancies. That is, RHACC activation in conjunction with RHOFC activation simply means automatic emotion regulation is occurring in response to negative emotions.

Despite the fact that Marshall and his colleagues did not have the benefit of findings from these later studies, they did concede that their original interpretation of the neuroimaging data might need to be revised in light of the findings on hypnotic paralysis. As they say following their experiments with hypnotically induced paralysis:

> Alternatively, these activations could represent the management of mental dissonance produced when the suggestion of paralysis of the left limb conflicts with the explicit instruction to move it. Such an account would equally apply to hysterical people where the activations could reflect the management of a similarly generated internal conflict. While the first interpretation predicts that the recorded activations are specific to hypnotic or hysterical limb paralysis, the second would predict that the pattern of activation might also be seen with the same testing strategy, irrespective of the specific hysterical symptom or its hypnotically produced counterpart (Marshall et al., 2000: 34).

These investigators' intuitions appear to be correct. When mental dissonance involves self-relevant discrepancies, accompanying negative emotions appear to be regulated in some, perhaps emotionally vulnerable individuals, by unconscious processes involving the RHOFC.

In the context of hysteria, the inhibition of such negative affect by the limbic system appears to be abnormally intense affecting lower level systems such as the

thalamus and basal ganglia which, in turn, modulate motor processes based on emotional and situational cues (Rolls, 1999; Vuilleumier, et al., 2001).

To avoid the confounding influence of mental conflict reported by Marshall and his colleagues, Vuilleumier et al., (2001) assessed brain functional activation in seven patients with unilateral hysterical sensorimotor loss, during passive vibratory stimulation of the affected limb. These researchers were of the view that the motor deficits observed by Marshall's team, were not caused by inhibition of the primary motor cortex normally under voluntary control (or at least experienced as being under voluntary control). Rather, they hypothesized that such deficits were the result of limbic system activity exerting an inhibitory effect on systems normally involved in implicit behaviour. This view is consistent with Rolls' thesis that behaviour which occurs as a result of emotional processing, takes a different route to action than that resulting from explicit processing (experienced as being consciously 'willed').

Recall that Rolls (1999) points out that outputs from the association cortex to the amygdala and OFC can occur via projections directly to structures such as the basal ganglia (including the striatum and ventral striatum) and then to the thalamus and premotor cortex to enable implicit behavioural responses based on the reward or punishment value (or re-evaulation) of the stimuli being processed. Again, this is a normal process underpinning operantly conditioned responses but in hysteria appears to be abnormal.

The results obtained by Vuilleumier and his colleagues, using single photon emission computerized tomography, revealed a consistent decrease of regional blood flow in the thalamus and basal ganglia contralateral to the deficit, as might be anticipated if these researchers' hypothesis was correct. Importantly, this hypoactivation resolved following recovery and thus, could be considered specific to hysterical paralyses. On the basis of these findings, together with existing knowledge on the anatomy and function of the implicit route to action, they

concluded:

> That a role of these subcortical circuits in hysterical conversion therefore lends strong support to the view that they may derive from primitive psychobiological adaptive mechanisms or stereotyped illness behaviour with self-preservation value ... somewhat similar to instinctive freezing or immobilization reaction in response to perceived threats ... We would suggest that hysterical paralysis might build upon such neural mechanisms to establish a selective inhibition of action through the modulation of specific basal ganglia and thalamocortical systems, with such inhibition being possibly triggered outside conscious will by various emotional stressors, through limbic inputs from amygdala and orbitofrontal cortex. Decreased activity in basal ganglia-thalamic circuits might set the motor system in a functional state characterized by impaired motor readiness and initiation, resulting in abnormal voluntary behaviour (Vuilleumier, et al., 2001:1091).

Thus, there appears to be a convergence of opinion based on theoretical and empirical considerations, that hysteria has its phylogenetic roots in defense mechanisms common to all animals under threat. In humans threat can be real or imagined. Through the principles of associative learning these defenses can be triggered automatically in people who have a developmental history of family relationships that are threatening or dysfunctional and may persist as 'symptoms' throughout the person's life-span.

Many so-called 'psychiatric' disorders may, on this view, be reconceptualized as exaggerated expressions of adaptive communicational states that are part of an organism's ordinary repertoire; a view that is central to what Nesse (2000) called 'evolutionary psychiatry'. From this perspective, patients suffering from disorders such as depression, anxiety, fatigue states, and somatization disorders, currently classified separately in the DSM-IV, should occupy a single category (Price, et al., 2004). What these conditions appear to have in common is that they are all components of innate defense mechanisms, the function of which is to protect the organism against attack by conspecifics. Such reactions in the context of interpersonal conflict, function to switch off the aggression of a conspecific or serve to minimize further harm. (Price, et al., 2004; Kozlowska, 2007).

The foregoing discussion implies that, contrary to our intuitions, much complex human decision-making and in particular social behaviour, may be the result of implicit emotional processing. As Rolls cautions, we should be careful not to postulate intentional states (that is, states with intentions, beliefs and desires) unless the evidence for them is strong. He suggests that when Goleman (1996) talks about 'emotional intelligence', the functions being performed may be those mediated by the OFC which enables us to respond in socially appropriate ways to changing emotional cues given by conspecifics during social interaction.

5.6 The illusion of conscious will

As we have seen, it has been argued that the activation of the DLPFC is uniquely associated with the subjective experience of deciding when to act and which action to perform. So dominant is this feeling or subjective experience of being in control of our actions, that even psychologists who have access to contrary evidence from neuroscience still tend to treat such feelings as evidence for this putative fact. As Spence (2001) says, 'we might hypothesize that in patients with hysterical motor phenomena, the DLPFC is one brain region where abnormal 'willed' action might have a neurophysiological correlate (Spence, 2001:243). But then later, in the same paper, he concedes that given the findings of Libet and others (Libet, 1993, 1996; Frith, 1996):

> ...our own 'intentions to act' may be merely the (late) phenomenological correlates of preceding (cognitively unconscious) 'intentions in action'...if they are, then although we may subjectively distinguish those 'acts' for which we feel responsible from those 'movements' which are ours but unintended, the notion of 'choice'(a choice which is conscious and directing action) may be itself illusory. It may be neurophysiologically detectable, but nevertheless not constitutive of the 'agency' we perceive ourselves as possessing. The DLPFC may play a key role in the generation of action, but we cannot say this is where intention is (Spence, 2001:247).

Spence is referring to Libet's well-known observation that the conscious intention to act is itself preceded by relatively long predictive trains of electrical activity (of the order of 300-400ms). This controversial finding has been the source of considerable debate both in psychological (e.g. Spence, 1996; Wegner and Bargh,

1998) and philosophical (e.g., Dennett, 1991) circles as it strikes at the heart of a widely held belief that conscious will precedes or at least shares its temporal inception with brain events. As Libet sums it up:

> The initiation of the voluntary act appears to be an unconscious cerebral process. Clearly, free will or free choice of whether to act now could not be the initiating agent, contrary to one widely held view. This is, of course, also contrary to each individual's own introspective feeling that he/she consciously initiates such voluntary acts; this provides an important empirical example of the possibility that the subjective experience of a mental causality need not reflect the actual causative relationship between mental and brain events (Libet, 1992:269).

It seems that conscious intention is not the beginning of the process of making voluntary movements but rather is one of the events in a concatenate of responses that eventually yields such movement. According to Wegner (2002:3) we generally but wrongly assume that the experience of consciously willing an action, and the causation of the action by the person's conscious mind, are the same thing. As it turns out, however, they are entirely distinct and the tendency to confuse them is the source of what he calls 'the illusion of conscious will'. Wegner tells us that action and the experience of having carried out that action are dependent on different brain systems although we usually experience action to follow conscious intention: 'We feel we are doing something when in truth we have not done it...the experience of will is merely a feeling that occurs to a person ...' (Wegner, 2002:14). That feeling is believed to be dependent upon activation of the DLPFC.

We have seen that when the system that actually makes emotional decisions becomes (neurally) dissociated from the system that provides us with the experience of acting, the illusion of conscious will is revealed. Patients who suffer damage to the OFC may know consciously what action to take but cannot initiate such action. Hysteria seems yet another condition in which this impotence of conscious will is unmasked.

Rolls (1999) suggests our feeling of free-will may arise, in part, from the belief

that we can initiate and control action even if the OFC and/or amygdala, are the final arbiters of which action is performed in response to rewarding and punishing stimuli. Free-will would, on this view, involve the use of language to check many moves ahead on a number of possible series of actions and their outcomes, and then based on this information make a choice from the likely outcomes of different possible series of actions. If, in contrast, choices were made only on the basis of the reinforcement value of immediately available cues, then as Rolls says, 'the choice strategy would be much more limited, and we might not want to use the term free will...' (Rolls, 1999:254). However, it is worth reiterating that according to the author, the actual computation of how rewarding or punishing a particular response option is, and thus whether to act, occurs in the implicit system.

Taken together, these data support the possibility that hysterical symptoms have their origins in the RH. As a number of investigators suggest, the RH is dominant for the processing of emotional/ motivational stimuli and the results of this processing are made available to the LH in the form of feelings or subjective experience which can be reported. The time lag that would necessarily occur for information to be transmitted across the corpus callosum may signal to the verbal LH and the conscious mind, that these events are happening to it and contrasts to the feeling of conscious will which, according to the above view, depends on activation of the left DLPFC. This would explain why hysterical symptoms are experienced as being outside the patient's control (Kozlowska, 2005).

The illusion of conscious will in the context of emotion regulation is pervasive in western societies and encapsulated in such concepts as 'will-power', 'self-control', and 'ego-strength'. Most people believe they can (or should) suppress their negative feelings and desist from acting on them. Yet domestic violence, murder, child abuse, drug addiction (and the list goes on), are testament to the fact that our ideals fall short of reality. Habitual suppression of emotion feelings and their behavioural expression, as Freud pointed out, only provides relief from

anxiety in the short-term. In the long-term, contemporary researchers have found that this emotion regulation strategy may lead to health problems involving the immune and cardiovascular systems (e.g., Tomarken & Davidson, 1994; Shedlar, et al., 1993; Weinberger, 1990; John & Gross, 2004). It seems paradoxical then, to find in the literature that repression is actually believed to be good for one's mental health preventing anxiety-related conditions from developing (Tomarken & Davidson, 2004; Coifman et al., 2007). In Chapter Six I examine the concept of repression and find that it remains shrouded in mystery and controversy. The theories about what repression entails both mentally and physically are myriad and as Garssen (2007) states, working one's way through them all can be compared to being lost in a maze.

However, clinical observation shows that children and adolescents diagnosed with hysterical disorders are only too well aware of their predicament and the negative emotions they try to keep hidden from caregivers who threaten their physical and psychological well-being. These observations and the recounting of personal experiences by children such as 'Trish' (Appendix I) provide insight into the misery and suffering that if left unaddressed can lead to somatoform disorders and co-morbid conditions in adulthood.

6 Hysteria and emotion regulation

From a developmental perspective, as a child matures, he is gradually able to use explicit or cognitive systems to modulate implicit emotional response tendencies. As we have seen, these prepotent responses involve changes in the behavioural, experiential, autonomic and neuroendocrine systems and are abnormal in hysteria, resulting in somatic phenomena the patient or his caregivers believe are signs of physical illness. Although increasing maturity allows the individual some degree of control over such tendencies and to modify many aspects of their behaviour, the neural activity underpinning many behavioural repertoires remains implicit, and patterns of behaviour that were established before the availability of explicit systems may continue to be triggered automatically without being subject to conscious control (Kozlowska, 2007). It has been argued that this is the case in hysteria.

Recent functional analyses of emotion emphasizing the benefits of emotion feelings in many aspects of our daily lives (e.g., Ekman, 1992; Oatley & Johnson-Laird, 1987; Frijda, 1986; Damasio, 1994) should not blind us to the fact that emotional response tendencies often need to be consciously modulated. However, research shows that some cognitive strategies appear to be healthier or more adaptive than others.

The findings discussed in the previous chapter would suggest that the most effective cognitive strategies would be those aimed at changing the appraisal of a stimulus event to which the affective systems are responding. For example, if a person is confronted with bad news, he might modulate or down regulate an emotional response by focusing on past similar situations where he has successfully dealt with the type of problem presented. As we shall see, neuroimaging studies appear to confirm Rolls' theory that in order for emotion regulation to occur the reappraisal would need to be represented in the OFC (Oschner, et al., 2004).

6.1 Unhealthy and healthy emotion regulation strategies

In the emerging field of emotion regulation studies, researchers have made a distinction between three different forms of emotion regulation: reappraisal, suppression and repression. As predicted, reappraisal appears to offer the most benefits in terms of mental and physical health and is often contrasted to suppression (John & Gross, 2004). In the next five sections I will be drawing on the findings of Gross and his colleague and their general review of the relevant literature comparing expressive suppression and reappraisal.

While the distinction between reappraisal and suppression is clear, the difference between suppression and repression, as well shall see later in the present chapter, is not. Freud used the two terms interchangeably and on his view, the same processes or 'forces' responsible for removing unwanted emotional thoughts were deployed to prevent their reappearance in consciousness (Freud, 1905). However, modern theorists generally distinguish between conscious suppression and unconscious repression (Coifman, et al., 2007).

The over-arching framework Gross and his colleagues use for their thesis is a process model of emotion regulation based on the premise that specific emotion-regulation strategies can be differentiated along the time-line of the unfolding emotional response. This model is also favoured by other emotion theorists (e.g., Arnold, 1960; Ekman, 1972; Frijda, 1986; Izard, 1977).

The process model of emotion regulation, suggests that repression, unlike suppression is an antecedent-focused emotion regulation strategy (Coifman, et al., 2007; John & Gross, 2004). In other words, repression, on this view, prevents a full-blown emotional response whereas suppression occurs after the emotional response or is a response-focused emotion regulation strategy.

Historically, repression has been conceptually associated with hysteria. As Freud argued, no repression—no hysteria. In this chapter I present evidence to show that

conscious suppression rather than unconscious repression is associated in some instances of childhood and adolescent hysteria and by implication adult hysteria. First, I outline the process model of emotion regulation, followed by an examination of the emotion regulation strategies—reappraisal and suppression, and their relative costs and benefits in terms of health and well-being. I then go on to discuss some of the methodological problems and conceptual confusion surrounding the study of repression.

6.2 The process model of emotion regulation

Briefly stated, this conception of emotion generation holds that an emotion begins with the evaluation of an emotional stimulus. When attended to and evaluated in certain ways, emotion stimuli trigger a co-ordinated set of response tendencies. Once these response tendencies arise, they may be modulated in various ways. Because emotion unfolds over time, emotion regulation strategies can be distinguished in terms of when they have their primary impact on the emotion generative process (John & Gross, 2004).

At the broadest level these researchers distinguish between antecedent-focused and response-focused emotion regulation strategies:

> Antecedent-focused strategies refer to things we do before the emotion response tendencies have become fully activated and have changed our behaviour and our peripheral physiological responding. Response-focused strategies refer to things we do once an emotion is already underway, i.e., after the response tendencies have already been generated (John & Gross, 2004:1303).

An emotion may be regulated at five points in the emotion generative process by (1) selection of the situation, (2) modification of the situation, (3) deployment of attention, (4) change of cognitions, and (5) modulation of experiential, behavioural and physiological responses. The first four of these are antecedent-focused, whereas the fifth is response-focused (John & Gross, 2004).

For example, a person may avoid certain places, people or activities they know

from past experience elicit negative emotions. They may modify an aversive stimulus in some way so as to alter the emotional impact, or selectively attend to some less threatening aspect of the stimulus. Once focused on a particular aspect of the situation, cognitive change in the form of reappraisal refers to constructing one of the many possible meanings that may be attached to that aspect e.g., a person attending a job interview may avoid feeling nervous by viewing the event as an opportunity to find out all he can about the company, rather than as a test of his worth.

Conversely, response modulation refers to various kinds of attempts to influence an emotion response once it has already been elicited. Let us suppose that John is unjustly reprimanded by his boss in front of a colleague; he feels angry and embarrassed. How might he cope with his anger? According to Richards & Gross (2000) the most common way of coping with emotion feelings, at least in western societies, is to suppress them, which generally means suppressing the *expression* of an emotion. In other words, suppression only regulates the expression of an emotion rather than the emotion itself (John & Gross, 2004).

6.3 The benefits of reappraisal for health and well-being

Over the years many investigators have reported that people feel better when confronted by stressful events if they use cognitive transformations, currently termed *reappraisals* to redefine potentially stressful situations in more positive ways (Mischel, 1976).

For example, when patients confronting major surgery were helped to reconstrue their forthcoming ordeal in a more positive light they tended to cope better with their traumas, seeming to experience less stress and requesting less pain relief medication and sedatives (Holmes & Houston, 1974). In a similar study, Langer and her colleagues encouraged patients to imagine the hospital experience as a holiday—a chance to escape from the pressures of work, and were given other similar techniques for emphasizing the positive side of their experience (Langer,

Janis & Wolfer, 1975). Taylor & Brown (1988) found that cancer patients who were able to put a 'positive spin' on their condition appeared to cope well with their disease even though they often knew their condition was deteriorating. They called such cognitive transformations 'positive illusions' and argued that such illusory thinking is common among humans. When stress and pain are inevitable the adage 'to look for the silver lining' or 'accentuate the positive' may be wise.

6.4 Emotional impulses and expressive suppression

The ability to suppress one's emotions and particularly to suppress one's emotional behaviour has intrigued philosophers for millennia, and more recently, psychologists. This concern is reflected in concepts such as 'will-power' and 'self-control'. Let us suppose that in addition to the unconscious inhibition of inappropriate (punished) emotional behaviours, humans are also capable of cognitively controlling the expression of an emotion. This process, which Gross and his colleagues refer to as expressive suppression is shown to have negative consequences for the individual in terms of health and well-being that do not apply to reappraisal.

Gross and his colleagues began with a number of hypotheses regarding the detrimental effects of expressive suppression in the social domain based on the process model of emotion regulation. These investigators appear to assume, as did Freud, that suppression begins with a conscious decision to inhibit prepotent responses:

> Upon making the decision to hide their feelings, individuals instigate on-line comparisons between how they think they are behaving on the one hand, and some salient standard on the other, such as a mental representation of an unemotional facial expression or the way the face feels when it is not expressing emotion (Richards & Gross, 2000:411).

There are considerable costs associated with suppression that may give rise to a lack of emotional intelligence. The person is so engaged with controlling his emotions and ensuring that he hides them from others that he fails to absorb information needed to respond appropriately to the situation. He may then appear

avoidant, seemingly not in tune with the subtle ebb and flow of social interaction. Applied relatively late in the process of emotion generation, attention tends to be diverted away from the interaction partner and focused on reducing the experience and expression of the negative emotion. These repeated efforts should consume cognitive resources that could otherwise be used for optimal performance in the context of social interaction (Gross & Levenson, 1997). Chronic suppression should then result in impaired interpersonal relationships. Furthermore, because suppression is effortful this fact should be reflected in autonomic measures.

To test the veracity of these hypotheses, Gross and his colleagues carried out numerous studies under controlled laboratory conditions with subjects instructed in how to use both emotion regulation strategies. From their research findings John & Gross (2004) concluded that using reappraisal to regulate emotions is associated with healthier patterns of affect, social functioning and well-being than is the use of suppression.

6.5 Reappraisal versus suppression

In one study, subjects were divided into three groups: a control group, and two experimental groups. Subjects were then shown film clips depicting gory scenes of dead people taken at various road accident sites. The control group simply viewed the film clips without attempting to consciously influence their emotional responses; the experimental groups were either asked to use reappraisal strategies taught to them prior to the study or to suppress their emotions.

Results indicated that suppression decreased the behavioural expression of negative emotion but not the subjective experience. Although participants who suppressed showed much less expressive behaviour, they felt as much negative emotion as participants who simply watched the film clips. Physiologically, participants who suppressed showed signs of greater physiological activation (in cardiovascular and electrodermal systems) than participants who simply watched.

By contrast, reappraisal (thinking about the film they were watching in such a way that they would not respond emotionally) decreased both the experience *and* the behavioural expression of negative emotion, without any increase in physiological activation. The physiological consequences of suppression are consistent with the view that suppression is an effortful form of self-regulation (as compared to either reappraisal or to no regulation).

To test the social consequences of suppression and reappraisal, John & Gross (2004) adapted a design from studies in social support: when individuals interact with another person who shows few of the facial expressions expected in that particular social interaction, they experience stress, and that stress can be indexed by their physiological responding. Similarly, subjects who interacted with people who used suppression experienced more stress (e.g., greater increases in blood-pressure) than subjects who interacted with people using reappraisal. The experimenters concluded that by disrupting the give and take of emotional communication, suppression might be considerably more disruptive to social interaction, relationships, and social support than reappraisal.

In another experiment these investigators examined memory for social information (e.g., names or facts about an individual seen on a slide) while participants were either reappraising or suppressing. As predicted, suppression, but not reappraisal, led to memory impairments for social information presented while the individual was regulating emotions. Other experiments of this nature have also shown that subjects who suppressed their emotional reactions during a conversation recalled fewer details about the conversation than did participants who either acted naturally or reappraised during the conversation.

More generally, people who regularly use suppression as a way of dealing with emotions, are less likely to be clearly aware of and express their upset than those who use suppression less frequently. They view such states less favourably and repair them less successfully. Suppression involves 'shutting down' emotions in a

way that interferes with attention to the emotion and prohibits coping via venting. For example, Rime et al., (1992) found that suppressors were less likely to share their emotional experiences with others and feel uncomfortable with close relationships which they actively avoid.

As expected, reappraisers are found to have quite different social experiences to those of suppressors. They experience and express more positive emotions, and in combination with their positive stance on challenging situations, they are likely to be sort after as friends and associates. In contrast to suppressors, reappraisers feel free to engage in social sharing of emotions, both positive and negative. Reappraisal is also positively related to mood repair, presumably because this strategy involves trying to think differently about a negative situation before affective systems are fully engaged (John & Gross, 2004).

John & Gross (2004) concluded from their studies and those conducted by their colleagues, that the frequent use of reappraisal promotes psychological well-being. After all, one of the key effects of reappraisal is diminishing the negative emotional impact of adversity. To the extent that depressive symptoms are either triggered or exacerbated by negative responses to challenge or losses, reappraisal should exert a protective effect against depressive symptoms. Given the positive emotional and social outcomes associated with reappraisal, it is not surprising that reappraisers report greater life satisfaction and higher self-esteem than suppressors (John & Gross, 2004).

6.6 Suppression and inauthenticity

One of the most significant findings made by Gross and his colleagues was that suppression can create in the individual a sense of discrepancy or incongruence between inner experience and outer expression as this emotion regulation strategy does not allow the person to express his true feelings. In other words, suppressors lack *authenticity* which may lead to negative feelings about the self and alienate the individual from others, further impeding the development of emotionally close

relationships.

Individuals who chronically use suppression are keenly aware of their lack of authenticity and they admit to deceiving others about their true inner feelings, attitudes and beliefs (John & Gross, 2004). They do so, they report, because they are concerned about not being accepted by others, suggesting that these individuals use suppression in personal relationships they care about and are afraid to lose. Thus, as these investigators see it, the motive for presenting oneself in ways that are discrepant from one's inner self is to avoid disapproval and social rejection. As we shall see, this hypothesis is supported by findings in the field of research on childhood conversion disorder and implies that chronic suppression in adults has its origins in early socialization experiences. In large measure, it is this awareness of being inauthentic that I argue separates the suppressor from the repressor.

As one would anticipate, given their keen awareness of inauthenticity, suppressors have been found to have lower levels of life satisfaction and well-being (Sheldon, et al., 1997); less life satisfaction; lower self-esteem; and a less optimistic attitude about the future.

6.7 Suppression and hysteria

Social forces powerfully shape, and to a significant degree, determine individual differences in emotion regulation strategies. Parents differ in their meta-emotion philosophy, defined as 'an organized set of feelings and thoughts about one's own emotions and one's children's emotions' (Gottman, et al., 1996:243). The 'emotion-coaching philosophy' (John & Gross, 2004) is characterized by attention to, and positive evaluation of, emotions, with explicit discussion of how best to manage them—a parental philosophy that predicts the encouragement of reappraisal. The 'dismissing philosophy' (John & Gross, 2004) by contrast, views emotions as dangerous and focuses on avoiding and minimizing them; thus affirming once more a link between early socialization and the habitual use of

expressive suppression as an emotion regulation strategy.

Consistent with these ideas is the finding that suppression of negative emotions is typical in a sub-group of children presenting with conversion disorder. Studies on conversion disorder typically describe two distinct patterns of intra-familial relationships (Kozlowska, 2003). The first group is characterized by a pattern of chaotic social and family circumstances which are evident to the clinician at the outset. Children living in such conditions are obviously stressed and the diagnosis of conversion disorder is more easily made (e.g., Goodyear, 1980; Steinhausen, et al., 1989; Volkmar, et al., 1984).

A second group is characterized by an apparent lack of social, family and psychological difficulties; here, the family presents as a socially respectable, harmonious unit, (Kozlowska, 2001, 2003; Seltzer, 1985). However, in a study of fifteen such families, Seltzer (1985) found that beneath this 'super-normal' presentation lay a tendency for parents to dismiss past significant traumatic events in their own lives. In situations where family therapy has been possible, it is often revealed that these emotionally and psychologically damaged adults had themselves been entrapped in problematic relationships characterized by a sense of injury, social humiliation, poverty, illegitimacy, victimization and violence. According to Kozlowska (2003), this dismissing philosophy is motivated by a view that sees the expression of negative emotions as dangerous and therefore to be suppressed (Schimmack & Hartmann 1997). In other words, such families appear to be defensively suppressing their distress as the following case presented by Kozlowska (2003:499) suggests:

> Jane was an 11year-old girl, the younger of two siblings, who presented with suicidal ideation and multiple conversion symptoms over a period of 12 months: Loss of upper-and lower-limb sensation and movement; intermittent blindness and deafness; and recurrent pseudo-seizures characterized by amnesia and jerkings of the whole body or by a loss of body tone, causing Jane to fall to the ground. Each episode lasted weeks to months, with pseudo-seizures being Jane's predominant and most pervasive symptom across and between episodes. No medical explanations of Jane's symptoms could be found despite investigations by multiple

medical teams.

Although there was no obvious precipitant to Jane's initial presentation, a history of multiple family stressors over a 3-year period was noted: Loss of a grandparent, severe mental illness, death of family friends, ongoing conflict and rejection of the family by the extended family, chronic conflict between the parents and Jane's school, and severe difficulties in the parental marital relationship related to Jane's father's workaholic tendencies and chronic absence from home. On family assessment, striking features included: Jane's smiling facial expression and complete denial of any feelings of distress and sadness; the family's lack of knowledge about how other members in the family felt; and the apparent emotional disconnection (which hid distress and anger) between Jane's parents.

In the majority of such cases, parents in this group predictably respond to the suggestion that relationship problems may be responsible for their child's condition with defensive denial. As clinicians report, attempts to raise such issues frequently result in a hostile response from the child's parents, who may be 'incredulous, displeased, angry or insulted' (Kozlowska, 2003:77). Steiner (1992) also observed that family members tended to corroborate the patients' self-reports regarding family cohesiveness and reported lower child-parent discrepancies than published norms, showing the importance of conformity in these families. In sum, enmeshment, conformity and conflict avoidance stand out as salient features of their coping behaviour.

The gradual admission to chronic feelings of sadness, fear, anger, hopelessness and helplessness expressed over the treatment period is typical in this sub-group of children raised in families where the communication of personal pain is prohibited. When one child, Karla, was asked by the clinician what she thought actually lead to her recovery, she replied:

> When I talked it through. I kind of didn't have a choice ... Because if I didn't talk then I wouldn't get out of hospital and I wanted to go home ...

> At first, I felt like, my body got really bad and I had these thoughts about what would happen or what they think of me ... my parents ... They would reject me because I told (Kozlowska, 2003:79).

As Breuer and Freud observed, when strong emotions are permitted to be released, and verbal expression is given to the distressing events, patients are often 'cured' from their long-standing symptoms. Recall that Breuer's patient Anna O. referred to the cathartic method as the 'talking cure'.

Consistent with the findings discussed above on suppression, there appears to be an incongruence between these children's private thoughts and feelings and their social behaviour with their distress being reflected in somatic symptoms. As Kozlowska explains it, 'For some children, the energy required to maintain the strategy [suppression] may become overwhelming and presentation with conversion disorder reflects a break-down of the strategy and gives some children an opportunity for psychological reorganization if sufficiently supported (Kozlowska, 2001). On her view, conversion symptoms demonstrate not only motor and sensory disturbances, but also 'dysynchrony between the external display of positive affect and inner experience' (Kozlowska, 2003:77).

The demonstration of a discrepancy between autonomic affective responses indicative of high arousal and anxiety in these children and their role-formatted, parent-pleasing actions negatively reinforced by the avoidance of punishment, is similar to the discrepancy found in adult individuals categorized as repressors but who might more appropriately be classified as 'suppressors'.

6.8 The difference between suppression and repression

As pointed out, the current trend among psychologists is to distinguish between suppression and repression. Suppression is almost unanimously held to be a conscious process (e.g., Valliant, 1990; Watson & Greer, 1983; Coifman et al., 2007), while there is less certainty about whether repression is, or is not, a conscious process. For example, the DSM-IV defines suppression as 'a defense

mechanism in which the person intentionally avoids thinking about disturbing problems, desires, feelings or experiences'. On the other hand, repressive coping behaviour is understood to emerge in a relatively automatic, self-deceptive manner, and thus is conceptualized as qualitatively distinct from emotion or thought suppression. According to Gross and his colleagues, people who use repression as an emotion regulation strategy differ from suppressors inasmuch as they use a technique which, like reappraisal, reduces the impact of a negative emotion stimulus (Coifman, et al., 2007).

In the laboratory, people identified as repressors evidence a greater ability than so-called 'normals' to divert their attention away from threatening stimuli during tasks measuring seemingly automatic biases of attention, but do not differ from other respondents on measures of deliberate emotional control or avoidance (Coifman, et al., 2007). On this view, repressors do not use reappraisal but another form of antecedent-focused emotion regulation John & Gross (2004) refer to as 'attentional deployment' (section 6.1) where attention is diverted away from the negative aspects of a potentially threatening stimulus, particularly when such information threatens the person's self-esteem (Garssen, 2007; Coifman, et al., 2007; Weinberger, 1990; Gil, 2005).

This strategy can be distinguished from reappraisal where the person chooses to focus on the positive aspects of a stressful situation and then proceeds to enhance those aspects, as the adage 'look for the silver lining' suggests. In other words, there is no attempt by the repressor to cognitively transform the threatening event by altering its meaning. The repressor selectively screens out the information he finds threatening.

If repression is an automatic or habitual cognitive skill as these authors suggest, it should be relatively effortless compared to suppression. Therefore, in contrast to the person who uses conscious emotion suppression, we would not expect to find signs of autonomic reactivity in the repressor when confronted with a laboratory

stressor. However, it turns out that the opposite is true. Repressors consistently evidence biological markers of distress under such conditions (e.g., elevated blood-pressure, heart-rate and cortisol levels). Gil (2005:28) summarizes these findings:

> From a mind-body perspective, researchers showed that self-reports of low distress, intense physiological and behavioral reactivity, and biased information processing form a maladaptive combination that places adult repressors at risk for health problems. That is, (1) adult repressors' health indices revealed a biochemical makeup associated with the development of chronic and serious health conditions (i.e. high blood-pressure, impaired immune functions, high levels of glucocorticoids, and high levels of blood lipids and (2) empirical studies among patients already diagnosed with cancer, HIV, multiple sclerosis or other serious conditions indicated that repressors exhibit a worse progression of disease, more recurrences, and more elevated mortality than non-repressors.

It is now widely recognized that a repressive adaptive style is associated with a poorer health prognosis and with biochemical markers of health difficulties.

These findings have perplexed investigators because they conflict with those gathered from self-report questionnaires showing that repressors are not anxious (Shedlar, et al., 1993; Tomarken & Davidson, 1994; Colvin & Block, 1994). The autonomic-verbal discrepancy is generally considered a marker of the repressive-defensive coping style (RDS—Weinberger, 1990). How to explain this discrepancy remains a contentious issue among psychologists who study the phenomenon. Some argue that methodological problems are to blame (Shedlar, et al., 1993; Colvin & Block, 1994), while others claim that the concept of repression, as it is currently understood, is flawed (Garssen, 2007; Paulhus, 1984). I will now examine how psychologists identify people who use the RDS and the problems associated with using self-report questionnaires to gain insight into their actual mental and emotional states.

6.9 Methodological issues

The current literature on repressive coping and ANS reactivity reveals great variation in experimental designs, including differences in choice of experimental

challenge, in choice of ANS measure, in sample characteristics, as well as dissimilarities in how the Weinberger paradigm (Weinberger, 1990) used to identify repressors, is operationalized (Garssen, 2007). These variations may have an impact on the results obtained explaining the contradictions observed. Construct validity may be another more intrinsic problem. For example, Weinberger used a self-report questionnaire designed originally as a means of controlling for the tendency of some subjects to routinely endorse socially desirable items. In other words, to identify people who fake being 'good'.

The tendency to respond to test questions in certain ways is referred to by psychologists as a 'response set'. Such sets have to be understood and controlled for in order to interpret self-reports and ratings properly. Since the early 1940's researchers have recognized that one cannot expect people would be either willing or able to reveal themselves accurately in response to items such as 'are you shy?' or 'do you worry a lot?' This would be particularly unreasonable when the respondent is emotionally upset, disturbed or aware that their answers may be used to make important decisions about them and their future.

For example the Minnesota Multiphasic Personality Inventory (MMPI) includes ten basic scales that were initially devised to help classify mental patients into types, e.g, the depressive personality, the hysteric, hypochondriac and so on. In addition to these basic scales three controls have been devised. The 'L' scale which measures the tendency to falsify about oneself by 'faking good'. High scores on this scale indicate that the individual has endorsed many items that suggest he does unlikely things such as daily reading all the newspaper editorials or never telling a lie. The K scale, a second control, was intended to indicate defensiveness in the form of a tendency to present oneself in a more socially desirable way. The F scale, the third control, sought to tap the intrusive effects of answering the items carelessly and confusedly, as indicated by describing oneself as having rare and improbable characteristics (Mischel, 1976).

Weinberger (1990) used a similar scale to the K scale in order to identify repressors, called the Marlowe-Crowne Social Desirability Scale (MC – Crowne & Marlowe, 1964). The MC is a 33 item self-report measure that assesses both the tendency to deny negative characteristics that are likely to be common in the general population (e.g., I have never intensely disliked anyone) and the tendency to ascribe to oneself positive characteristics that are thought to be rare in the general population (e.g., I never hesitate to go out of my way to help someone in trouble).

Social desirability was also studied as a personality characteristic by these investigators who reasonably assumed the trait was motivated by a strong need for social approval. How might this motive manifest itself behaviourally? According to Crowne & Marlowe:

> Dependence on the approval of others should make it difficult to assert one's independence, and so the approval-motivated person should be susceptible to social influence, compliant, and conforming ... We thought that we had found the perfect exemplar when one approval-motivated subject, a nearly complete conformer, commented brightly about her conformity in the post-experimental interview, "Yes, of course. That's teamwork, isn't it?" (Crowne & Marlowe 1964).

It is noteworthy that in the 1960s, the MC was used by psychologists to identify conformers rather than repressors, although conformity and repression were seen by Crowne and Marlowe, (1964, 1991) to be related, and both traits, on their view, had their origins in an abnormally high need for approval indicative of a person with vulnerable self-esteem. In general, it was found that when compared to normal or control groups, high conformers were more cautious, conventional and persuasible (Mischel, 1976; Mann, 1969).

In turn, the research that followed Crowne & Marlow's work, suggested that this personality trait may have its developmental origins in early attachment problems with caregivers. I will discuss attachment theory as it relates to hysteria in Chapter Seven, however, it has already been noted that the compulsive-compliant

attachment style is evident in some children presenting with hysterical disorders. For our purposes, it is significant that one of the most illuminating studies on the antecedents of socially desirable responding found that harsh parental practices predicted subsequent social desirability responding, particularly maternal criticism and hostility. Furthermore, such factors had a stronger effect when they occurred during infancy (Allaman, et al., 1972). Over the ensuing years, many similar studies have confirmed these findings (for a review see Gil, 2005).

Clinical evidence would suggest that contrary to Freud's beliefs, conscious suppression rather than unconscious repression precedes hysteria and that it is the break-down of this strategy that results in stress-related somatic symptoms.

However, we are still left with the problem of how to explain the high levels of autonomic arousal observed in people identified as repressors using the MC. Could it be the case that the MC is, in fact, an instrument for identifying suppression rather than repression? This interpretation would be consistent with the hypothesis put forward by Gross and his colleagues (Coifman, et al., 2007), namely, that repression is an antecedent-focused emotion regulation strategy and thus should not be effortful. Suppression, on the other hand, *is* effortful and found to be associated with heightened ANS activity. I will now argue that the answer is not that simple.

6.10 Repression and conformity

A clue to what might be going on here comes from the well-studied phenomenon of conformity. Repression and conformity appear to be opposite sides of the same coin so what we know about the conformer may help elucidate the personality profile of the repressor, and, at the same time, explain the pattern of ANS reactivity observed.

The most important finding for our purposes is that people who manifest high conformity are not a homogenous group. Some may conform because they have

internalized the rules or social norms they enact and endorse on the MC, while others may be motivated by the need for approval or the fear of social rejection (Mann, 1969). Mann (1969) referred to these two types of conformity as 'true conformity' and 'expedient conformity' respectively. Importantly, the MC does not distinguish between these different groups. I would suggest that this conflation is responsible for the confusing and often contradictory results of experiments using the MC as a measure of repression noted by Garssen (2007) in his review of the literature.

On the above view, the true conformer/repressor actually *believes* in the social values and standards of behaviour he has internalized and acts in accordance with them. In contrast, the expedient conformer/suppressor only conforms because she seeks approval from significant others or fears social rejection. The children presenting with conversion reactions described by Kozlowska (2007) seem to be in the latter group. If this *is* the case, then the MC would be capturing both the true conformer/repressor *and* the expedient conformer/suppressor as both groups would be anticipated to score highly on measures of social desirability.

Paulhus (1984, 1990, 1991, 1998) has similarly argued that the MC captures two different groups providing some support for the above theory. He distinguishes between these two groups on the basis of self-deception. Paulhus (1984) found that under threat of public disclosure, socially desirable responding increased significantly more among people scoring high on the MC and a measure of self-deception, than those who genuinely believed the items they endorsed were true. This observation is consistent with early studies on conformity behaviour (Mann, 1969) and more recently with studies on repression where some people with high scores on the MC are observed to give less 'repressed' answers on measures of social desirability when they can be anonymous (Sapolsky, 1998).

If this interpretation is correct, it might explain why people with high scores on the MC who are suppressors rather than repressors, show corresponding high

levels of autonomic reactivity, however, we would not expect repressors to evidence the same high levels of arousal.

From his interpretation of the data Sapolsky (1998) argues that repressors have chronically activated stress-responses because they keep their emotions and their world under such tight control. They report that they don't have many stressors and this may be true, but the lesson we can draw from the repressive personality type, according to Sapolsky, is that, 'sometimes, it can be enormously stressful to construct a world without stressors' (Sapolsky, 1998:286).

It seems that contrary to the idea that repression is effortless (John & Gross, 2004; Coifman, et al., 2007) people using this strategy expend considerable energy and cognitive resources on planning their lives in such a way as to minimize stressful events. That is, people who use a repressive coping style tend to avoid unpleasant events that elicit negative emotions. When they cannot avoid an emotional situation, their response is likely to be just as tightly circumscribed. For example, when repressors and nonrepressors were asked to recall an experience associated with a specific, strong emotion, both groups reported the particular emotion with equal intensity. However, when asked what else they were feeling, nonrepressors typically reported an array of additional, nondominant feelings: 'Well, it mostly made me angry, but also a little sad, and a little disgusted too ...' (Sapolsky, 1998:285). Repressors steadfastly report no secondary emotions—black and white feelings, with little tolerance for subtle blends and, similarly, little tolerance for the expression of complicated emotions in others (Sapolsky, 1998). The tendency for repressors to refrain from protracted emotion processing is congruent with the finding that in contrast to reappraisers, repressors tend not to cognitively process emotional events but rather divert their attention away from aversive stimuli.

The majority of psychologists who study repression use the Weinberger paradigm because it is seen to have extensive validity in that scores correlate (1) with the degree of conformity to group pressure and responsiveness to social

reinforcement, (2) with the likelihood of expressing aggression, and (3) with sensitivity to perceived situational demands (Brodie et al., 1995). However, on the views expressed above, it is not a valid construct as it cannot distinguish between people who repress their emotions because they genuinely subscribe to a belief that is still pervasive in western societies, namely, that emotions—particularly negatively valenced emotion—are dangerous and to be controlled, and those who suppress the expression of their emotions because it is expedient to do so.

A separate issue to be considered is whether the repressor/true conformer is actually deceiving himself. It seems to me that if a person actually thinks and behaves in accordance with the values and norms considered socially desirable, he may be considered over-socialized, but it does not necessarily follow that he is self-deceived. Self-deception often manifests itself in the ability to say one thing and do another without any apparent cognizance of the discrepancy between the verbal and physical act. It is a judgment usually made by an observer regarding another's behaviour and the construct lacks adequate scientific support. Thus, to distinguish repressors from suppressors on the basis of self-deception (Paulhus, 1984) is not particularly useful. The fact that much human behaviour is now acknowledged to emanate from implicit systems and processes outside of conscious awareness seems to me to provide a more solid basis for understanding the discrepancy between explicit propositions and behaviour particularly when such behaviour is in response to rewarding or punishing stimuli.

A more objective way to analyze the verbal-autonomic dichotomy is to examine the neural correlates of repression. This does not settle the question of how to distinguish between true conformers and expedient conformers, but it may help settle the question of how to interpret the high arousal states of these people.

6.11 The neural correlates of repression

Based on previous neurobiological considerations regarding emotional and cognitive control, we might anticipate that both suppression and repression, if

consciously implemented, activate LH prefrontal systems leading to feelings equated with self-control rather than distress. On the other hand, the RH has been associated with feelings of anxiety and other dysphoric emotions generated by a perceived lack of control.

Tomarken & Davidson (1994) approached the problem of how to interpret heightened arousal in people classified as repressors with these findings in mind. They began with the observation that EEG asymmetry in anterior (i.e. prefrontal) regions of the brain is a potent biological marker of affective style and psychopathology. As noted in Chapter Five, relative LH frontal hypoactivation has been linked to increased negative affect, decreased positive affect or both. Although neuroimaging studies on hysteria are not conclusive, they are consistent with a more general body of evidence that suggests affective disorders are associated with LH hypoactivation and a predominant RH activation.

These investigators reasoned that if, contrary to their self-reports, repressors were actually distressed, they would demonstrate relative right anterior activation particularly when compared with low-anxious subjects who typically have low scores on the MC. The latter have consistently demonstrated decreased autonomic and neuroendocrine activation when compared with repressors. The results of their EEG studies showed that repressors use their LH rather than RH when confronted with a laboratory stressor. The data showed that repressors were differentiated from this comparison group primarily on the degree of power density in homologous LH midfrontal sites, and the exclusive activation of LH rather than both LH *and* RH lateral regions.

Tomarken & Davidson (1994), suggest that this finding supports the hypothesis that heightened autonomic and neuroendocrine activation observed in people classified as repressors might not reflect distress per se but rather the mobilization of processes that serve to inhibit distress or facilitate goal-directed behaviour, a view that is consistent with the above analysis. The finding that low-anxious

subjects activate both LH and RH frontal lobes implies they use a more balanced emotion regulation style than repressors who may overly rely on cognitive control.

It would seem that just as people who suffer from hysteria and other anxiety-related disorders rely on automatic emotion regulation when emotionally challenged (evidenced by RH frontal activation), those individuals who habitually repress or suppress their emotions rely exclusively on the use of controlled strategies which, over time, become habitual. However, cognitively mediated habitual repression or suppression should not be confused with automatic emotion regulation subserved by the OFC and amygdala. As Mauss, et al., (2007) observe, most research carried out in the field of emotion regulation has left out automatic emotion regulation (AER) defined as a:

> ...goal-driven driven change to any aspect of one's emotions without making a conscious decision to do so, without paying attention to the process of regulating one's emotions, and without engaging in deliberate control (Mauss, et al., 2007:3)

The automatic control of responses to rewarding and punishing stimuli (emotions) via the OFC, amygdala and ANS (sympathetic and parasympathetic) from birth has been the subject of discussion throughout the present thesis.

Clinical evidence suggests that in the case of hysteria, the chronic use of expressive suppression only acts to reduce threat and feelings of anxiety in the present. It does nothing to alleviate the *cause* of the individual's distress, unless the child (or adult) is removed from the fear stimulus (e.g., an abusive parent or partner). When suppression can no longer be sustained in the context of chronic distress, the break-down of this strategy (rather than repression) results in increased levels of fear-induced arousal, as might be anticipated if refraining from emotion expression functioned to protect the individual from harm and/or to gain approval.

On the other hand, if repressors are true conformers, people who believe that it is

wise to plan for all contingencies; to discipline their emotions so they do not hold sway over reason; and generally act in ways that conform to social norms, it seems unlikely that we would find them on the psychiatrist's couch. We can see why then, some psychologists might understand repression to be conducive to mental health. Repressors, it would seem, have become what Freud metaphorically called the *superego*, the voice in our head that tells us what we should or should not say and do. In other words, the 'internalized parent' has robbed them of their independence in thought or deed. The 'benefit' is that such a person would suffer none of the conflict or guilt experienced by the expedient conformer or suppressor, as their private beliefs and public behaviours are consistent.

However, the rigid conformist is generally not considered by most social psychologists to be an ideal product of socialization. Asch (1952), whose thinking on the subject has been responsible for most of the seminal research on conformity behaviour, considered that independence and refusal to yield to social pressures are of social and personal significance:

> The act of independence is productive from the social point of view, since it is the only way to correct errors and to steer the social process in accordance with felt requirements ... to be independent is to assert the *authentic* value of one's own experience (Asch, 1952:495-497).

We have seen from the studies carried out by Gross and his colleagues that feelings of inauthenticity are characteristic of suppressors, people who, on the view argued above, are probably expedient conformers.

Nevertheless, the essence of conformity, evidenced by socially desirable behaviour, is yielding to group pressure. Thus, both suppressors and repressors are likely to be susceptible to the influence of authority figures or valued reference groups and to lack the independence of opinion that Asch and others saw as integral to intellectual and emotional independence. The difference between the repressor and the suppressor is that the latter is aware he is compromising the value of his own experience. This dilemma, which stems from

the interdependence of human beings, appears to begin with compliance to parental pressure which, as we have seen in the case of children who present with conversion disorder, is not only excessive but often entails abuse.

There is considerable evidence to show that both the repressor and suppressor are raised within a family environment that demands blind obedience to family rules and regulations (Gil, 2005). Like conformity, obedience may be productive and socially adaptive particularly during childhood. However, over-control and over-socialization may result in a person who is capable of carrying out cruel and destructive behaviour, depending upon the beliefs, attitudes and behaviours of significant others. Consider the blind obedience to irrational social and political regimes such as Nazi Germany before and during World War II. Many experiments carried out by social psychologists during the 1960's showed with great regularity that apparently decent and responsible people will yield to the pressure of authority figures, often inflicting harsh and callous behaviour on innocent victims for no greater reason than they were instructed to do so by the experimenter (Milgram, 1965; Mann, 1969).

According to the view argued for here, the repressive-defensive coping style (RDS) and suppression are strategies used by individuals who share a common developmental history. If Gross and his colleagues are right, people who use reappraisal are generally those raised in a family environment that positively evaluates emotions and encourages the discussion of how best to manage them. Contrary to those individuals who use repression and suppression, reappraisers do not feel that negative emotions are dangerous and thus something to be avoided. However, they also acknowledge that emphasizing the positive aspects of a stressful event instead of focusing on the negative, helps both themselves and others to feel better.

Furthermore, the process model of emotion regulation supports the notion that repression, used as a coping strategy in the context of stress, does not entail the

use of reappraisal or narrative but only attentional deployment. That is the repressor turns away from or refuses to acknowledge the threatening information.

It is now possible to see the extent to which reappraisal differs from repression and suppression as a means of regulating emotions. However, some researchers have failed to distinguish between repression and reappraisal since both styles involve self-enhancement (Tomarken & Davidson, 1994). For example, Tomarken & Davidson have used the work of Taylor & Brown (1988), who found that positive cognitive reappraisals (or 'positive illusions') are conducive to mental health, to support their claim that the self-enhancing cognitive style characteristic of defensive repression may also confer the same mental health benefits. Yet, Taylor & Brown (1994, 2003) vehemently refute the idea that such positive reappraisals and repression are functionally similar:

> Repression and denial alter reality, whereas illusions simply interpret it in the best possible light. Defenses distort the facts, leading people to hold misperceptions of internal and external reality. Through illusions on the other hand, people make the most of bad situations by adopting a maximally positive perspective (Taylor & Brown, 1994).

As pointed out in section 6.3, a patient diagnosed with terminal cancer may decide to put a 'positive spin' on her deteriorating condition even though she may be fully aware that her health has worsened. Within the context of that acknowledgement, she has chosen to adopt a more positive and cheerful attitude than her condition warrants. Conversely, a patient using denial and repression might refuse to acknowledge her condition, or avoid thinking about it, in an attempt to control her negative emotions. This is an important topic in behavioural medicine as the tendency to repress may influence the reporting of somatic symptoms and the severity of symptoms if they are reported (Garssen, 2007).

6.12 The neural correlates of reappraisal versus repression

Recently, support for the distinction between repression and reappraisal has come from neuroimaging studies. In a nutshell, reappraisal differs from repression inasmuch as the former has the capacity, if executed convincingly, to interfere

with bottom-up processes involved in the appraisal of emotion events (Ochsner et al., 2002, 2004). These investigators observed that amelioration or down-regulation of a negative emotion response relies on the representation of the reappraisal narrative in RHOFC. This finding is consistent with Rolls' hypothesis discussed in the previous chapter, namely, that altering the affective value of a stimulus may depend critically upon representation of an event in RHOFC and/or amygdala (Rolls, 1999).

Conversely, the repressive coping style is associated with activation of the LHPFC and is remarkable for the lack of right frontal involvement compared to non-repressors during stressful laboratory tasks (Tomarken & Davidson, 1994). Since emotion down-regulation relies critically on RHOFC this may explain the observed high physiological reactivity in both repressors and suppressors. However, this hypothesis requires further empirical investigation given the different interpretations of physiological arousal in people classified as repressors.

Taylor & Brown (1994) seem justified then, in claiming that there is a difference between repression and the type of cognitive reappraisal they call 'positive illusions'. However, to put it bluntly, how successful reappraisal is in terms of emotion regulation would depend upon the degree to which a person can 'fool' themselves into believing their positive (in this instance) reappraisal.

According to these investigators, positive illusions are highly prevalent in normal thought and predictive of criteria traditionally associated with mental health. On their view the illusion-adjustment relation only breaks down or becomes pathological at high levels of illusion: 'It is absolutely clear that certain illusions or distortions (e.g., delusions of grandeur, hallucinations, gross misperception of physical reality) are associated with mental illness' (Taylor & Brown, 1994:26).

Over the last two decades, the idea that such cognitive distortions foster mental health has gained considerable attention and acceptance among many academic

psychologists and health professionals alike. However, as others see it, this paradigm shift in the way mental health is perceived threatens the time-honoured view held by many philosophers and more recently psychologists, that the distortion of reality and the cultivation of illusion are maladaptive (Colvin & Block, 1994). According to western philosophical tradition spanning more than twenty centuries, the unique quality of humankind is the ability to engage in rational, adaptive thought and to distinguish the real from the apparent. From a psychological perspective the ability to fool or deceive oneself would seem to be a fatal invention of nature as successful psychological adaptation requires an accurate evaluation of one's own motives and capacities (Allport, 1940).

Since Freud, mental health practitioners have been even more emphatic in their views citing 'reality orientation' as of critical importance for mental health. For example, in a monograph submitted to the Joint Commission on Mental Illness and Health in 1958, the author concluded that the mentally healthy person is someone 'able to take in matters one wishes were different, without distorting them to fit these wishes—that is, without inventing cues not actually existing' (Jahoda, 1958:51). Certainly, embedded in the practices of psychoanalysts, humanists and cognitive behavioural therapists, is the goal of training clinical populations to obtain deeper self-insight in order that their clients may discern such incongruities and correct them, or to come to terms with their failures as well as their successes. On this view, the cultivation of self-enhancing perceptions and illusions of control are seen to subvert the faculty of introspection resulting in a lack of self-awareness or self-knowledge, in which case, as Wells & Iyengar (2005:83) argue:

> ... the act of introspection serves not as a tool to increase self-awareness, but instead induces the individual to exhibit systematic biases toward upholding unrealistic positive self perceptions.

In an ideal world we should, as Maslow (1968) claimed, be able to accept our own human nature with all of its frailties, in the same way that one takes or accepts the characteristics of nature. In other words we should be able to accept

discrepancies between our ideal self-image and our behaviours in the world without feeling real concern or the necessity to distort our self-perceptions.

However, we do not live in an ideal world. In order to preserve a positive self-image individuals within our culture are motivated to perceive themselves and to be perceived by others in certain socially desirable ways which often run counter to our true thoughts and feelings. Studies have repeatedly demonstrated that when people engage in behaviours counter to their self-image they may distort such self-inconsistent cognitions. These distortions can take many different forms: denial, minimizing and avoiding discrepant cognitions about themselves, or by reappraising events in order to see them in a more positive light. As we have seen positive reappraisal is conducive to mental and physical health because, if successful, it down-regulates an aversive emotional response either to a perception or the memory of an emotional event.

In the context of child abuse and domestic violence, the idea that practitioners help people to develop a repressive-defensive coping style in order to deal with life stressors reveals, I believe, the danger of academic psychology divorced from clinical or practical reality. Yet, as Kozlowska (2003) reports, even in the field, physicians confronted with strong parental denial tend to retreat from dialogue about contributing psychological factors in the etiology of their child's symptoms. In turn, the physician's retreat into silence ensures that shameful or threatening emotional and interpersonal issues remain hidden, and that the family's projected image and apparent solid character are not undermined.

In the following chapter, I shall examine how this defensive stance has influenced our understanding of clinical hysteria and underlies a view that is still pervasive among practicing psychologists, namely, that hysteria is 'all in the mind'.

7 Reconceptualizing hysteria

Being honest and open with others about what we think and how we feel can be dangerous as we saw in the last chapter. Depending on early experiences with caregivers we may learn to be dishonest about these internal mental states – we can become, as John & Gross (2004) point out, 'inauthentic' or 'false'. Individuals, who chronically suppress their emotional impulses from fear of rejection or the need for approval, often admit to their therapists that they deceive others and further, that this lack of congruence between inner experience and outer expression causes them to feel negative about themselves. From the research examined, children raised by hostile, critical caregivers are likely to be candidates for conversion hysteria as Freud suggested (Freud, 1933). However, it is evident from the self-disclosed experiences of children presenting with conversion reactions that contrary to Freud, their inner experiences are not hidden from consciousness but from others. The symptoms presented, at least in this group of children, appear to be the result of their inability to sustain expressive suppression which, coming late in the emotion-generative process, requires the individual to exert considerable effort over prepotent impulses.

It has been noted that the region of the brain most likely to involve conscious emotional control such as suppression is the dorsolateral prefrontal cortex (DLPFC) identified by researchers as the part of the brain responsible for feelings of conscious control. Conversely, the activation of the orbitofrontal cortex (OFC) is seen in cases of hysteria and associated feelings of depression or anxiety (hypoarousal and hyperarousal respectively) mediated in turn by peptides (chemical messengers) such as opioids and adrenaline. When produced in excess amounts under conditions of extreme stress, these chemicals may produce abnormal physical, emotional and cognitive effects. As Sapolsky (1998) maintains, such changes in the state of the organism are real, and can be explained scientifically, if not in detail at this stage, then at least sufficiently to create doubt

in the mind of the reader that conversion reactions are simply the products of the patient's imagination.

The question that will be addressed in this closing chapter is this: Can the dualistic medical model which forces symptoms into one of two main categories—organic or psychological (or a 'bit of both'), accommodate problems of stress coping mechanisms that lie at the core of infant, child and adult hysteria?

I shall begin this examination with an appraisal of the current mechanistic paradigm and how this model has helped create the present untenable position between doctor and patient—one that is characterized by hostility and mistrust. I do not intend here to defend or argue against the Cartesian theory of mind-body dualism. I simply relate a concern held by many theorists both within and outside the discipline of philosophy, regarding the pervasive belief that mind can be separated, at least in principle, from the brain and bodily function.

This overview will be followed by an analysis of the latest developments in the organismic approach to understanding somatoform disorders where symptoms are conceptualized as components of innate defense strategies observed in infrahumans and humans alike (e.g., Price, et al., 2004; Kozlowska, 2007). Like Freud, these investigators trace the origins of somatization and other co-morbid disorders to early attachment relationships particularly with primary caregivers. I focus on Kozlowska's recent attempt to explain the developmental origins of conversion disorders within the framework of attachment theory. The model Kozlowska references is the dynamic-maturational model of attachment (DMM – Crittenden, 2006) as it focuses on the behaviour of children at risk. Unlike traditional theories of adult psychopatholgy, attachment theory takes as its starting point patterns of attachment (strategies) developed between caregivers and their infants and how these different patterns influence the individual's later interpersonal relationships.

7.1 The biomedical model of illness and classical world-view

For over four hundred years the core philosophical assumptions put forward by Rene Descartes have influenced the way western societies think and talk about the world and human nature. These assumptions are (a) the physical world is analogous to a machine composed of discrete parts located in time and space; (b) complex phenomena are ultimately reducible to these elementary parts and their relationships, and (c) the human organism is a physical entity separable from the nonphysical mind (or soul). Together, these assumptions form the basis of the mechanistic world-view.

Applied to the study of human development, the Cartesian paradigm yields a reactive model of development. The individual is seen as inherently at rest. Activity and therefore change, is the result of external forces. Further, change is quantitative rather than qualitative. For example, many of the debates in developmental psychology reflect the reduction and polarization of complex phenomena and their effect on behaviour, e.g., the nature versus nurture debate. How much of each is involved? Similarly, in medicine, illness is reduced to the physical versus mental.

When practitioners are confronted with symptoms that cannot be explained solely by appealing to organic or physical variables, they tend to ask to what degree the patient's condition is psychological or functional. As Halligan (2001:xii) states the problem: 'How are we to interpret the patient who does indeed have visible lesions or abnormal biochemistry but in whom we nonetheless suspect that the full range and intensity of the symptoms is not plausibly accounted for by these organic factors alone?' This way of framing the problem often results in questions related to deception or self-deception.

Consider the following hypothetical. A woman, healthy in most respects, develops severe back pain as the result of a fall. The pain becomes chronic and she imagines that it may be caused by an as yet, undiagnosed tumour pressing on

the spinal nerves. A CT scan and other diagnostic tests are carried out. They reveal some degenerative changes in the spine but the doctor considers that the perceived intensity of pain reported is not consistent with such minimal damage. The doctor suspects that there is a functional overlay. On her next visit, he tells his patient that her worse fears have been disconfirmed—there is no tumour. He suggests she may be over-anxious and perhaps recommends relaxation techniques or therapy. The doctor is surprised when his patient refuses to accept the findings. She insists he must have 'missed something' as she is not imagining her pain and argues that her anxiety is due to her condition. Thus begins a long history of 'doctor shopping' to track down her 'mystery illness'.

According to Wells (1996) possible precipitating causes of so-called 'functional' symptoms are rarely explored with the patient. Rather, as he observes, 'Most [patients] are sent home with the suggestion they will soon be better or with an admonition to "get going"' (Wells, 1996:36). It is rare, in his experience, for patients to be offered specific practical advice with ongoing support about how to manage their condition. Simply reassuring the patient that everything is 'normal' does not work. Neither does providing them with a psychiatric diagnosis such as 'conversion disorder'. The patient will understandably feel that the doctor is refusing to acknowledge the reality of the symptoms and clinical experience suggests that they will feel misunderstood, insulted or even abused.

When a person experiences unusual or altered body states whether in the form of mental phenomena, for example, dizziness, feelings of detachment; or somatic phenomena, such as the inability to feel a limb, speak or see, the most natural response is to infer that 'something is wrong' physically. It is taught from childhood that a visit to the doctor seems to be a sensible course of action in such situations. There is an expectation that after an adequate assessment the doctor will reveal the cause of the symptoms and state clearly what needs to be done in order to get better.

As Kozlowska (2005) sees it, 'avoidance of dialogue about unexplained medical symptoms functions to sidestep the practitioner's ignorance and to avoid hostility in the doctor-patient relationship'. Miller (1988) calls this stance 'the agnostic silent position'. Other doctors might communicate to their patients that they feel 'out of their depth' or worse, imply that patient is faking their symptoms (Kozlowska, 2005). Jureidini & Taylor (2002:124) observe that: 'Many doctors tend to use the term 'psychological' as a euphemism for non-structural, probably ephemeral, marginally volitional, possibly contrived or even dissembled'. The idea that hysteria involves a 'weakness of will' or 'lack of ego strength' and at worst, out-right deception, is evident from the literature (Spence, 2001).

These less than satisfactory positions adopted toward patients diagnosed with hysteria often exacerbate the patient's condition. Although some patients may not show overt signs of their true feelings, a person who is distressed and genuinely perplexed by his symptoms, can only feel more alienated and helpless in the hands of a doctor who believes they derive from her 'imagination' but offers no explanation of how the mind can cause the experience of such gross abnormalities. Social pain theorists show us that emotional conflict and perceived relational devaluation can directly add to the individual's suffering (MacDonald, 2005) and may affect his ability to engage in treatment.

We can now perhaps understand the reason for the widely acknowledged poor quality of doctor-patient relations that seems irresolvable in the case of hysteria and why the patient's condition is often observed to worsen as a corollary of the relationship itself.

7.2 Redefining the problem of hysteria

There are a number of reasons why a person who genuinely experiences strange, disturbing, or incapacitating bodily symptoms might refuse to believe the problem is all in their mind or imagination. As stated above, the major reason is that the patient who cannot move her limbs conceptualizes her condition as physical

necessitating a physical 'cure'. Few people in modern western societies, even those who are religious, would think of consulting their priest to heal them unless a medical solution is not forthcoming. Most believe that physical problems have physical causes. Among those patients who present with symptoms which cannot be explained (or adequately explained) within the present biomedical model, there is a significant group who do not accept the medical evidence showing there is nothing physically wrong with them. According to a number of psychologists, how to explain this disbelief is the main problem of hysteria. As Taylor writes, understanding the physiology of hysterical disorders is not as 'crucial' as understanding how and why:

> ...for a variety of motives, some people transiently or chronically... lay claim to physical ailments, impairments and disorders which they do not have, and for which they are prepared at times to manufacture the evidence (Taylor, 1989:391).

For these clinicians then, contemporary theories that explain conversion symptoms as part of the human emotional response to threat, reflecting either errors in how information is processed and represented, or the motor component of an automatic emotional response, might be of interest, however, they do not explain 'how a person can sustain a belief based on incomplete evidence' (Jureidini & Taylor, 2002). Adherents to the psychological view do not deny that such self-deception may begin with a physiological response to stress or perhaps with some physical trauma. However, what defines clinical hysteria for them is the false belief or illusion that one is suffering from a serious neurological disease; an idea that may flourish as a result of outside influence. As Jureidini & Taylor (2002:124) state: 'Hysteria may result when a normal physiological response has been elaborated into sickness by the system in which it occurs'. The 'system' might be the family unit, particularly if the patient is a child, and/or the medical system.

It would seem that the lack of a biomedical explanation for hysterical symptoms

and the general acceptance of their origins as psychological, has seen a subsequent shift in defining the problem of hysteria. That is, hysteria has been reframed in such a way as to circumvent the need for a neurophysiological explanation of symptoms.

7.3 Hysteria and self-deception

Turner (1999) has argued that only a cognitive, psychologically informed account of self-deception can cover the range of phenomena observed in hysteria. He cites Mele (1997), who proposes that a self-deceived individual often systematically manipulates the evidence to support his or her hypothesis. Mele (1997) argues that a person who employs such strategies as positive and negative misinterpretation, selective attending and selective evidence gathering, and who is also 'primed', (that is, motivationally biased) can become self-deceived. This highly influential deflationary account of self-deception requires only that the body of evidence possessed by the subject at the time of entering into self-deception provides greater warrant for the true belief (P) than for the false belief (not-P). It does not require that the subject actually hold the true belief (e.g., 'I am not sick') nor does it require the process of entering into self-deception be an intentional one.

On the above view, the term 'hysteria' refers to the patient's misinterpretation of evidence and selective attention to only a certain part of the overall evidence. In the process, the patient arrives at a mistaken conclusion about his or her own health or the nature of his or her past experiences (Bass, 2001). The fact that there is no requirement that the patient at any stage or at any level know the truth meets the criterion set by the DSM that the symptom is not intentionally produced or feigned (DSM-IV). That is, the patient must at all times, hold only the false belief (not-P).

Although Mele's theory of self-deception does not require an intentionalist explanation, the term does imply that the person is aware of P at least during the

initial stages of the process. As Guttenplan (1996:560) says: 'it should be clear that deceiving oneself, if it can happen, is more in the nature of a project. It takes time and a degree of careful, devious, planning to get oneself to ignore what would otherwise be plainly admitted'. Perhaps the term 'self-deception' is not a useful metaphor, at least in the context of clinical hysteria, for describing the refusal of some people to accept that there is nothing physically wrong with them. If the patient strongly believes he is physically ill because, say, he has lost the use of his leg, and is subsequently told that there is no organic reason for his condition, he may experience what psychologists call 'cognitive dissonance' (Festinger, 1957).

7.4 Cognitive dissonance and distorting the facts

Cognitive dissonance is an aversive, tension-producing mental state that people are motivated to reduce in the easiest (most cognitively economical) way possible (Festinger, 1957). In particular, conflicting self-relevant cognitions are most likely to result in the positive biasing of information that is consistent with existing self cognitions and the avoidance of conflicting cognitions (Aronson & Carlsmith, 1962). Thus, the distortion of information Mele sees as characteristic of self-deception may occur as the result of consistency-seeking among, in the case of hysteria, self-relevant cognitions about the individual's perceived physical state and conflicting medical evidence which indicates that there is nothing organically wrong. (Recall that cognitive conflict was induced in the patient diagnosed with hysterical paralysis, contaminating the results and conclusions drawn by Marshall and his colleagues).

Dissonance reduction appears to be a process that involves both automatic and controlled processing, and may, or may not involve affective sytems depending on the nature of the task (Thagard, 1998; Schultz & Lepper, 1997, 1988). Using connectionist network modelling cognitive scientists have shown that the motivation to seek cognitive consistency can be understood primarily in terms of constraint satisfaction; an automatic process that entails the simultaneous

adjustment of 'elements' to satisfy as many internal and external constraints as possible (Schultz & Lepper, 1998). Incoherence or inconsistencies trigger feelings of tension motivating consistency-seeking behaviour which is thought to be mainly a controlled, effortful, process involving LH systems. This view is congruent with what is known about the interaction of implicit and explicit processes in problem-solving. As Sloman (1996) suggests people use relatively automatic, associative (implicit) processes as well as more controlled, rule-based (explicit) processes. The two systems may operate simultaneously, with the rule-based system sometimes suppressing the expression of the associative one and *vice versa.*

Applied to the problem of why some patients refuse to accept their symptoms are imaginary or psychological, the above model suggests that the medical evidence is not sufficiently convincing to overcome the 'reality' of the person's perceived bodily disturbance. If the physician was able to explain the nature of the patient's predicament in terms that can be understood by him and with the appropriate degree of compassion, the current impasse may be resolved in a way that is satisfactory to both the physician and patient.

Individuals who experience heart palpitations, tremors, digestive problems, and a variety of other familiar systems of distress, are often prepared to accept that psychological and emotional factors may be playing an important role in their condition. However, as Taylor (1989) has pointed out, the animal defense behaviour he calls 'playing possum' and Kozlowska (2007) refers to as the 'freeze' response, yields symptoms that are less familiar to most people including medical practitioners, and may therefore be interpreted as signs of neurological problems. According to Taylor (1989:395), 'While the response to threat in human beings through flight or fighting back is amply described in student textbooks of physiology and psychology, little is said about responses to events in which these options are both precluded.'

On my view, self-deception is not a useful concept for understanding either the symptoms of what might be considered 'disorders of inhibition' (Crittenden, 2006) or the response of patients who refuse to believe their symptoms are psychogenic. Importantly, it does not explain symptoms in young children diagnosed with conversion reactions, a point I will return to later in the present chapter.

7.5 Stress, false body states and illusion

One way of conceptualizing the dilemma observed in older patients is by reference to a similar type of predicament referred to as 'pseudocyeisis' or 'false pregnancy'. At first glance, the illusory condition might appear, like hysteria, to involve the type of self-deception described by Mele. However, matters are not so simple. The illusion of pregnancy involves the interaction of social, emotional, cognitive and physiological variables.

False pregnancy is perhaps one of the most bizarre illustrations of the interaction between such variables, particularly between physical processes and cognitive processes. Many doctors think that this condition arises in the context of extreme anxiety about one's failure to conceive (Ramachandran & Blakeslee, 1999). For example, Queen Mary I of England famously suffered false pregnancy under pressure to continue the royal line. The illusion is associated with physiological changes that occur in normal pregnancy: cessation of menstruation, breast enlargement, morning sickness, and so on, except, of course, there is no fetus.

Just as it has been proposed that physical or emotional distress involves physical changes in the body and brain which, if prolonged, may result in somatoform disorders, Ramachandran and Blakeslee (1999) propose that in false pregnancy, prolonged stress and depression may result in physiological changes that are mistaken for symptoms of pregnancy. For example, there may be a reduction in neurotransmitters such as dopamine and noradrenaline affecting both follicle-stimulating hormone (FSH), which causes ovulation, and a substance called

prolactin-inhibition factor. Low levels of these hormones lead to the cessation of ovulation and menstruation and an elevation in the level of prolactin causing breast enlargement and lactation, nipple tingling and possibly maternal behaviour (this has yet to be shown in humans), along with an increased production of estrogen and progesterone by the ovaries. The woman's abdomen may also become distended due to a stress-induced accumulation of gas or because of constipation. In turn, abdominal distension causes a lowering of the diaphragm, a pushing forward of the pelvic portion of the spine and a dramatic growth of the greater omentum, (a pendulous apron of fat that hangs loose in front of the intestines).

These symptoms are interpreted by the woman as signs of pregnancy rather than of stress and thus begins a process of feedback between brain systems and the body proper which maintains the false belief until medical tests show the woman is not pregnant or she fails to deliver. In other words, her perceived condition is partly sustained through positive feedback or reinforcement. As she sees her abdomen enlarge and feels her diaphragm fall, the more pregnant she looks which, in turn, affirms her belief. In rare cases, an actual uterine enlargement can occur.

In most instances, when the truth is revealed the woman is forced to accept that her belief is false. However, in one documented case, the illusion was maintained. According to the doctor's case notes, he had entered into the pretense by telling the patient that her 'baby' was 'stillborn' and thought that would be an end to the matter. Several weeks later, the patient returned with her abdomen still enlarged, and happily told him that he had failed to deliver the 'twin' (quoted in Ramachandran & Blakeslee, 1999). In this type of situation, a false belief may be extremely difficult to eradicate. Similarly, in hysteria, if the patient is receiving false information regarding her body state, she may not be convinced there is nothing physically wrong with her. Medical evidence is not always irrefutable—false negatives are possible whereas it is more difficult to deny the

absence of a fetus.

A combination of factors contribute to the illusion of pregnancy; stress, physiological changes, imagination, belief and unconscious learning. In some cases of false neurological symptoms the same processes may also combine to create and maintain the illusion of disease. As Damasio (2003) and others suggest, stress-induced opioids appear to be responsible for altering body representations in some instances of conversion disorder. If Damasio is correct, the patient's body state is being misrepresented and thus the system that supports the phenomenological self is deceived into thinking that the symptom is a real manifestation of a physical illness.

In turn, the person's (false) belief that he is ill may give rise to complex feedback loops involved in generating and maintaining the false paralysis or other sensory/motor disturbances. This hypothesis is based on the finding, discussed in the previous chapter, that cognitive reappraisals can interfere with bottom-up processing of a stimulus (section 6.17).

Using the false pregnancy analogy, we can see that even the deflationary view of self-deception does not adequately describe the complicated multi-dimensional processes leading to the false belief that one is sick. Rather, it deals with what happens *after* the belief is formed and the patient is told he or she is imagining her condition.

Ramachandran & Blakeslee (1999:221) claim that such 'odd' clinical phenomena which confound current biomedic explanation 'illustrates our ignorance and illuminates the need for conducting scientific experiments on such patients.' Despite the methodological problems associated with early neuroimaging studies such as those carried out by Marshall and his colleagues, such research, together with increasing knowledge regarding the neuroendocrinological effects of stress on bodily representations, are providing valuable insights into the neural and

physiological aspects of conditions that test the veracity of the mind-body divide. The distinction may be no more than a 'pedagogic device for instructing medical students and not a useful construct for understanding human health, disease, and behaviour' (Ramachandran & Blakeslee, 1998:221).

7.6 Towards an organismic view of somatoform disorders

As the time for the DSM-V approaches debate about somatoform disorders (including conversion disorders) is intensifying. Impetus for change in their categorization is growing with the number of critics who claim that the current classification promotes mind-body dualism and perpetuates the devaluation of patients who present with symptoms that cannot be explained in terms of organic illness (e.g., Kozlowska, 2007; Noyes, et al., 2008). Although patients with psychiatric disorders generally are stigmatized, individuals with somatoform disorders are singled out because their symptoms mimic those associated with various diseases (Porter, 1995). As we have seen, this observation is often interpreted as a sign that the patient is deliberately faking her condition.

A number of changes in the classification of somatoform disorders have been proposed but few address the need to change the dualistic thinking that has lead to the conceptualization of these disorders as non-legitimate disturbances for which patients themselves are responsible (Kirmayer, 1988). This is puzzling as many critics of the current classificatory system claim that somatoform disorders are creations of the dualistic thinking underpinning the Western medical model (Kozlowska, 2007; see Noyes, et al., 2008 for a current review of problems with the classification of somatoform disorders in DSM-IV).

I shall argue that the classical, mechanistic paradigm is not a useful framework for understanding the emerging view of hysterical symptoms as animal defense behaviours. It perpetuates the idea previously examined, namely, that hysteria involves an imitation of physical conditions by persons who believe they suffer from some disease (McHugh & Slavney, 1998). Rather than adopting a broader

conceptualization of somatoform disorders as some commentators suggest (e.g., Rief & Sharpe, 2004; Kozlowska, 2007) on my view, what is required is a change in paradigms. Specifically, we need to make a shift in our thinking from a paradigm that encourages mind-body dualism to a systems view which encourages us to think of ourselves as dynamic self-regulating biological systems rather than 'minds' or 'souls'.

As Claude Bernard (1813-1878) argued, each organism has an internal environment in which its organs and tissues live and in a healthy organism this internal environment remains essentially constant even when the external environment fluctuates considerably. We have seen that Breuer & Freud (1895) applied this notion to cerebral function or 'the organ of ideation', and more recently, connectionist modelling has shown us that the majority of cognitive functions carried out by the body's control system (the brain and nervous system) operate in accordance with the principle of self-regulation (homeostasis).

Congruent with the organismic paradigm is the view put forward by Bowlby (1969), founder of attachment theory. Bowlby argued that the human infant is innately capable of controlling his environment rather than being solely at the mercy of external forces as the classical paradigm claims. For the present purposes, attachment theory provides a useful framework for collating the emerging, often fragmented, ideas and findings discussed throughout this work, not easily accommodated by the existing dualistic medical model.

7.7 Attachment theory

A fundamental tenet of attachment theory is that humans have an innate propensity to organize self-protective and, after puberty, sexual strategies (Bowlby, 1969; Crittenden, 1997, 2006). According to this etiologic model, somatoform disorders can be understood as exaggerations of normal responses to threat and are therefore envisaged as deviations of quantity rather than quality (Noyes, et al., 2008).

Although the concept of 'attachment' is familiar to psychologists, it may require elucidation for those working in other academic disciplines. So, first I ask the question: 'What is attachment?' According to Ainsworth and her colleagues, it is an interactive process which takes place initially between an infant and its primary caregiver:

> An attachment may be defined as an affectional tie that one person or animal forms between himself and another specific one—a tie that binds them together in space and endures over time (Ainsworth et al in Woodhead et al., 1992:31).

The term and concept were introduced by Bowlby (1969, 1973, 1980). Like so many other psychologists, Bowlby wanted to preserve some of Freud's insights about parent-child relationships and their repercussions for the child's later interpersonal and social behaviour, by casting them in a more scientifically defensible framework. Unimpressed by drive theory and Freud's deterministic view of human development, Bowlby believed that infant behaviour was logical and purposeful. He hypothesized that the goal of the infant's control system (the brain and nervous system) is to first establish a strong attachment with the primary caregiver (most often the mother) who will provide a safe, secure, base for her infant's need to explore his environment. Attachment behaviours such as approaching, following, clinging, smiling, calling, and crying are seen as innate strategies designed to promote proximity to, or contact with, the caregiver. As the dyadic relationship develops these behaviours become more sophisticated and evolve into patterns of interaction. These patterns coalesce over time and are organized into what Bowlby (1973) called 'working models'. Such models allow the child to simulate his or her behaviour and the responses of significant others so that the consequences of future behavioural goals can be reliably predicted.

According to this theory, attachment provides a frame of reference and the security necessary for the child to undertake the long years of social learning that occurs during the preschool years and continues through into adulthood. It also

provides a major incentive in the acquisition of socially appropriate behaviour and, by implication, the inhibition of socially inappropriate behaviour. The desire to gain the approval of significant adults is seen to be a powerful motivation in learning to control equally powerful but less desirable urges (Mischel, 1976).

7.8 Different styles of attachment

By observing the caregiver-child interaction prior to, during, and after, a brief separation, Ainsworth (1969, 1979) identified three types of attachment in white, middle-class, American families: secure attachment, anxious attachment, and avoidant attachment. The secure child protests when her mother leaves the room, seeks her out while she is gone, and greets her with delight when she returns. The child also explores more when her mother is present. An anxious attachment is demonstrated by the distress of the child when mother leaves. He gains little relief upon her return, and is loathe to explore even when she is present. Avoidant attachment is shown by relative indifference to mother. The child rarely cries when she leaves the room and there is little positive response on return. Curiosity is unaffected by mother's presence. This experimental paradigm called the 'Strange Situation' is widely used in clinical assessment and is particularly helpful in cases of suspected abuse in young children.

Crittenden (in Belsky & Nezworski, 1988) identified a fourth style of attachment during her work with children considered at risk. She observed a group of children who became compulsively compliant or conforming in response to overcontrolling, abusive mothers. They also inhibited signals of displeasure even in situations that warranted protest. These children were often classified as securely attached as their behaviour most closely approximated that group. This attachment style is of particular interest to us because it is characteristic of children and adults classified as repressors (or suppressors) and also the sub-group of children demonstrating a compulsively compliant attachment style in conjunction with conversion symptoms (sections 6.7).

Crittenden's finding suggests that individuals who compulsively suppress their emotions and those presenting with somatoform disorders have developed an insecure attachment style during early socialization. For example, if a mother repeatedly responds to her infant's expressions of negative affect (e.g., crying, agitated body movements) with anger, or withdrawal of attention and care, as clinicians have observed, the infant is likely to inhibit such negative affect; an observation consistent with the principles of operant conditioning. If this pattern of interaction continues throughout childhood, what Crittenden (2006) calls 'disorders of inhibition' may develop.

7.9 The Dynamic-Maturational Model of attachment (DMM)

In her DMM of attachment, Crittenden (1995, 2006) refers to the pattern of attachment (or self-protective strategy) developed in response to punishment or neglect by caregivers, as 'Type A'. Individuals using this strategy learn to minimize awareness of negative affect, and 'to do that which they expect will be reinforced and avoid doing that which they expect will be punished' (Crittenden, 2006:106). Specifically, the strategy is the outcome of processing the temporal order of stimulation (that is, the order in which stimuli occur). On this view, disorders of inhibition and compulsion may be tied to too great a reliance on awareness of the rules governing reward and punishment.

Anxiety may be much higher, however, when the child is uncertain about the behaviours that will lead to punishment and those that will not. It may be especially difficult for a child to cope adequately if punishment from a parent (and/or significant others) is unpredictable so that he is unsure of what to expect. In that case the child may experience a more generalized dread, because threat and punishment are possible at almost any time and place. While this type of threat typically elicits avoidance behaviour or 'flight' in laboratory animals, avoidance in young humans is problematic, particularly when the source of threat is a primary caregiver from whom he cannot escape.

Crittenden (2006) also identifies a lack of consistent parental responses to a child's behaviour as a criterion for developing what the author calls 'Type C' strategies. Lacking internal working models for guiding behaviour, the child relies on emotion cues as they occur in himself in response to his social environment (Crittenden, 2006). The crucial stimuli he attends to are negative. In a gradient of increasing intensity such primary aversive stimuli range from a need for comfort—to anger, fear, and pain. In other words, while individuals using Type A strategies tend to minimize their awareness of such negative stimuli and rely on rules associated with reward and punishment to guide their behaviour, people using Type C strategies do the exact opposite. As a result, their behaviour is often exaggerated. This is particularly true in the case of pain behaviour with many children and adults reporting feelings of pain that are not commensurate with their injury or illness (section 7.1).

Again, if the behaviour is reinforced by the social environment, (parents, medical practitioners, etc.), such exaggerated displays may become habitual ways of communicating distress and for gaining care and attention, thereby reducing anxiety. On this view, many (but not all) of the anxiety disorders are tied to too great a reliance on the organizing, motivating, and communicative functions of negative affect (Crittenden, 2006). Such disorders include anxious fearfulness, agitation, disorders of aggression, and separation-based disorders (Crittenden, 2006).

Kozlowska (2007) has attempted to expand Crittenden's model of attachment by incorporating recent neurobiological findings into its framework. The author also points to the important role of classical and operant conditioning in the development of strategies or patterns of attachment.

7.10 The phylogenetic origins of conversion disorders

Kozlowska (2007) has chosen the DMM as a framework for understanding conversion reactions as having their phylogenetic roots in two different innate

animal defense behaviours: the 'freeze response' and 'appeasement defense behaviours' discussed briefly in section 4.7.2. From this perspective, conversion symptoms reflect two distinct, threat-elicited emotional responses which are primed in context-dependent developmental experiences (pathways), and underpinned by different neurobiological mechanisms. Stated simply for the moment, Kozlowska identifies these two defense reactions with the basic attachment strategies proposed by Crittenden (2006), namely, Types A and C respectively. Using various case studies Kozlowska describes the different conditions that are likely to chronically elicit one or the other of these defense strategies.

7.10.1 The freeze response and Type A strategy

The opioid-mediated freeze response (immobilization) is likely to be elicited in the context of punishment from caregivers. It involves a widespread inhibition of motor and sensory responses to distress, including co-morbid conditions characteristic of hypoarousal, such as inhibition of negative affect, pain processing, and depression. These reactions are seen to occur in infants and young children whose cognitive systems are not sufficiently developed to enable faking or the formation of false beliefs. As we saw in the case of baby Harry (section 3.5), developmentally, automatic emotion regulation (AER) occurs prior to and without the intervention of brain systems that would eventually allow Harry to become aware of such emotions and to consciously suppress them.

Thus, until a child is capable of sustaining suppression, clinicians often observe manifestations of fear interspersed with motor inhibition. For example, one case reported by Kozlowska (2007) involved a 15-month-old toddler, Peter, who presented with intermittent freezing of his left arm, jerky movements, intermittent stilling of his body, and strained vocalizations. When unable to maintain inhibition, intrusions of negative affect (e.g., anger, fear, or desire for comfort) were observed (Kozlowska, 2007).

Clinically, a failure to completely inhibit the disposition to act, observed in these distressed children, emerges in the form of 'out-of-control' body movements (pseudo-seizures), or an unexplained loss of muscle tone, accompanied by an apparent loss of consciousness and memory which Kozlowska (2007) explains as an 'intact inhibition of subjective awareness'. Many older children report symptoms of limb paralysis before or after pseudo-seizures, which the author interprets as the child's intense efforts to regain control (inhibit motor response). However, again, it needs to be pointed out that AER and cognitively mediated expressive suppression, according to the previous analysis, are subserved by different regions, (and hemispheres), of the brain.

With neurological maturation, some children may also display concomitant false positive affect or 'parent-pleasing' (rewarded) behaviours as part of their clinical presentation, indicating that operant conditioning has occurred. That is, the child who displays false positive affect has learned there is something she can do to secure a minimum of safety and comfort in an otherwise threatening family environment. For example, Mirna, a four-year-old pre-schooler, displayed intermittent freezing of her left arm during the Strange Situation procedure when in the presence of her father. As the investigator reported, on several occasions, when her father returned to the room, Mirna greeted him with 'over-bright positive affect, including a 'happy' face. This apparent cheerfulness was belied by asymmetrical limb movements (one arm swinging as she moved, the other being frozen); the frenetic nature of her play; and the suddenly anxious high pitch of her voice', (Kozlowska, 2007:494). To what extent this child was aware of her conditioned behaviour, and thus, capable of feigning 'happiness', is a matter of conjecture. However, at the age of four, implicit rather than explicit deception is most likely.

As we have also seen, submission is not always evidenced by appeasement displays. Other children, as Perry (2001) and his colleagues have observed, remain in an opioid-mediated hypoarousal state possibly because there is nothing

they can do to avoid abuse. They learn to 'dissociate' or 'switch-off' in the midst of terror.

7.10.2 Proposed neurobiological correlates of the freeze response

Based on neuroscientific research, Kozlowska understands the freeze response, particularly in infants and young children, to be mediated by the amygdala and related lower-level systems. However, according to recent research, this would only be true if the fear stimulus was a natural predator—an unconditioned stimulus (Corcoran & Quirk, 2007). Recall that these investigators found that although the amygdala is necessary for the acquisition and expression of unconditioned fears, the integrity of the OFC is essential for the execution of a classically conditioned (learned) fear response. In the animal world a parent is not generally considered to be a predator of its offspring, although child psychologists might argue otherwise.

While freezing or immobilization is thought to have evolved in the context of predator-prey situations, a number of contemporary studies on hunter-gatherer tribes emphasize that conspecific-induced mortality has been an important feature of human evolutionary history (Cantor, 2005). The freeze response has thus evolved in the context of both predatory *and* conspecific threat. As these researchers remind us, high rates of child abuse are a pervasive feature of so-called 'safe' western societies (Perry, 2001; May-Chahal & Cawson, 2005; Kozlowska, 2007).

However, the findings from neuroimaging studies conducted with individuals diagnosed with conversion disorder, although few in number, tend to show that the OFC rather than the amygdala is activated when individuals diagnosed with various hysterical paralyses attempt to move the afflicted limb. This finding, in conjunction with the Corcoran & Quirk (2007) study, suggests that the parent-child relationship is not intrinsically one of predator-prey. An infant must first learn to associate a caregiver with threat.

In the course of socialization it is common for significant persons who nurture the child and care for him, and to whom he becomes most deeply attached, to also be a source of threat. Thus, the same social stimulus (e.g., the mother) associated with positive rewards and feelings of pleasure, is also associated with punishment and feelings of fear or anxiety. The phenomena of 'ambivalence' and 'conflict', given a prominent role in the etiology of hysteria since Freud, may result whenever the same persons who evoke positive feelings and approach tendencies are also the sources of negative emotions and avoidance reactions. To a more or less degree, all humans suffer emotional ambivalence towards their parents as a corollary of socialization; an observation that has led to many child psychologists criticizing harsh child-rearing practices associated with the Protestant ethic (Mischel, 1976). As Mischel (1976) points out, this duality is common in life and does not end with childhood. In an adult's life, for example, the same spouse who gives love may also be the source of bitter frustration.

In the normal course of events, anxiety tied to such ambivalence and conflict will probably be minimal. However, as shown, disorders of inhibition may develop when a parent is unduly overly controlling or abusive. Over conforming children, that is, 'good' children who are compulsively compliant, (Crittenden, 2006; Kozlowska, 2007) tend to use this strategy in order to avoid punishment and to gain the approval of hard to please parent/s.

Western societies have now perhaps swung too far in the opposite direction, as so often appears to be the case, particularly on social issues such as child-rearing. As Crittenden (2006) has argued, anxiety may be particularly high in children who lack clear guidelines for predicting the outcome of their behaviour. According to Kozlowska (2007) this situation is typical for children raised in chaotic family environments where parents are subject to unpredictable outbursts of violent behaviour. While children with over-controlling parents learn to manage their predicament by adhering to rules that govern reward and punishment, children

who lack such guidelines may, as Crittenden (2006) points out, use emotional information as a guide to behaviour. Lacking models for predicting how parents will respond to their aggression, they use the contingencies tied to exaggerated affective displays to elicit more predictable responses from parents. It cannot be overemphasized that such behaviour is not initially an act of explicit deception. A view that is shared by Kozlowska (2007):

> The organization of attachment behaviours, (or strategies) in infancy and childhood occurs in the context of repeated sequences of parent-child interactions. It involves implicit learning (Skinnerian and Pavlovian learning principles) and does not require the child to be subjectively aware as to how they are behaving (Kozlowska, 2007:493).

In the context of conflict with a caregiver, children who have developed what Crittenden (2006) calls Type C strategies, often use appeasement displays to signal submission to the angry adult. Appeasement behaviours are operantly conditioned responses that the child has learned disarm parental aggression (Price, et al., 2004; Kozlowska, 2007). Thus, submission may be an innate response triggered in an animal (including humans) when dealing with a dominant, aggressive conspecific, but the form the signal of submission takes depends on the animal's species specific responses to danger and learning history. That is, nature *and* nurture interact to produce behaviour that is adaptive within the context of the conflict situation.

7.10.3 Submission with appeasement displays and the Type C strategy

Submission and appeasement are seen by Kozlowska (2007) as the phylogenetic origins of Type C strategies (Crittenden, 2006). Perhaps the most obvious point to make about appeasement displays is that in infrahumans they are observed to occur in the context of intraspecies conflict rather than predator-prey situations (see section 7.10.2). Appeasement displays would not help a rat to deal with the threat posed by the close proximity of a cat. Nor does it aid the infant or child under threat from physical or sexual abuse who has learned from past experience that there is nothing he or she can do to deter the abuser from attack.

Although submission and appeasement are terms often used interchangeably by investigators who support the hypothesis that somatoform disorders are reflections of this defense mechanism, I tend to agree with Kozlowska (2007), that appeasement displays signal both submission *and* the submitting animal's willingness to reestablish friendly relations with the dominant, angry, conspecific. On this view, we could say that submission is not always coupled with appeasement displays or, for that matter, with dissociation as defined by Perry (2001).

The child who responds to threat with submission and appeasement displays can be identified by signals that as Kozlowska (2007) observes, often appear contradictory but when understood in the context of operant conditioning make sense. Children and adults who respond to emotion signals from conspecifics rather than from learned 'if ... then' rules, are reacting adaptively to conflicting signals actually received from their caregivers during interaction.

Price and his colleagues refer to the alternation of extreme emotional behaviours in the same individual as escalation and de-escalation. In some cases such extreme hyperarousal and hypoarousal states are manifested in manic and depressed behaviours respectively, suggesting that yet another little understood psychiatric disorder (manic-depression or bi-polar disorder) may have its origins in early childhood attachment experiences that foster Type C strategies.

As pointed out, appeasement displays may take many forms depending on the person's level of arousal and the behaviour subject to operant conditioning. On the behavioural view, any response which serves to disarm the aggressor or is successful in gaining attention and care, will be reinforced. Thus, if the child uses illness as a part of his behavioural repertoire, such behaviour would either have been directly rewarded in the past or observed to be rewarded in others. As Stuart & Noyes (1999:34) state:

> Early exposure to illness increases the likelihood that distress will be

> manifested somatically. When under stress as adults, somatizers use physical complaints to elicit care.

This view is also shared by Kozlowska (2007) Although it is common for many medical practitioners to understand illness behaviour or somatization as a bid for attention, the patient is often dismissed or treated with impatience for wasting valuable time. This lack of compassion shown by the busy general practitioner is understandable, however, it only serves to exacerbate the patient's feelings of rejection and, as a consequence, her illness behaviour, often resulting in 'doctor-shopping' (see section 7.1).

The intense distress felt by individuals who have developed Type C strategies is very real. Children manifesting a predominantly hyperarousal state show increased muscle tone (in contrast to a decrease in muscle tone observed in children using Type A strategies); increased startle response; low-grade increases in temperature; disturbed patterns of sleep, and cardiovascular indicators of distress, such as increased heart rate, and pulse (Perry, 2001). Such symptoms of hyperaousal may alternate with chronic fatigue and depression (Price, et al., 2004) indicative of hypoarousal. As Perry (2001) and Schore (2003) point out, children raised in a dysfunctional or violent family environment often appear to shift between these extreme states of arousal manifested in behaviours that phenotypically appear contradictory.

7.10.4 Implicit or explicit deception?

Although the Type C strategy and its variants are learned implicitly it is difficult to state categorically that an older child's appeasement displays are not deliberately contrived. As previously noted, learning theorists have shown that once an individual has learned that she can manipulate others by acting in a certain way, the potential for explicit deceit is not only possible but likely. Consider the following case reported by Kozlowska (2007) as an example of what the author calls 'implicit deceit' but might be interpreted as explicit deceit by others.

Louis, was a 10 year-old boy who four months prior to his presentation of symptoms had been assaulted by class-mates. In the attack he had suffered an uncomplicated crush fracture of his T10 vertebra. Despite medical opinion that he should be asymptomatic, the child continued to present to the hospital with recurrent falls, intermittent leg weakness, and incapacitating pain.

The clinician reports that Louis was an anxious child, the second of three children of a socially isolated immigrant family. His mother suffered from panic attacks in the context of a major depression. His father suffered depression and back-pain in the context of a decade-old work-related injury. There was marital discord and inconsistency in how each parent managed their child's behaviour. Louis's mother tried to set behavioural limits by pleading or using various threats, whereas his father gave in to most demands but would intermittently explode with anger.

Louis's symptoms were florid. On attempting to walk, the child clutched onto staff, walls, and furniture for support, and he moved with an exaggerated, slow limp. Attempts to walk were accompanied by loud, painful wailing, along with frequent demands for analgesia and for use of a wheelchair. Louis's parents responded to these displays by soothing him, feeding him, carrying him, and organizing their lives around his needs. They bought clothes, toys, and videos—even shopping in the middle of the night to soothe Louis's wailing. When his parents became frustrated, angry, or tearful, Louis would disarm their frustration and reengage them in caregiving interactions by displaying his vulnerability via coy facial expressions alongside displays of intensified pain and deterioration of gait.

Although we cannot say conclusively that Louis was deliberately manipulating his parents and medical professionals, it does seem likely on the evidence. As Kozlowska (2007) acknowledges, children using Type C strategies coerce

(manipulate) their unpredictable parents into responding in ways that meet their need for attention or to disarm aggression. Also, this interpretation is consistent with the view expressed by the author in a later section where she describes explicit deception as involving 'the conscious intent to deceive by careful regulation of what the deceived individual is allowed to know and is manipulated to feel and do' (Kozlowska, 2007:503).

Kozlowska (2007) is right to argue that implicit deception does not require the child be subjectively aware as to how they are behaving but nor can we preclude such awareness. I would suggest that the above case used by Kozlowska as an exemplar of implicit deception, contains evidence to support the claim that Louis was deliberately using his past injury as a means of manipulating his parents and medical staff and thus, according to the author's definition, this patient's behaviour suggests the use of explicit deception.

As Kozlowska (2007) goes on to report, explicit deception may be manifested in patients whose symptoms originally reflected an implicit need for attention and comfort but who have deliberately chosen not to abandon illness behaviour as a means of controlling other people or situations. For example, feigned back-pain may be used to evade unpleasant duties or social obligations. Because the systematic, strategic use of explicit deception over an extended time requires significant neurological maturation, patients who feign unexplained neurological symptoms and chronic impairment should more commonly be adults, not children (Kozlowska, 2007).

This hypothesis is consistent with clinical observation, where physicians treating adults have historically encountered more feigning than have pediatricians (Turner, 1999; Kozlowska, 2007). However, Jureidini & Taylor (2002) have observed the sustained use of illness behaviour in older children. No doubt children raised in threatening family environments that call for extreme defense strategies quickly learn to be manipulative in order to protect themselves from

harm.

7.10.5 Proposed neurobiological correlates of appeasement displays and Type C strategies

Although Kozlowska (2007) claims that the neural substrate for appeasement displays are different to those for the freeze response, we can only infer, on the basis of our previous knowledge, what systems are involved as the author does not enlighten us. For some sort of clarity on the neural and endocrinological correlates of appeasement displays and Type C strategies we need to turn to the work of other investigators.

First, submission has been functionally equated to flight by Price and his colleagues, as both strategies entail a de-escalation of behaviour or a discontinuity of escalation characteristic of the fight response (Price, et al., 2004). In other words, submission and appeasement may terminate a conspecific encounter as does flight, and in addition, restores harmony with the angered conspecific. In de-escalation the individual gives up any chance of winning, but reduces the cost of losing (Price, et al., 2004). As these investigators explain, 'De-escalation may take the form of departure, but in group-living animals submission with appeasement display represents the more common form of de-escalation' (Price, et al., 2004:3).

De-escalation and escalation are viewed by Price and his colleagues as alternative agonistic strategies that result in normal communicative behavioural states but at times manifest as depression and mania, respectively. The concepts of de-escalation and escalation in relation to social behaviour may possibly be underpinned by the activity of the two branches of the ANS responsible for regulating behaviour—the parasympathetic branch via the opioid system may result in a de-escalation of behaviour that is unrewarded, while the sympathetic branch via adrenaline and noradrenaline may result in an escalation of behaviour. Both responses are influenced by the OFC and amygdala which , as we have seen,

compute the costs and benefits associated with behavioural goals according to past experience and the present situation.

If this view is correct, exaggerated appeasement displays such as illness behaviours, if implicitly generated, would involve the parasympathetic branch of the ANS and opioid system, resulting in a de-escalation of inappropriate (unrewarded) behaviour. However, if explicitly or deliberately enacted in order to manipulate the behaviour of others, previous studies on the neural correlates of feigned behaviour would suggest the involvement of different brain regions to those observed in implicitly motivated behaviour. Feigners exhibit hypofunction of the RH frontal cortex and activation of the LHDLPFC (Spence, 2000; Oakley, et al., 1999). The DLPFC as we know is specifically activated in consciously willed action.

Thus, neuroimagining could, in principle, help the clinician to know whether a child like Louis is deliberately feigning or whether, like the chalk-tossing professor, such behaviour is generated under the influence of operant conditioning and thus implicit.

These considerations raise the question of whether appeasement displays should be exclusively identified with Type C strategies as Trish, classified as using a Type A strategy, switched to a Type C strategy (explicit deceit) when threatened with the prospect of returning to her mother. In addition, children who display rewarded behaviours in order to avoid punishment or to elicit parental approval (typical of children observed to use Type A strategies), could be seen as using a form of appeasement. If submission is not inherently linked to appeasement displays as I have argued, but can also be manifested in dissociative states and behaviours, then I see no reason why we should not consider implicit deception to be a characteristic of a successful Type A attachment style. Just as the child using Type A strategies may fake positive emotions, so the child using Type C strategies may fake illness behaviours. Both strategies are first learned implicitly

through operant conditioning. If, and when, the association between a behaviour and response are consciously recognized, the child can use the strategy to deliberately avoid punishment or gain reward.

For the purposes of treatment Kozlowska (2007), following Crittenden (2006), argues that a distinction should be made between pseudo-neurological disturbances that have their origins in the freeze response (Type A strategies) and those enacted as a means of gaining care and attention from otherwise disattentive or inadequate caregivers (Type C strategies).

7.11 Clinical and methodological implications

Children who present with conversion symptoms (Type A strategies) will require individual and family psychological interventions that (a) promote recognition of family and contextual factors threatening the child's emotional wellbeing; (b) modify unhelpful parental behaviours and expectations; and (c) assist the child to have a better awareness of body states, and an increased capacity to communicate negative feelings. In a small subset of these families where parents are extremely hostile, child protection issues may need to be addressed (Kozlowska, 2003, 2007).

On the other hand, children who habitually use illness behaviour as a means of communicating submission and appeasement, or to regulate parental behaviour, require a different type of intervention. Treatment would include family therapy that aimed to (1) increase parental consistency; (2) decrease levels of anxiety and unpredictability in the family system; and (3) render the child's appeasement displays nonfunctional in terms of eliciting comfort and protection (Kozlowska, 2007).

According to Kozlowska, the capacity to differentiate between these two distinct groups of patients is deemed to be important not only for treatment but also for the correct interpretation of functional imaging studies (Kozlowska, 2007).

However, people using either Type A or Type C strategies are both likely to show activation of the RHOFC given that AER appears to selectively involve this brain region in humans. Further studies with larger patient groups are needed to clarify these preliminary findings.

In conclusion, Kozlowska's use of the DMM of attachment as an integrating framework for neurobiological and evolutionary theories of conversion reactions might be seen as politically expedient. Attachment theory and associated methodologies are well accepted among orthodox clinicians and may thus provide a way of introducing concepts into the field of psychiatry that may otherwise meet with radical dissent.

Attachment theory relies heavily on the principles of associative learning identified by early behaviourists and I am not convinced that its tenets provide any new significant knowledge about the behaviour of animal's under threat. Perhaps its main advantage lies in the fact that it specifically describes human conditioning in controlled conditions, and is therefore more intellectually acceptable to some psychologists and psychiatrists who find it difficult to accept the relevance of infrahuman research to human behaviour.

This is not to say that all we need to understand hysteria is a grasp of classical and operant conditioning. A study of the endrocrinological and neural correlates of defense moves us beyond a strictly behavioural account of such disorders. It has been shown that genetically programmed neuroendocrine responses to threatening stimuli which normally prepare the organism for defense, form the bases of much somatic phenomena characteristic of hysteria. For example, the discovery of opioid receptors in areas of the brain sensitive to rewarding and punishing stimuli, has helped to explain the sensory and motor disturbances experienced in these chronically distressed individuals. In turn, the subjective perception of such disturbances and the way the person interprets them, also needs to be factored into any causal explanation of hysteria, given the effect of conscious cognition on the

systems responsible for processing affective stimuli.

Kozlowska (2003, 2007) has attempted a synthesis of these factors in her theory of conversion disorder, paving the way for a biopsychosocial model of hysteria which promises to bridge the Cartesian mind-body divide.

Conclusion

The attachment theory of somatization suggests that the tendency to develop stress-related symptoms exists within each of us given that ambivalence and conflict between caregiver and child is an inevitable corollary of the socialization process. Few people escape the experience without bearing some emotional and psychological 'scars', which continue to affect their interpersonal relationships throughout the lifespan, particularly when they are feeling insecure or anxious.

For some individuals raised in the type of dysfunctional family environments discussed, over-burdened homeostatic mechanisms may fail to regulate their arousal states, resulting in somatic and mental problems that cannot be explained within the current biomedical framework. The person suffering hyperarousal or hypoarousal states and their physical and social consequences feels and behaves abnormally, but is she ill?

From the conventional medical viewpoint she is not, as there is no organic pathogen or lesion to account for her symptoms. Nor is there evidence of biochemical or neurophysiological brain dysfunction to qualify her problem as a mental disease.

Somatoform disorders should not then, be considered 'mental' illnesses. Rather, the cause of the abnormal physical and mental states are social adjustment reactions to stressful living conditions, and therefore, not properly the concern of the physician-psychiatrist. As so often observed from Freud onward, when stress is reduced by venting or, by changing the patient's environment, the symptoms abate or disappear altogether.

Functional brain imaging also confirms the transient nature of abnormal patterns of activation associated with conversion hysteria —when the symptoms cease the

pattern of activation returns to normal. If there were actual organic damage, simply expressing one's distress would not have the observed result. The endocrinological and neurobiological changes that occur in people diagnosed with somatoform disorders or sub-types such as conversion disorder, are the biological reactions to threat or perceived threat—this much seems obvious from the research findings.

The question then becomes: 'Do we need a new medical model as proposed by many clinicians who argue that the existing model promotes mind-body dualism?' The biomedical approach to disease has been successful beyond all expectations, and there is no reason to believe that advances in medicine, surgical techniques, and so on, will not continue to alleviate the misery and suffering caused by physical disease.

On the other hand, medicine cannot help to alleviate the misery and suffering caused by social conditions that are not conducive to well-being—conditions that exceed the person's capacity for adjustment. Ameliorating such suffering is not, however, within the brief of the physician. Society provides nonmedical professionals for this task: social workers, family therapists, counsellors, and so on. The role of the doctor is essentially to locate and diagnose disease in the patient and to offer treatment and advice that will, if successful, restore his health.

If the doctor suspects that the person's living conditions or habits are contributing factors in the development and persistence of disease, e.g., alcoholism, drug dependency, or abuse, the best that can be done is to refer the patient to an appropriate skilled professional. Similarly, if a parent reports her child is sick because he cannot move a limb or has a behavioural problem, after satisfying himself that the problem is not caused by disease, the doctor has no alternative but to inform the parent accordingly and suggest she seek the help of a family counsellor. If the child shows signs of physical or psychological abuse, then he is obliged to report his findings to the appropriate authority for follow up.

General practitioners, particularly in modern western societies, do not have the time or skills to take on the role of educator and psychotherapist. Even if their role was to be extended, we have seen that suggesting to a patient, or the parent of a patient, that the symptoms presented may be due to stress, is likely to receive a hostile response.

I would suspect that although the literature reveals much defensive cynicism regarding patients with somatoform disorders, most practitioners do consider the psychosocial aspects of their patient's condition. In fact, they overemphasize them— probably due to a lack of knowledge about the biological effects of stress. As Taylor (1989) has pointed out, medical students learn a little about the 'flight or fight' response but nothing about reactions to threat in circumstances where neither reaction is an option.

Even if it were to become routine to identify the neurobiological markers discussed in the preceding chapters, they would mean little to either the patient or doctor without the conceptual tools necessary for relating these findings to subjective experience and behaviour. As indicated at the outset of the present work, education, both in the public and professional domains, is the key to providing the much needed concepts that will help us to not only understand the problem of conversion hysteria but also the more philosophical mind-body problem.

APPENDIX

'Trish' – A case study

Removed due to copyright restrictions. Case study can be found on pages 82-85 in the following article:

Kozlowska, K 2003, 'Good Children with Conversion Disorder: Breaking the Silence', *Clinical Child Psychology and Psychiatry*, vol. 8, no. 1, pp. 73-90. https://doi.org/10.1177/1359104503008001008

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