“Scared Stiff”: Catatonia as an Evolutionary-Based Fear Response

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Catatonia, long viewed as a motor disorder, may be better understood as a fear response, akin to the animal defense strategy tonic immobility (after G. G. Gallup & J. D. Maser, 1977). This proposal, consistent with K. L. Kahlbaum’s (1874/1973) original conception, is based on similarities between catatonia and tonic immobility (“death feint”) as well as evidence that catatonia is associated with anxiety and agitation and responds dramatically to benzodiazepines. It is argued that catatonia originally derived from ancestral encounters with carnivores whose predatory instincts were triggered by movement but is now inappropriately expressed in very different modern threat situations. Found in a wide range of psychiatric and serious medical conditions, catatonia may represent a common “end state” response to feelings of imminent doom and can serve as a template to understand other psychiatric disorders.

Catatonia is clearly one of the most perplexing of all psychiatric syndromes. For no apparent reason, a person may become mute, freeze for minutes or hours on end without any discernable awareness of the outside world, appear seemingly impervious to pain, and allow their limbs to be bent in all sorts of awkward positions. How can such a strange response, found in a wide range of psychiatric and medical conditions, be understood? Is it simply due to something having gone terribly wrong in the person’s brain, perhaps in areas responsible for movement? Or is the response one that has been bred into the human species for generations on end but was designed to deal with very different situations from the ones in which it is currently expressed? Although these are clearly not mutually exclusive possibilities, it is the latter perspective, rarely considered, that will be emphasized here.

Historical Overview

The concept of catatonia originated with the German physician Karl Kahlbaum, who coined the term in 1869 and described it in detail in his 1874 monograph “Die Katatonia oder das Spannungs-irresein,” which has been translated as “Catatonia or the Tension-Insanity” (Mora, 1973). Kahlbaum (1874/1973) based the concept of catatonia on a previously described condition, melancholia attonita—“astonished” or “thunderstruck” melancholia—so-called because of the astonished expression often seen on sufferers’ faces (Goldar, 1988; J. Johnson, 1984). He viewed catatonia as a progressive disorder with strong links to affective and organic conditions, usually beginning with a change of mood but sometimes starting abruptly, particularly after “very severe physical or mental stress . . . [such as] a very terrifying experience” (Kahlbaum, 1874/1973, p. 31). Kahlbaum described the primary characteristics of catatonia as follows:

The typical signs . . . may be described as a state in which the patient remains entirely motionless, without speaking, and with a rigid, mask-like facies, the eyes focused at a distance; he seems devoid of any will to move or react to any stimuli; there may be fully developed “waxed” flexibility, as in cataleptic states, or only indications, distinct, nevertheless, of this striking phenomenon. The general impression conveyed by such patients is one of profound mental anguish, or an immobility induced by severe mental shock. (Kahlbaum, 1874/1973, p. 8)

As is obvious from the above quotes, Kahlbaum (1874/1973) was not unaware that persons with catatonia gave the appearance of having been frightened. Indeed, an alternate translation of the same passage, quoted (but not referenced) in Goldar and Starkstein (1995), reads: “All in all, these patients give the impression of the deepest mental pain, of being frozen after a great fright” (p. 202). In a milder form, of course, this experience is not unknown to the general population, as reflected in the common expression—being “scared stiff.”

The thesis that catatonia may primarily be a fear reaction, closely related to the animal defense strategy tonic immobility (TI; immobility characterized by muscular tension), is not a new one. Although TI has occasionally been referred to as catatonic in the animal literature (as opposed to the more common usage of cataleptic) and proposed in passing as a potential model for catatonia (Klemm, 1971; Svorad, 1957), the first (and only) sustained exploration of the relation between these two conditions was mounted in 1977 by animal researcher Gordon Gallup and psychiatrist Jack Maser.1 Noting the numerous behavioral parallels between TI and catatonia (such as immobility, decreased vocalization, analgesia, “waxy flexibility,” and evidence of alertness), they concluded that catatonia could contain evolutionary-based “fragments of primitive defenses against predators that now misfire under conditions of exaggerated stress” (Gallup & Maser, 1977, p. 357).

This position was later taken up by Perkins, who, in a 1982 article entitled “Catatonia: The Ultimate Response to Fear?,” characterized catatonia as a primitive form of expression released under conditions of extreme stress. Subsequently, Orland and

1 Even so, Gallup and Maser’s (1977) contribution is primarily about TI. Just a small portion of the chapter is dedicated to a discussion of catatonia, and only seven references on catatonia, three from prior to 1950 and one from a textbook, are cited.
Daghestani (1987), in a case study, raised the possibility that organically based catatonias could be "psychological reaction(s) to overwhelming fear and the stress of the organic brain disease state itself" (p. 490). Berrios (1981), reviewing the historical concept of stupor (one of the core components of catatonia), wondered whether it might be a "vestigial ethological response" (p. 474) and proposed freezing in animals as a research model. Finally, Krystal (1993) argued that cataplexy or catatonoid reactions seen in humans under conditions of profound helplessness were akin to animal immobility, both of which he characterized as surrender responses in the face of unavoidable danger.

However, none of these writings appears to have had much influence on the concept of catatonia. The notion of catatonia as a fear reaction has been given short shrift, as the view of catatonia as a motor disorder remains overwhelmingly dominant. As an indication of this, Perkins’s (1982) article has been cited only once in the 20 years since its publication (by Orland & Daghestani, 1987) according to the Social Sciences Citation Index (which references only articles and hence not Gallup & Maser’s, 1977, contribution). Although Perkins’s article is largely a review of several case studies, this lack of citation no doubt reflects limited interest in the question posed by his title. As this thesis, appearing in various forms over the past 25 years, has generated so little interest, why should it be reconsidered now? There are a number of compelling reasons to do so.

First, the last 15 years has seen a resurgence of interest in individual in catatonia, with the resulting developments:

1. Catatonia is no longer viewed solely as a subtype of schizophrenia. Rather, it is now seen as a largely non-specific, present in close to 10% of psychiatric—primarily affectively disordered—patients (Taylor & Fink, 2003) and appearing in a wide range of serious medical conditions (Carroll, Kennedy, & Goforth, 2000; Fink, 2001; Fink & Taylor, 2003; Peralta, Cuesta, Serrano, & Martinez-Larrea, 2001; Pfuhlmann & Stoeber, 2001). This is reflected in the inclusion of catatonia as a modifier for both affective disorders and medical conditions as well as a subtype of schizophrenia in the Diagnostic and Statistical Manual of Mental Disorders (4th ed.; DSM–IV; American Psychiatric Association, 1994).

2. Benzodiazepines (BZs) have been found to be extremely effective in treating catatonia, in some cases completely relieving symptoms within a matter of minutes (Bush, Fink, Petrides, Dowling, & Francis, 1996b; Schmidt, Standhart, Deuschle, Drancoli, & Heuser, 1999; Ungvari, Kau, Wai-Kwong, & Shing, 2001). Although the mechanisms underlying this response have been debated (Menza & Harris, 1989), it appears most likely that it is the powerful antianxiety properties of BZs (as opposed to their anticonvulsant or muscle relaxant properties, for example) that are implicated (Northoff, Steinke, et al., 1999; Northoff et al., 1995; Rosebush & Mazurek, 1999).

3. The subjective state of many persons undergoing catatonic experiences is reported to be of intense anxiety (Northoff, Krill, et al., 1998; Rosebush, Hildebrand, Furlong, & Mazurek, 1990; Rosebush & Stewart, 1989); despite their apparent unresponsiveness, they often have a surprising level of awareness of events going on around them (Rosebush & Mazurek, 1999).

In addition, recent research on catatonia has revealed further areas of overlap with TI in animals—such as frontal dysfunction (Klemm, 1971; Northoff, 2002; Taylor, 1990)—not previously noted. Advances in understanding the role of the orbitofrontal cortex, specifically implicated in catatonia (Northoff, 2002), in regulating affect and emotionally driven behavior is also of relevance, as is Porges’s (1995, 1997, 2001) polyvagal theory of the role of the vagus nerve and autonomic nervous system in regulating emotion, conflict, and communication. Finally, the burgeoning field of evolutionary psychology and psychiatry, and in particular evolutionary factors proposed to underlie depressive disorders, provides an appropriate framework for such an exploration (Brune, 2002; Buss, 1995; Dixon, 1998; Gilbert, 2001; Gilbert & Allan, 1998; Jones & Blackshaw, 2000). Thus, the time appears right for a reconsideration of the role of fear in catatonia, with special focus on its relation to the animal defense strategy TI.

Initial Formulation

The thesis put forward here is that catatonia is a relic of ancient defensive strategies, developed during an extended period of evolution in which humans had to face predators in much the same way many animals do today and designed to maximize an individual’s chances of surviving a potentially lethal attack. It is argued that catatonic episodes reflect an inappropriate expression of these strategies, primarily in situations in which the individual feels himself or herself to be under profound threat—from either internal (i.e., toxic reactions) or external sources.

Specifically, it is proposed that different components of catatonia are differentially related to particular animal defense strategies. Thus, the core catatonic symptoms of stupor, mutism, and immobility are directly linked to TI. However, catatonia, as it is currently formulated, consists of more than just these symptoms, which make up catatonic stupor (also called akinetic or retarded catatonia). Catatonic excitement ("apparently purposeless agitation not influenced by external stimuli"; American Psychiatric Association, 2000, p. 764) is not infrequently seen, as are behavioral or communicative abnormalities such as repeating an examiner’s movements or statements (echopraxia or echolalia) or negativism ("apparently motiveless resistance to instructions or attempts to be moved"; American Psychiatric Association, 2000, p. 765). It is proposed that catatonic excitement (or hyperkinetic catatonia), although not directly related to TI, is related to the sympathetically mediated fight-flight responses that typically precede or follow TI.2 The remaining associated features, which I have termed behavioral–communicative symptoms, although having no direct
Animal Defense Reactions of Immobility

One of the basic tenets of evolutionary psychology is that biological evolution, as reflected by changes in our genetic structure, lags far behind cultural and societal evolution (Nesse & Williams, 1994; Tooby & Cosmides, 1990). As such, physical and mental disorders can result from a “mismatch” between the environments in which behaviors were developed and the current environment in which they are expressed (Cosmides & Tooby, 1999; Nesse & Williams, 1994). In the case of catatonia, the argument would be that this reaction developed to deal with situations in which our ancestors found themselves on a regular basis, namely, face to face with a predator. As the failure to

3 This is also known as predatory imminence (Fanselow & Lester, 1988).
develop effective coping mechanisms in such situations would lead to likely death, it is easy to see why there would be strong selection pressures favoring those genes coding for effective strategies, such as TI. Further, such genetic adaptations would likely include high tolerance for false positives; the consequences of mistakenly responding with a fear response to an innocuous situation are far outweighed (evolutionarily) by the adaptive consequences of not responding to a genuine danger situation (Nesse & Williams, 1994). Nesse (2001) has referred to this as the “smoke detector principle” (p. 75).

Tooby and Cosmides (1990) considered the emotions generated in such situations as crucially important, arguing that they carry signals or codes for the expression of behavioral programs developed in our ancestral past; “the operation of human psychological mechanisms are orchestrated by emotions that frame present circumstances in terms of the evolutionary past” (p. 420).

There is clear behavioral evidence of such relics in some human responses to fear and danger. Perhaps most obvious in this regard is piloerection: the hair on the nape of our necks standing up when we are frightened or enraged. Although this serves no obvious adaptive purpose in humans, it allows other species, such as canines, to appear larger and more imposing to opponents. Likewise, Marks (1987) argued that stranger anxiety is largely a relic from our developmental history or from the history of species phylogenetically related to us, in which infants were at risk of being killed by strange males hoping to induce ovulation in their mother.

Among anxiety disorders, both phobias and panic disorder have been linked to evolutionary forces. Several researchers have argued that phobias, which tend to cluster around themes of animals (particularly spiders and snakes), social situations, blood or injuries, and open spaces, represent common danger cues in human evolution (Ohman, 1992; Seligman, 1971). Likewise, panic disorder, in one prominent theory, is proposed to result from the “misfiring” of an evolutionary-based suffocation alarm (Klein, 1993).

Most important for this thesis, depression has been hypothesized to relate to animal defense responses in general and TI in particular (Dixon, 1998; Gilbert & Allan, 1998). In this formulation, TI (or in Dixon’s, 1998, term, arrested flight)4 is linked with aspects of depression, such as social withdrawal, reduced eye contact, and psychomotor retardation (Dixon, 1998; Gilbert & Allan, 1998). Dixon (1998) characterized arrested flight–TI as a last resort defense strategy triggered by conditions of inescapable proximal threat and characterized by cryptic immobile postures, a lack of overt surveillance, and cutoffs. This latter behavior is designed to minimize aversive stimulation (i.e., the predator), thereby reducing arousal levels, and is typically manifested by the threatened animal closing its eyes or averting its head. Dixon considered arrested flight–TI to be “the behavioral hallmark of the severely depressed individual” (p. 436). Gilbert and Allan (1998) have argued that this link is mediated by feelings of entrapment and defeat, concepts which they have found, in a sample of students and depressed individuals, to more strongly predict depression than the concept of helplessness; the latter does not convey what they consider central to the phenomenology of depression—a strong motivation for flight that is blocked by internal or external factors.

Thus, several forms of anxiety and depression have been linked to ancestral conditions from which they may have derived. Could catatonia be another, perhaps more direct, link? To answer this question, a brief review of catatonia and TI is required.

An Overview of Catatonia

Catatonic behavior is defined in the Diagnostic and Statistical Manual of Mental Disorders (4th ed., rev.; American Psychiatric Association, 2000) as the following:

Catalepsy is the maintenance of postures, often odd in appearance, for long periods of time; whereas stupor is unresponsiveness, hypoactivity, and reduced or altered arousal—apparent analgesia to painful stimuli may be present in severe cases (Fink & Taylor, 2003). Echolalia and echopraxia involve the copying or mimicking of the examiner’s utterances or movements, respectively. In the DSM–IV, two of the above symptom clusters are required for catatonic schizophrenia or affective disorders with catatonic features, whereas only one is required for catatonic features due to a medical condition.

There has been a recent proliferation of scales and approaches for diagnosing catatonia (Braequung, Krueger, Shugar, Hoeffler, & Boerner, 2000; Bush, Fink, Petrides, Dowling, & Francis, 1996a; Fink & Taylor, 2003; Northoff, Koch, et al., 1999; Peralta & Cuesta, 2001). The closest to the DSM–IV approach would be that of Bush et al. (1996a) and Peralta and Cuesta (2001), who require two or three symptoms, respectively, from among those listed in the DSM–IV or others, including verberigation (repetition of phrases or sentences), mannerisms (odd, apparently purposeful movements), or waxy flexibility (initial resistance followed by submission to limbs being repositioned—likened to the bending of a warm candle). As can be noted, some signs of catatonia—immobility, posturing, mutism, or excitement, for example—can be observed, whereas others—such as waxy flexibility, echolalia, or negativism—can only be demonstrated through interaction with others.

Fink and Taylor (2003), who have long argued for the acceptance of catatonia as a distinct diagnostic entity (see Fink & Taylor, 1991), took a different tack, distinguishing between different forms of catatonia. Their criteria are:

A. Immobility, mutism, or stupor of at least 1 hr duration, associated with at least one of the following: catalepsy, automatic obedience, or posturing, observed or elicited on two or more occasions.

4 Dixon’s (1998) description of arrested flight leaves little doubt that he is describing the same phenomenon as TI. He prefers the term arrested flight to emphasize that this condition arises in situations in which the animal wishes to escape but cannot, as all escape routes are blocked.
B. In the absence of immobility, mutism, or stupor, at least two of the following, which can be observed or elicited on two or more occasions: stereotypy, echophenomena, catatlepsy, automatic obedience, posturing, negativism, or ambitendency (Fink & Taylor, 2003, p. 116).

**Automatic obedience** is defined as an “exaggerated cooperation with the examiner’s request,” whereas **ambitendency** is appearing “motorically stuck in indecisive, hesitant movements” (Bush et al., 1996a, p. 135). Fink and Taylor (2003) allowed for three subtypes of catatonia to be generated from these criteria. **Retarded or akinetic catatonia** (which they refer to as nonmalignant catatonia) is characterized by the A criteria above, **excited catatonia** (or delirious mania in their terminology) consists of the B criteria above plus severe mania or excitement, and **malignant** catatonia consists of the A criteria plus fever and autonomic instability (Fink & Taylor, 2003). In so doing, they sensibly distinguished between the form of catatonia—excited or retarded—and the seriousness or level of risk—malignant (or lethal) or nonmalignant (or benign).

Their approach also emphasizes the centrality of immobility, mutism, and stupor to a diagnosis of catatonia. These symptoms have consistently been found to be the most common in catatonic populations (Bush et al., 1996a; Morrison, 1973; Peralta & Cuesta, 2001; Rosebush et al., 1990). Most factor analytic studies conducted on catatonic symptoms confirm Fink and Taylor’s (2003) approach, finding factors corresponding to stupor, mutism, and immobility on the one hand (unrelated to diagnosis) and other catatonic symptoms (including excitement or correlated with excitement or mania) on the other (Abrams, Taylor, & Coleman Stolurow, 1979; Peralta et al., 2001; Starkstein et al., 1996). Although some had thought catatonia to be quite rare (Mahendra, 1981), four recent well-designed studies found prevalence rates of between 7% and 9% on acute inpatient psychiatric units (Bush et al., 1996a; Peralta & Cuesta, 2001; Rosebush et al., 1990; Ungvari, Leung, Wong, & Lau, 1994).

Catatonia has been the subject of numerous reviews over the last 30 years, most of which were published since 1990, demonstrating renewed interest in the disorder (Clark & Rickards, 1999; Fink, 1997; Fink & Taylor, 1991, 2001; Gelenberg, 1976; L. R. Gjesing, 1974; Goldar & Starkstein, 1995; J. Johnson, 1993; Peralta, Cuesta, Serrano, & Mata, 1997; Pfuhlmann & Stoever, 2001; Ries, 1985; Taylor & Fink, 2003). Indeed, this resurgence of interest appears to be accelerating, as the last few years have seen an entire journal issue—the European Archives of Psychiatry and Clinical Neurosciences (Stoever & Ungvari, 2001)—dedicated to catatonia, the publication of a Behavioral and Brain Sciences review (Northoff, 2002), and the release of two major books related to catatonia (Fink & Taylor, 2003; Mann, Caroff, Keck, & Lazarus, 2003).

This renewal of interest revolves primarily around four areas: (a) the appropriate placement of catatonia in psychiatric nosology, (b) the relationship among catatonia, lethal catatonia, and neuroleptic malignant syndrome (NMS), (c) the treatment of catatonia with BZs and/or electroconvulsive therapy (ECT), and (d) brain functioning and neurochemistry. These issues, along with the phenomenology of catatonia, are discussed in turn.

**Catatonia’s Placement in Psychiatric Nosology**

The history of the concept of catatonia has been well reviewed elsewhere (Berrios, 1981; Gelenberg, 1976; Goldar & Starkstein, 1995; J. Johnson, 1993) and is not discussed in detail here. Briefly, Kahlbaum (1874/1973) believed that catatonia was an organically based degenerative condition, similar to general paresis, which passed through a number of largely affects phases ultimately leading to dementia. He thought the typical pattern to be an initial melancholia, followed by mania (also described as rage, frenzy, and excitement) and then stupor, but noted that the stuporous phase could progress directly from melancholia, or even “out of the blue,” after particularly severe stress. Stressors, according to Kahlbaum, were not uncommon antecedents to catatonia and could include the death of a loved one, conflictual relationships, or financial problems.

Ignoring Kahlbaum’s (1874/1973) association of catatonia with affective conditions, Kraepelin (1899) co-opted it into his dementia praecox construct. Later, Bleuler (1911/1950) included it as a component of schizophrenia. Despite protests that catatonia was linked to affective conditions (Hoch, 1921; Kirby, 1913), it was widely viewed as pathognomic for schizophrenia for most of the 20th century, appearing only as a subtype of schizophrenia for all editions of the American Psychiatric Association’s Diagnostic and Statistical Manual of Mental Disorders until the publication of the DSM–IV in 1994 (American Psychiatric Association, 1994). The change in the DSM–IV, which allowed for the presence of catatonia in affective disorders and medical conditions, followed a series of publications in which catatonia was found more commonly in affective disorders than in schizophrenia (Abrams & Taylor, 1976; Abrams et al., 1979; Gelenberg, 1976; J. Johnson, 1984; Morrison, 1973; Ries, 1985)7 and in a wide range of serious medical conditions, including various neurological conditions (such as encephalitis), toxic conditions, metabolic disorders, and idiosyncratic responses to drugs or medications (Barnes, Saunders, Walls, Saunders, & Kirk, 1986; Carroll, 1992; Gelenberg, 1976; Popkin & Tucker, 1992). Early reviews suggested that the syndrome of catatonia was expressed similarly whether it was associated with various psychiatric disorders or with medical conditions (Fein & McGrath, 1990; Gelenberg, 1976). A recent exhaustive review by Carroll et al. (2000) confirmed this perception, concluding that

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5 Of note, in an approach dating back to Kraepelin (1899), Bush et al. (1996a) recommended that automatic obedience be tested by asking the person to stick out their tongue so the examiner can stick a pin in it. Ambitendency is tested by the examiner extending his or her hand and firmly stating, “Do not shake my hand!”

6 However, there is also evidence that catatonia as a whole, including retarded and excited components, can be thought of as a syndrome. Abrams and Taylor (1976) found neither the subtype of catatonia (excited vs. retarded) nor the number of symptoms predictive of either associated diagnosis or treatment response, and Oulis et al. (1997) found evidence for one factor underlying both excited and retarded forms of catatonia.

7 Whereas early studies emphasized a link between catatonia and mania (Abrams & Taylor, 1976; Abrams et al., 1979), more recent studies have found that catatonia in mania is overwhelmingly associated with mixed manic states (admixtures of mania and depression or rapid fluctuations in mood; Braeunig, Krueger, & Shugur, 1999; Krueger & Braeunig, 2000). This suggests that it may be the depressive component of both manic and depressive disorders that is linked with catatonia.
psychiatric and medical catatonias are essentially indistinguishable from each other.

**NMS and Lethal–Malignant Catatonia**

NMS is a potentially lethal condition, characterized by severe akinesia, muscular rigidity, autonomic dysfunction, and fever, occurring after treatment with neuroleptic (antipsychotic) medications. A mortality rate of 19% has been reported (76% in cases reported prior to 1970), most strongly associated with cardiovascular collapse or arrhythmias (Caroff, 1980) or renal failure (Shalev, Hermesh, & Munitz, 1989). It was originally felt to be an idiosyncratic toxic reaction to medications, sharing some symptoms with, but distinct from, catatonia (Caroff, 1980). However, in 1986, Mann et al. conducted an exhaustive historical review of so-called lethal catatonia, drawing similarities to NMS.

Lethal catatonia had been described since the early 1830s as a condition with no clear medical etiology, characterized typically by extreme hyperactivity followed by stuporous exhaustion (or occasionally only stupor without hyperactivity), with fever and autonomic instability (Ljubisavljevic & Schneider, 2002; Mann et al., 2003). It could develop in a wide range of organic and functional conditions, including various psychiatric disorders, cerebrovascular disorders, tumors, head trauma, infections, metabolic disorders, toxic disorders, and seizure disorders (Mann et al., 2003).

The mortality rate has improved over the years, from 75%–100% in the historical (pre-1960) series to 62% in cases reported between 1960 and 1986 (Mann et al., 1986). Most dramatically, the rate has fallen to 9% for cases reported since 1986. This substantial decrease in mortality, attributed to timely and appropriate interventions (Mann et al., 2003), has led some to propose the term lethal be replaced with the term malignant (Fink & Taylor, 2003; Mann et al., 2003).

Causes of death in early cases, typically occurring during a stuporous phase with extreme hyperthermia, have been attributed to "cardiovascular collapse" (Mann et al., 1986), "vagal hypotonia and cardiac arrest" (Billig & Freeman, 1944; Shulack, 1944, cited in Mann et al., 1986), or "adrenocortical insufficiency" due to mental and physical stress (Lingjaerde, 1963, cited in Mann et al., 1986). Of the more recent cases of death, "negative" results have been found in almost 80% of the autopsies, leading Mann et al. (2003) to characterize them as psychogenic in nature.

The relationship between NMS and malignant catatonia has been the basis for some controversy. Although Mann et al. (1986, 2003) have argued on the basis of similarities in presentation, physiological abnormalities—such as elevated creatine phosphokinase and reduced serum iron (Philbrick & Rummans, 1994; White, 1992)—and mortality, that NMS is simply an iatrogenic form of malignant catatonia, others have disagreed (Castillo, Rubin, & Holsober-Trachslher, 1989; Fleischhacker, Unterweger, Kane, & Hinterhuber, 1990). However, the "variant of catatonia" position has been bolstered by recent research, such as White and Robin's (2000) finding that 17 consecutive NMS patients had histories of catatonic episodes unassociated with medication use; the majority of those in the field now appear to accept this position (Ahuja & Nehru, 1990; Fink, 1996; Fink & Taylor, 2003; Fricchione, 1985; Fricchione, Mann, & Caroff, 2000).

**Phenomenology**

There are three published studies that have commented on the subjective state of catatonia at length—Rosebush and Stewart (1989), Rosebush et al. (1990), and Northoff, Krill, et al. (1998). Rosebush and Stewart (1989) identified 20 individuals who experienced 24 episodes of NMS over a period of 6 years. They reported observing a striking, frightened facial expression in all cases. Eleven of the 20 patients were later able to describe feeling at the time a desire to speak but an inability to do so and a sense of impending doom along with overwhelming anxiety. Rosebush and Stewart did not report whether the remaining patients denied anxiety or could not recall their mental state.

In a subsequent study, Rosebush et al. (1990) located 12 catatonic subjects out of 140 consecutive admissions (8.6%). Ten of the 12 patients (83%) responded dramatically to lorazepam, with complete resolution of the catatonic symptoms after one dose. Rosebush et al. reported that all 10 responders described feeling intense anxiety during the course of the catatonic episode. Of the 2 nonresponders, 1 did not endorse anxiety and the other could not recall the episode. In some cases, the anxiety appeared related to the extrapyramidal side effects of the antipsychotic medications they had received. Thus, 3 patients misinterpreted such extrapyramidal side effects symptoms as being "controlled" or "turned into a robot." Summarizing their findings in a recent editorial, Rosebush and Mazurek (1999) concluded that the vast majority of persons experiencing catatonic stupor report feeling "frozen" or "petrified" and experience "extreme fear" during the episode (pp. 396–397).

Northoff and colleagues’ (Northoff, Krill, et al., 1998; Northoff et al., 1995, 2000) researches have led them to similar conclusions. In a study of the subjective state of persons experiencing catatonic stupor, Northoff, Krill, et al. (1998) found, on the basis of assessments prior to treatment (including the Hamilton Anxiety Scale; Hamilton, 1959), that BZ responders (defined by the complete resolution of catatonic symptoms within 24 hr—59% of the sample) reported significantly more anxiety, intense feelings, and "blockade of movements by emotions" than did nonresponders (Northoff, Krill, et al., 1998). In a more recent study, also using the Hamilton Anxiety Scale, Northoff et al. (2000) found strong and significant correlations between anxiety and catatonic symptoms ($r = .60–.68$).

As these studies have all retrospectively assessed emotional experiences during catatonic episodes, they have been unable to determine whether the anxiety preceded the catatonic episode or developed subsequent to them, perhaps in response to an inability to move.8 Although Rosebush and Mazurek (1999) contend that the fear precedes the immobility in most of their cases, no relevant studies have been conducted; this is perhaps understandable, as it is very difficult to predict when someone will enter a catatonic state. There have, however, been case reports of individuals developing stupor after reports of anxiety (Chandler, 1991; Wetzel, Heuser, & Benkert, 1987). Particularly illustrative is that of Wetzel et al. (1987), who described the complete alleviation of symptoms of catatonia with one dose of a BZ, followed by their reinstatement.

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8 This possibility, however, appears unlikely in the light of research on catatonic stupor, which has found persons strikingly unaware of their unusual body positions or movements (Northoff, Krill, et al., 1998).
after administration of a BZ antagonist (Ro 15-1788; PerkinElmer, Boston). They reported,

Almost immediately after injection, the patient complained of subsequently occurring dizziness, nausea, increasing anxiety, and overwhelming fears concerning accidents of family members. These fears condensed to certainty. Two minutes later, the patient was in a state identical to that before application of lorazepam, being mute and stuporous. (Wetz et al., 1987, pp. 240–241)

Thus, although limited research has been conducted to date, severe anxiety has been reported in psychiatric (Chandler, 1991; Northoff, Krill, et al., 1998; Rosebush et al., 1990; Wetz et al., 1987), iatrogenic (Rosebush & Stewart, 1989), and medical (Orland & Daghestani, 1987) catatonia. All studies that have asked persons subsequent to episodes of catatonic stupor what they experienced at the time found the majority to report intense anxiety, which abated with BZ treatment.

**Treatment**

The resurgence of interest in catatonia has perhaps been most stimulated by the discovery that BZs such as lorazepam alleviate catatonic symptoms in 70%–90% of acute cases (Ungvari et al., 2001). This was initially reported in the treatment of NMS or neuroleptic-induced catatonia (Fricchione, Cassem, Hooberman, & Hobson, 1983; Lew & Tollefson, 1983) but was quickly discovered to be true for both psychiatric and medically related catatonia as well (Bernstein & Levin, 1993; Delisie, 1991; Greenfield, Conrad, Kincare, & Bowers, 1987; Harris & Menza, 1989; Menza & Harris, 1989; Salam, Pillai, & Beresford, 1987). Several dozen case studies have now confirmed the efficacy of BZ in not only retarded but also excited forms of catatonia (e.g., Cottencin, Thomas, Vaiva, Rascel, & Goudemand, 1999; Harris & Menza, 1989; Lee, Schwartz, & Hallmayer, 2000; Ungvari, Leung, & Lee, 1994). In one controlled study of BZ in catatonia, complete resolution of catatonic signs to the first dose occurred in 83% of the sample (Rosebush et al., 1990); in another study, one dose of lorazepam resulted in 22% demonstrating a “dramatic” resolution of symptoms and the remaining 78% experiencing “clinically significant relief” (Ungvari, Leung, Wong, & Lau, 1994). Of note, the improvement appears to relate only to catatonic signs; concomitant psychotic symptoms remain largely unchanged (Ungvari et al., 2001).

Most authors have concluded that BZs are beneficial in catatonia by virtue of their antianxiety properties (Northoff, Steinke, et al., 1999; Rosebush & Mazurek, 1999; Wetz et al., 1988). Consistent with this, those who respond to BZ consistently report more anxiety pretreatment than those who do not (Northoff, Krill, et al., 1998; Northoff et al., 1995; Rosebush et al., 1990), and BZ-mediated improvement in catatonic symptoms (and amantadine as well; Northoff, Eckert, & Fritze, 1997) coincides with substantial decreases in reported anxiety (Northoff, Krill, et al., 1998; Northoff et al., 1995; Wetz et al., 1987). However, it is not possible to rule out, on a pharmacological basis, that BZ’s efficacy in catatonia may be anticonvulsant—as opposed to antianxiety—in nature, as some have contended (Fink & Taylor, 2003; Kritzinger & Jordaan, 2001; Raitiere, 1986). Most BZs have potent anticonvulsant effects, and ECT, which is effective in catatonia (Bush et al., 1996b; Escobar et al., 2000; Rohland, Carroll, & Jacoby, 1993), raises the threshold for seizures; antiseizure medications, such as carbamazepine and amobarbital, also appear to be effective in catatonia (Kritzinger & Jordaan, 2001; McCall, Shelp, & McDonald, 1992; Rankel & Rankel, 1988). Conversely, zolpidem, which has little anticonvulsant activity (Salva & Costa, 1995), also appears to improve catatonia (Thomas, Rascle, Maastricht, Maron, & Vaiva, 1997; Zaw & Bates, 1997).

Moreover, although BZ and ECT both appear to be effective in catatonia, there are several reasons to believe that their mechanisms of action may be different: (a) Six to eight sessions of ECT are typically required to produce improvement in catatonic symptoms (Escobar et al., 2000), whereas BZ’s effects may be seen within minutes (Rosebush et al., 1990); (b) ECT may improve depression associated with catatonia (Bush et al., 1996b), whereas BZ appears to affect only the catatonic symptoms (Ungvari et al., 2001); (c) ECT appears to be effective in reducing catatonic symptoms in individuals who do not respond to BZ (Bush et al., 1996b); and (d) a combination of BZ and ECT, either sequentially or concurrently, appears more effective than either alone (Petrides, Divadeneam, Bush, & Francis, 1997).

Further, it appears likely that ECT’s potency in catatonia is due more to its antidepressant than anticonvulsant properties. Catatonia is associated with severe depression (Krueger & Braueunig, 2000; Starkstein et al., 1996), the primary psychiatric indication for ECT (Rasmussen, 2003; UK ECT Review Group, 2003), and severe psychomotor retardation in depression is particularly predictive of a positive response to ECT (Rasmussen, 2003). In addition, ECT is more effective in alleviating catatonic symptoms in affective disorders than in schizophrenia (Abrams & Taylor, 1977; Escobar et al., 2000; Rohland et al., 1993), and the anticonvulsant carbamazepine, effective in catatonia (Kritzinger & Jordaan, 2001; Rankel & Rankel, 1988), is used as a treatment for mood disorders as well (Birkhimer, Curtis, & Jann, 1985; Israel & Beaudry, 1988). Thus, a feasible alternative to the suggestion that both BZ and ECT act as anticonvulsants is that catatonia contains components of both anxiety and depression in varying degrees (or that there are catatonia subgroups with different proportions of anxiety and depression) and that BZ acts primarily on anxiety symptoms whereas ECT acts primarily on depressive symptoms.

**Brain Functioning and Neurochemistry**

It has been argued that both BZ and ECT as well as most other substances effective in catatonia (e.g., valproic acid, amobarbital)

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9 This argument is bolstered by the fact that anxiety and depression often appear together. One quarter to over 60% of individuals with bipolar disorder also meet criteria for an anxiety disorder (Henry et al., 2003; McElroy et al., 2001; Tamam & Ozpoyraz, 2002), and anxiety is present in both manic and depressed states (Young, Cooke, Robb, Levitt, & Joffe, 1993). Half of those diagnosed with major depression also have an anxiety disorder (Sanderson, Beck, & Beck, 1990; Zimmerman, Cheilinski, & McDermut, 2002), and one third of those with an anxiety disorder have comorbid dysthymia or major depression (Sanderson, DiNardo, Ruppee, & Barlow, 1990). Further, as anxiety increases with the severity of depression (Krishnan, 2003) and catatonia is associated with more severe depressive symptoms in both manic (Krueger & Braueunig, 2000) and depressive (Starkstein et al., 1996) disorders, it is particularly likely that anxiety is common in the depressive states that accompany catatonia.
improve catatonia via their action on the gamma-aminobutyric acid (GABA) inhibitory system, and specifically through GABA\textsubscript{\alpha} receptors (Carroll, 1999; McCall et al., 1992; Northoff, Steinke, et al., 1999). Indeed, Carroll (1999) has argued that GABA\textsubscript{\alpha} receptors play a crucial role in catatonia, noting that GABA\textsubscript{\alpha} agonists other than BZ, such as zolpidem, also appear to improve catatonia. Fink and Taylor (2003) agreed, noting that “too much GABA\textsubscript{\alpha} or too little GABA\textsubscript{\alpha} activity contributes to the expression of catatonia” (p. 187). However, other substances effective in catatonia, such as amantadine, do not directly interact with GABA but rather decrease glutamate and increase dopamine transmission (Northoff, Eckert, & Fritz, 1997). Further, medications that produce NMS, a variant of catatonia, block dopamine, and almost all drugs that increase serotonin produce a toxic reaction some have linked to catatonia (Fink & Taylor, 2003). As a consequence, Mann et al. (2003), while arguing for a primary role for dopamine in malignant catatonia, acknowledged that other neurotransmitters, including GABA, are directly or indirectly involved, and Fink and Taylor (2003) caution that it would be oversimplified to consider any one neurotransmitter to be the basis for catatonia. Northoff (2002) came to a similar conclusion, noting that changes in other neurotransmitter systems, such as dopamine or serotonin, can develop secondarily to changes in the GABA system.

Until the last few years, the only studies to systematically assess physiological functioning or biochemistry in catatonia involved individuals suffering from periodic catatonia—successive episodes of stupor, excitement, or both. In a remarkable series of studies conducted by the Norwegian psychiatrist R. Gjessing in the 1920s and 1930s, summarized in a 1938 article (R. Gjessing, 1938), and subsequent studies conducted by his son, Leiv Gjessing, summarized in a 1974 article (L. R. Gjessing, 1974), individuals suffering from periodic catatonia were assessed on a daily basis, over periods of months and years, on numerous physiological indices—such as pulse, blood pressure, oxygen consumption, temperature, leucocytes, nitrogen balance, and levels of various metabolites—before, during, and after episodes of catatonic excitement and stupor. Both researchers found reliable increases in heart rate, temperature, blood pressure, and oxygen consumption, in both excitement and stupor phases (L. R. Gjessing, 1974; R. Gjessing, 1938), and L. R. Gjessing (1974) found increases in a number of metabolites, particularly adrenergic–noradrenergic. L. R. Gjessing (1974) characterized catatonic stupor as having a more rapid onset and greater autonomic instability (demonstrated by frequently changing pupil widths) than catatonic excitement. These were not insignificant changes, as R. Gjessing (1938) reported, for example, sustained increases in pulse rate from 60–70 to 110–120 beats per minute. Strikingly, these changes were noted even when stupor set in during sleep and were sometimes maintained for several days despite an almost complete lack of movement.

Both researchers concluded that overactivity of the sympathetic nervous system (SNS) was central to the onset of periodic catatonic episodes; R. Gjessing (1938) characterized this as an “adrenal-sympathetic impulse” similar to Cannon’s emergency reaction, whereas L. R. Gjessing (1974) noted similarities to reactions to adrenaline injections. Of note, these changes are consistently described as coinciding with the onset of the stupor (and excitement) and in one publication are described as occurring during the transition into the catatonic phase (Takahashi & Gjes sing, 1972).

Such a formulation is consistent with that recently put forward by Gurrera (1999) on NMS. Gurrera argued that a “hyperactive and unregulated” SNS, released from inhibition by the frontal cortex (via the hypothalamus), explains most, if not all, of the signs and symptoms of NMS.

A primary dysfunction of the frontal lobes has been more strongly emphasized by others. Taylor (1990) noted that the symptoms of catatonia were often described in the neurological literature as evidence for frontal lobe disease or dysfunction. He proposed that catatonia might be due to a dopamine imbalance in a “frontal lobe–basal ganglia–brainstem system” (Taylor, 1990, p. 65). Northoff and colleagues (Northoff, Braus, et al., 1999; Northoff et al., 2001; Northoff et al., in press; Northoff, Steinke et al., 1998, 1999; Northoff et al., 2000), who have conducted an important series of regional cerebral blood flow, single photon emission computed tomography, and functional magnetic resonance imaging studies on brain functioning in catatonia, took a somewhat different tack. In a recently published Behavioral and Brain Sciences review, Northoff (2002) argued that reduced right orbitofrontal cortex functioning, mediated by the GABAergic system, is a hallmark of catatonia and leads to an inability to inhibit subcortical structures such as the basal ganglia. Of note, this reduced right orbitofrontal activity (left-hemisphere functioning appears largely unaffected) correlates significantly with measures of anxiety in these patients and is reversed by BZ (Northoff, 2002). Further, Northoff discovered that in currently nonsymptomatic catatonic patients, connectivity between orbitofrontal cortex and premotor–motor cortex was functional at baseline but became impaired during negative emotional stimulation (i.e., distressing photographs; Northoff et al., in press, cited in Northoff, 2002). He speculated that this occurs via connections between the orbitofrontal cortex, limbic system, and prefrontal cortex—which modulates the motor cortex and premotor area (Northoff, 2002). Finally, Northoff acknowledged that the deficits in right orbitofrontal functioning found in catatonia may derive from activity in the amygdala, with which it is strongly reciprocally connected.

**Catatonia and TI**

**Behavioral Characteristics**

Catatonia shares a number of behavioral characteristics with TI, first noted by Gallup and Maser (1977). Specifically, both catatonic stupor and TI are characterized by (a) immobility; (b) posturing; (c) stupor (lack of response); (d) waxy flexibility; (e) reduced or absent vocalization; (f) fixed, unfocused gaze or stare; (g) analgesia; and (h) evidence of alertness, despite unresponsive ness (Bush et al., 1996a; Fink & Taylor, 2003; Gallup, 1974; Gallup, Boren, Suarez, Wallnau, & Gagliardi, 1980; L. R. Gjes sing, Harding, Jenner, & Johannessen, 1967; Kahlbaum, 1874/ 1973; Klemm, 1971, 2001; Ratner, 1958, 1967; Rosebush & Maze rek, 1999; Taylor, 1990). In addition, TI demonstrates an abrupt onset, almost invariably subsequent to struggling or fighting behavior, and often terminates with spirited struggle or escape (Hofer, 1970; Sargeant & Eberhardt, 1975); likewise, catatonic stupor often begins abruptly and is not infrequently preceded or followed by hyperactivity or excitement (American Psychiatric Association, 2000; Fink & Taylor, 2003; L. R. Gjessing, 1974; Morrison, 1973). This hyperactivity often includes assaultive beh-
behavior, at times directly out of a stuporous state (Bush et al., 1996a; Fink & Taylor, 2003; Kahlbaum, 1874/1973; Morrison, 1973). In contrast to the motor symptoms of catatonia, the behavioral—communicative symptoms, such as echophenomena and ambidexterity, do not show any clear parallels to TI—obviously, those involving spoken language could not.

**Mortality**

There is evidence that TI can, in and of itself, be fatal (Gallup, 1977; Hofer, 1970). Gallup (1977) discovered, during the course of his research, that chickens occasionally died while immobilized; in all instances, the chickens had received manipulations designed to increase fear. Likewise, Hofer (1970), working with wild rodents, discovered that over 25% died without apparent cause during the first week of captivity. The deaths appeared related to pronounced arrhythmias of vagal origin that developed during prolonged states of immobility and not to captivity per se.

The deaths arising from NMS and malignant catatonia appear not dissimilar to those described above, attributed to factors such as renal failure (Shalet et al., 1989), cardiovascular collapse, or arrhythmias (Carroll, 1999; Mann et al., 1986). Of note, although such states are often characterized by hyperactivity, the deaths typically occur during a final stuporous phase (Mann et al., 1986, 2003).

**Physiology and Neurochemistry**

Significant changes in heart rate and respiration are found in TI, with heart rate dropping or initially rising and then dropping below baseline (Carl, 1977; Hofer, 1970; Nash, Gallup, & Czech, 1976; Ratner, 1967)—though in some species, such as pigeons, it remains high (Ratner, 1967)—and respiration typically increasing (Hofer, 1970; Nash et al., 1976). Core temperature also usually drops (Eddy & Gallup, 1990; Whishaw, Schallert, & Kolb, 1979), and pupils are dilated (Gallup & Maser, 1977). Thus, one sees a combination of sympathetic and parasympathetic activation in TI.

Catatonia, in both excited and stupor phases, appears to be characterized by autonomic instability, with sympathetic activation predominating (L. R. Gjessing, 1974; R. Gjessing, 1938). There is also evidence, however, of some parasympathetic activation in stupor (L. R. Gjessing, 1974), and when catatonia proceeds to death, it is largely parasympathetically mediated (Mann et al., 1986; Shalet et al., 1989).

One study has found morphine to induce TI in rats, in a form that appears similar to behaviorally induced or naturally occurring TI (de Ryck & Teitelbaum, 1984). (In contrast, neuroleptic-induced catalepsy appears to have some significant differences from TI and may be more akin to attentive immobility.) This has led some researchers to posit a role for endogenous opiates in TI (Leite-Panissi, Rodrigues, Bretegani, & Menescal-de-Oliveira, 2001). Specifically, de Ryck & Teitelbaum (1984) found rats administered morphine to demonstrate rigid immobility with insensitivity to pain, alternating with explosive bursts of uncontrolled locomotion. Morphine injections into the amygdala or the midbrain periaqueductal gray (PAG) facilitate this response (Klemm, 1989). Although there is not direct evidence for endogenous opiate activity in catatonia, Taylor (1990) has speculated that the insensitivity to pain seen in catatonic stupor may be opioid mediated.

The GABA system appears to play a prominent role in TI, as the GABAergic agonist muscimol decreases TI, whereas the GABAergic antagonist bicuculline increases it (Monassi, Leite-Panissi, & Menescal-de-Oliveira, 1999). However, there is little evidence at this point that BZs (which are well-known to exert their anxiolytic effects via the GABA system) decrease TI; although they reliably decrease attentive immobility (Kaln, 1993), two studies found BZ to actually increase TI in chickens (Rager, Gallup, & Beckstead, 1986) and guinea pigs (Olsen, Hogg, & Lapiz, 2002). The serotonergic system, closely entwined with the GABA system (Graeff, 1993; Kahn, van Pragg, Wetzelr, Asnis, & Barr, 1988), has also been implicated in TI (Gallup & Rager, 1996; Olsen et al., 2002). Thus, at this point, it can only be stated that TI appears to result from a complex, not yet understood, interplay between GABAergic, opioid, and serotonergic systems. As noted above, many researchers see catatonic disturbances as being GABA mediated (Carroll, 1999; Fink & Taylor, 2003; McCall et al., 1992; Nordhoff, Steinke, et al., 1999), though some accord a prominent role to dopamine as well (Fink & Taylor, 2003; Mann et al., 2003; Taylor, 1990).

**Brain Functioning**

The structures most involved in the functioning (or inhibition) of TI appear to be the frontal lobes, limbic system, and brainstem. Svorad first noted in 1957 that TI was most common in species with limited development of cerebral cortex and appeared in higher species only under pathological conditions. Klemm (1971) agreed with Svorad’s position, further noting that decorticated rats expressed immobility after surgery but not before and that immobility was common in the young of some species, before the cortex was fully developed, but rare at maturity. He concluded that the neocortex inhibited TI. Subsequently, Klemm (1989) proposed that this disinhibition of cortical areas was facilitated by limbic activity, most likely the amygdala, which controlled the opioid-based expression of TI through the GABA system’s influence on lower brain structures.

Other researchers have largely endorsed Klemm’s (1989) proposal, noting further that the amygdala projects to the PAC in the brainstem and could thus potentiate the activity of the latter (Fanselow, 1991; Ramos, Leite-Panissi, Monassi, & Menescal-de-Oliveira, 1999). The PAC is believed by many researchers to be the brain structure most directly responsible for the expression of TI as well as other evolutionary-based defense reactions, as direct stimulation of certain portions of the PAC (as well as microinjections of morphine, as noted above) produce behavior largely indistinguishable from TI (Fanselow, 1991; Fendt & Fanselow, 1999; Gray & McNaughton, 2000; Leite-Panissi, Coimbra, & Menescal-de-Oliveira, 2003; Monassi et al., 1999).

Catatonia has also been hypothesized to result from a dysfunction of the frontal lobes (Taylor, 1990), in particular the right orbitofrontal lobe (Nordhoff, 2002). It is argued that such a dysfunction releases cortical and subcortical motor control regions (Nordhoff, 2002; Taylor, 1990) and that brainstem areas may also be involved (Taylor, 1990). In addition, Nordhoff (2002) and to a lesser extent Fink and Taylor (2003) appear to recognize that such disturbances may be partially or primarily driven by limbic inter-
ference in general (Fink & Taylor, 2003) or by the amygdala in particular (Northoff, 2002). Finally, although Taylor (1990) implicated the brainstem in general, there is no evidence to date implicating PAG activity in catatonia.

Summary

A summary of the main findings resulting from this comparison of catatonia with TI can be seen in Table 1. Catatonia shares with TI a significant number of behavioral characteristics, most of which appear to be GABA mediated. They are also both occasionally punctuated by bursts of explosive activity and in some cases lead to death, which appears largely vagal mediated. In addition, both have been hypothesized to involve frontal lobe dysfunction or disinhibition, releasing motor control regions, possibly under the influence of the amygdala. Thus, there are substantial similarities between TI and catatonia, strongly suggesting that the latter is derived from the former.

However, there are some important differences as well. There is evidence that dopamine may play a role in catatonia but not in TI, whereas the role of serotonin is more clearly demonstrated in the latter than in the former. Further, catatonia responds to BZ, whereas TI does not (though only two studies on BZ in TI have been located). If TI is to be thought of as an appropriate model for catatonia, how can this important difference be reconciled? Clues come from focusing on catatonia as an inappropriate expression of TI.

The Nature of Catatonia

Animal Defense Strategies and Varieties of Catatonia

As stated before, it is not catatonia, properly speaking, that is associated with TI but only one form of catatonia—akinetic cata-

tonia or catatonic stupor. However, other components of catatonia may be related to other animal defense strategies, namely, fight or flight. The three components of catatonia described above—catatonic stupor, catatonic excitement, and the behavioral-communicative features—as well as one form of catatonia—malignant catatonia—are related to animal defense strategies and autonomic functioning (drawing heavily on Porges’s, 1995, 1997, 2001, polyvagal theory) in Table 2 and in the sections below.

Catatonic stupor as an inappropriate expression of TI. Catatonic stupor responds robustly to BZ, whereas TI does not. This apparent discrepancy could be explained if TI is conceptualized as a fear response and catatonic stupor is conceptualized as an anxiety response. Although fear and anxiety have been used somewhat interchangeably throughout this article, this distinction now becomes critical to an understanding of catatonia. Summarizing a wealth of data in The Neuropsychology of Anxiety, Gray and McNaughton (2000) argued that BZs are uniquely effective in reducing anxiety but are ineffective against fear states; in fact, BZ response forms a core component of their definition of anxiety. So, they argued that generalized anxiety disorder, which does respond to BZ, is appropriately considered an anxiety response, whereas phobias, which do not respond to BZ (and have a clearly identifiable object), are best considered a fear response (Gray & McNaughton, 2000). Blanchard and Blanchard (1990) related fear to a present threat and anxiety to a potential threat; it is important to note that this distinction partly rests on whether the fear-producing source can be localized. Although the source of danger is generally not an issue in naturally occurring or experimentally induced TI, it may be an issue in catatonia, in which the danger, despite being experienced as present and overwhelming, may be impossible to localize. That is, in catatonia, the fear response, evolutionarily appropriate in situations of confrontation with a predator, is triggered in situations in which no predator can be found. The sensation of imminent doom in the absence of any localizable source of danger transforms the fear response into an anxiety response. Thus, in this hypothesis, catatonic stupor would be seen as an anxiety response and predicted to respond to BZ, whereas TI would be seen as a fear response and unresponsive to BZ—which is what has been found. Nonetheless, this should be considered speculative, as only two studies have explored the impact of BZ on TI, and in both studies, the danger—in the form of the experimenter who induced TI—was localizable (Olsen et al., 2002; Rager et al., 1986); it remains to be seen whether TI, induced electrically or pharmacologically, which would present no object from which to flee, would be responsive to BZ.

Catatonic excitement as an inappropriate expression of fight–flight. Aspects of catatonic excitement can also be understood as an artifact of an inability to localize the source of danger. Indeed, the DSM–IV definition of catatonic excitement—“apparently purposeless” (American Psychiatric Association, 2000, p. 764)—is entirely consistent with this. Further, assaultiveness, common in catatonic excitement (American Psychiatric Association, 2000; Fink & Taylor, 2003; Morrison, 1973), is described as undirected or unfocussed (American Psychiatric Association, 2000). Combativeness, a component of a prominent catatonia rating scale, is described as being expressed “usually in an undirected manner, with no, or only a facile explanation afterwards” (Bush et al., 1996a, p. 135). This behavior is understandable if, as I propose, the catatonic individual

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Table 1

Comparison of Catatonia With Tonic Immobility

<table>
<thead>
<tr>
<th>Domains</th>
<th>Catatonia</th>
<th>Tonic immobility</th>
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<tbody>
<tr>
<td>Behavioral characteristics</td>
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<tr>
<td>Immobility</td>
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<td>x</td>
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<tr>
<td>Mutism</td>
<td>x</td>
<td>x</td>
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<td>Stupor</td>
<td>x</td>
<td>x</td>
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<tr>
<td>Catelepsy</td>
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<td>x</td>
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<td>Excitement</td>
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<tr>
<td>Analgesia</td>
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<tr>
<td>Echolalia–echopraxia</td>
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<td>–</td>
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<tr>
<td>Automatic obedience</td>
<td>x</td>
<td>–</td>
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<tr>
<td>Stereotypies</td>
<td>x</td>
<td>–</td>
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<tr>
<td>Physiology and neurochemistry</td>
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<tr>
<td>Sympathetic activation</td>
<td>x</td>
<td>x</td>
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<tr>
<td>Parasympathetic activation</td>
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<td>x</td>
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<tr>
<td>GABA involved</td>
<td>x</td>
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<tr>
<td>Opioid involved</td>
<td>?</td>
<td>x</td>
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<tr>
<td>Dopamine involved</td>
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<td>–</td>
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<tr>
<td>Serotonin involved</td>
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<td>x</td>
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<tr>
<td>BZ response</td>
<td>x</td>
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<tr>
<td>Brain functioning</td>
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<tr>
<td>Frontal dysfunction</td>
<td>x</td>
<td>x</td>
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<tr>
<td>Amygdala involvement</td>
<td>?</td>
<td>x</td>
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<tr>
<td>Periaqueductal gray–brainstem involvement</td>
<td>?</td>
<td>x</td>
</tr>
</tbody>
</table>

Note. x = present; – = not present; ? = limited or conflicting evidence; GABA = gamma-aminobutyric acid; BZ = benzodiazepine.
experiences a palpable sense of danger in a situation in which there is no clearly recognizable object to attack or from which to flee—a consequence of this behavioral pattern being elicited in an evolutionary inappropriate threat situation. Under such circumstances, unsuspecting passersby may be attacked for no apparent reason (Fink & Taylor, 2003). Consistent with this hypothesis, uncharacteristic undirected escape behavior can be produced in rats (i.e., running off a 1 m high table) following morphine injections into the PAG (de Ryck & Teitelbaum, 1984), triggering a flight response in a situation lacking a suitable object from which to flee.

**Associated catatonia features and communication deficits.** Engagement of the SNS in catatonic excitement or stupor may also affect the type of communicative behavior available to an individual. According to Porges’s polyvagal theory of emotion and defensive behavior (so-called because he believes mammals have evolved two dissociable vagal systems; Porges, 1995, 1997, 2001), sympathetic activation results in an inhibition of the ventral vagal complex (VVC), a parasympathetic component of the autonomic nervous system unique to mammals that allows for rapid and subtle cardiac modulation in situations perceived as safe and innervates the muscles of the face and vocal system. Thus, inhibition of the VVC results in a decreased capacity to use sophisticated forms of communication with words and gestures—the first line of defense in situations of conflict. The loss of such capacity would be consistent with the mutism frequently seen in catatonia as well as symptoms such as negativism, automatic obedience, or echolalia and echopraxia. If one’s capacity to communicate is severely curtailed, imitating an examiner (echophenomena) or routinely obeying (automatic obedience) or denying (negativism) requests may be the level at which one can interact. In addition, several of these behaviors (waxy flexibility, automatic obedience) appear submissive, which would also be consistent with a defense reaction.

These symptoms (which would meet DSM–IV criteria for catatonic behavior but not, in the absence of motor excitement, Fink and Taylor’s, 2003, criteria for catatonia) are not typically viewed as communicative but rather are seen as evidence of stimulus-bound behavior arising from frontal lobe dysfunction (Mann et al., 2003; Taylor & Fink, 2003). However, most can be elicited only through interaction with another person making requests of the individual or manipulating his or her limbs (Taylor & Fink, 2003). As these individuals are often mute or demonstrate little productive speech, it is not unreasonable to suggest that these symptoms, arising in the context of a social interaction, be broadly considered communicative in nature.

In and of themselves, these symptoms, which do not have analogues in animal defense reactions, are likely less related to anxiety than states of stupor or excitability. Chronic catatonic patients, who primarily exhibit these symptoms, do not demonstrate autonomic instability or respond to BZ (Bush, Petrides, & Francis, 1997; Ungvari, Chiu, Chow, Lau, & Tang, 1999).

In addition, though admittedly speculative, it is possible that some of the postures seen in catatonia—such as “psychological pillow”—may contain fragments of a fear response. For example, one is better able to perceive potential dangers while lying in bed than if one were fully supine. Further, the twisted odd head positions frequently seen in catatonic stupor (as well as the fixed eye gaze) could conceivably derive from the cutoff positions of TI (Dixon, 1998), reducing visual input from the predator and thereby decreasing distress and arousal.

**Malignant catatonia: Scared to death?** In almost 80% of the autopsied deaths in cases of malignant catatonia since 1960, no medical cause of death could be found (Mann et al., 2003). Mann et al. (2003) have characterized cases such as these as psychogenic, or psychologically caused, which likely indicates that death is due to a vagal surge (Porges, 1997, 2001). As the deaths in Mann et al.’s (1986) older series also appear to have been overwhelmingly vagal in nature—as were the animal deaths during fear-inducing procedures in TI studies (Gallup, 1977; Hofer, 1970)—the possibility that these individuals could have been “scared to death” cannot be discounted.

The typical pattern seen in malignant catatonia (and in NMS) of an extended period of frenetic activity followed by stupor and then death is entirely consistent with Porges’s (1995, 1997, 2001) model of a progression from sympathetic activation to vagal (dorsal vagal complex [DVC])-mediated immobility and death in situations of extreme threat. The DVC, evolutionarily designed for reptiles for whom bradycardia and apnea are adaptive during situations of extreme threat, is primarily used in mammals for digestive purposes but also becomes engaged when

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10 Consistent with this, patients in Rosebush and Stewart’s (1989) study who exhibited frightened faces and reported overwhelming anxiety spoke of wishing to speak but feeling unable to do so.
metabolic resources are scarce or when immobilization is required. However, in mammals, DVC engagement cannot be tolerated for long and as such is used only as a last resort, after engagement of the sympathetic system (i.e., flight–fight) fails to resolve the conflict (Porges, 1997). It is proposed that this is precisely what occurs in malignant catatonia, in which extended sympathetic activity fails to resolve the “problem”—however that might be perceived—exposing the individual to a relatively undiluted DVC response, which, as Porges (1997) noted, is associated with feelings of extreme terror and fatal heart abnormalities. Indeed, Porges (1997, 2001) and Marks (1987) both speculated that so-called voodoo death (Cannon, 1957), which is characterized by prolonged motionless prior to death, results from a similar mechanism.

Affect and Etiology

In most cases catatonia is preceded by grief and anxiety, and in general by depressive moods and affects aimed against the patient by himself. Very common are anguish related to unhappy love, or self-reproach resulting from secret sexual misdemeanors. (Kahlbaum, 1874/1973, p. 33)

Despite Kahlbaum’s (1874/1973) observations, and the current acceptance of catatonia’s association with affective disorders, the possible role of affect in the genesis of catatonic symptoms has been largely overlooked. Indeed, Fink and Taylor (2003), on the basis of ECT’s efficacy in catatonia, have proposed that catatonia may be related to seizure disorders; however, a few swipes of Occam’s razor should be sufficient to see that severe depression, alleviated by ECT and strongly associated with catatonia, is a much more likely candidate. Further, recent models proposed for catatonia’s pathophysiology place little emphasis on affect (Fink & Taylor, 2003; Gurrera, 1999; Mann et al., 2003; Northoff, 2002), though—as will be seen in the next section—such considerations are not inconsistent with any of them.

When catatonia develops in the context of a medical condition, it is typically serious and often life threatening. Catatonia does not arise alongside relatively mild or benign medical conditions. The fact that the expression of catatonia does not vary whether it is associated with life-threatening medical conditions or profoundly dysphoric affective states (Carroll et al., 2000) and may respond to treatments that do not affect the underlying condition (Fink & Taylor, 1991; Ungvari et al., 2001) suggests that catatonia may best be viewed not as a component of these various conditions but as a reaction to them. Such a position is consonant with that of Patricia Rosebush (2003), who leads the largest prospective study of catatonia in the world, following 95 patients through 120 catatonic episodes to date. In a recent editorial, Rosebush and Mazurek’s (1999) observation that experiences of fear typically precede the development of catatonic symptoms, confirmed in case studies (Chandler, 1991; Wetzel et al., 1987), argues against this formulation. Alternatively, as suggested by Northoff (2002), the experience of overwhelming anxiety may be due to the loss of a cognitive capacity to contain (or extinguish) normal anxieties, consistent with decreased functioning of the orbitofrontal cortex. Thus, anxieties that might normally be managed in the course of daily life, such as worries that a loved one might die (Orland & Dagherestani, 1987), could take on gargantuan proportions and lead to catatonic symptoms. Of course, this possibility is not inconsistent with the notion of catatonia as a fear response but does not require the presence of anything other than normal anxieties or fears.

However, none of the above is to suggest that catatonia could not develop in the absence of anxiety, as a significant minority of affected individuals in Northoff et al.’s (1998) and Rosebush and Stewart’s (1989) studies did not report it. This should be viewed with caution, however, as in Rosebush and Stewart’s study, all 20 NMS sufferers exhibited “striking, frightened facial expressions” despite only 11 later describing experiencing “overwhelming anxiety” at the time. This, along with reports of amnesia for catatonic situations that trigger it (Nesse & Williams, 1994). The mechanism mediating this response may well be the emotions themselves, a particular admixture of anxiety and depression, “carying the code,” as Tooby and Cosmides (1990) contend, for this ancient behavioral pattern. Clearly, it may not be inappropriate to have a sense of impending doom when afflicted with a serious medical condition, as one’s life may well literally be at risk. Although this may not objectively be the case in psychiatric conditions, profound depression, overwhelming anxiety, or intense psychotic symptoms can no doubt trigger similar perceptions of inescapable proximal threat (Dixon, 1998). Further, this perception need not be a conscious one, as fear and anxiety can be produced solely through unconscious mechanisms (Armony & LeDoux, 1997; Nadel & Jacobs, 1996; Robinson, 1998). I would suggest that the anxious depression underlying catatonia is associated with a specific cluster of experiences—a perception of inescapable but at the same time amorphous danger, a sense of defeat and entrapment, and a sensation of imminent doom. Dixon’s (1998) notion of arrested flight—with all escape routes being blocked—is also of relevance. Recall that the perception of entrapment—not limited to physical contact—has been proposed to underlie not only TI in animals (Ratner, 1967) but also depression in humans (Dixon, 1998; Gilbert & Allan, 1998).

There are, of course, alternative explanations for the presence of anxiety in persons experiencing catatonic symptoms. Theoretically, the anxiety might simply be an epiphenomenon of biologically based changes, accompanying, but not driving, sympathetic activation or altered orbitofrontal functioning. However, Rosebush and Mazurek’s (1999) observation that experiences of fear typically precede the development of catatonic symptoms, confirmed in case studies (Chandler, 1991; Wetzel et al., 1987), argues against this formulation. Alternatively, as suggested by Northoff (2002), the experience of overwhelming anxiety may be due to the loss of a cognitive capacity to contain (or extinguish) normal anxieties, consistent with decreased functioning of the orbitofrontal cortex. Thus, anxieties that might normally be managed in the course of daily life, such as worries that a loved one might die (Orland & Dagherestani, 1987), could take on gargantuan proportions and lead to catatonic symptoms. Of course, this possibility is not inconsistent with the notion of catatonia as a fear response but does not require the presence of anything other than normal anxieties or fears.

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\[ \text{11 In all fairness to Fink and Taylor (2003), these are not necessarily mutually exclusive possibilities. Some (e.g., Post, Putnam, Contel, & Goldman, 1984) have posited that ECT exerts its action in depression through anticonvulsant–antikindling effects in the amygdala.} \]

\[ \text{12 This would also be true of traumatic situations, such as World War I battles, which have been reported to trigger episodes of catatonic stupor (Kardiner & Spiegel, 1947).} \]
episodes (Rosebush et al., 1990), suggests that some individuals who do not recall anxiety may nonetheless have been anxious during the catatonic episode. Even so, it is certainly conceivable that abnormalities in the motor loop, for example, in and of themselves could cause catatonic symptoms. However, it appears likely that in the majority of cases of catatonia, anxiety plays a major causative role.

Models of Pathophysiology

The mechanism through which affect may produce catatonia can be seen by an exploration of the various models of pathophysiology proposed, presented in Table 3. All models posit a primary role for the frontal cortex, with Northoff’s (2002) strongly emphasizing the right orbitofrontal cortex. Although the orbitofrontal cortex has a wide range of functions, one of the most important, particularly in the right hemisphere, is regulation of affective experience (Joseph, 1999; Schore, 1994, 2001, 2002). The orbitofrontal cortex has strong reciprocal connections with the limbic system, hypothalamus, and vagal nerve (Nauta, 1971, 1979), which are all more pronounced in the right hemisphere than in the left (Porges, Doussard-Roosevelt, & Maita, 1994; Schore, 1994, 2001, 2002). Through these connections, the orbitofrontal cortex monitors and modulates all aspects of affective experience, including the intensity and duration of emotions experienced and autonomic functioning (Dias, Robbins, & Roberts, 1996; Nauta, 1971, 1979; Schore, 1994). It is possible that the orbitofrontal cortex has a similar role in catatonia, as Northoff’s (2002) findings of decreased right orbitofrontal functioning reversed by BZ administration closely mirror work on panic disorder (Malizia, 2000, cited in Nutt & Malizia, 2001; Malizia et al., 1998). Indeed, Malizia et al. (1998) have argued that the right orbitofrontal cortex is central to the mediation of anxiety.

For his part, Northoff (2002) appears to have some recognition of this, as he accorded a primary role to anxiety in his conception of catatonia and allowed for the possibility that the deficit in right orbitofrontal functioning could be due to amygdala activity. He goes further in his response to commentaries on his *Behavioral and Brain Sciences* review, four of which emphasize the amygdala (Aleman & Kahn, 2002; Miu & Olteanu, 2002; Platek & Gallup, 2002; Savodnik, 2002), by stating that he presumes that the amygdala—whose primary role lies in processing fear stimuli and coordinating fear responses (Fendt & Fanselow, 1999; LeDoux, 1998)—plays a role in the pathophysiology of catatonia (Northoff, 2002, p. 593). The other models of pathophysiology presented in Table 3 are not so explicit on this point, but would all potentially allow a role for the amygdala. Fink and Taylor (2003) noted that “intense mood states” can lead to “limbic interference” in the pathophysiological circuit they propose, whereas Mann et al. (2003), Fink and Taylor (2003), and Gurrera (1999) all allowed a role for stress in the precipitation of catatonic episodes. Gurrera went so far as to suggest that “acute psychic distress” may be responsible for the frontal impairment underlying the disinhibition of the SNS. Although no studies have yet evaluated amygdala functioning in catatonia, the fact that both BZ and N-methyl-D-aspartate (NMDA)-antagonists block freezing when injected into the amygdala (Fendt & Fanselow, 1999) and improve catatonic symptoms (Northoff et al., 1997; Ungvari et al., 2001) is suggestive.

Finally, it is important to recall that R. Gjessing (1938), more than 65 years ago, linked physiological changes in catatonia to the

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<td><strong>Key components</strong></td>
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<td>Prefrontal cortex</td>
<td>Frontal cortex</td>
<td>Orbitofrontal cortex</td>
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<td>Anterior cingulate</td>
<td>Thalamus</td>
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<td>Basal ganglia</td>
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<td>SNS</td>
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<td><strong>Proposed mechanism</strong></td>
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<td>Frontal (motor) circuit dysfunction. Disconnection between motor regulatory and perceptual-integrating systems.</td>
<td>Dysfunction in multiple basal ganglia-thalamocortical circuits.</td>
<td>Deficit in right orbitofrontal cortex leading to abnormal modulation of basal ganglia.</td>
<td>Disruption in inhibition from frontal cortex (via hypothalamus) leading to dysregulated SNS hyperactivity.</td>
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<td><strong>Neurotransmitters emphasized</strong></td>
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<td>Dopamine–GABA</td>
<td>Dopamine</td>
<td>GABA</td>
<td>Dopamine</td>
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<td><strong>Role for amygdala?</strong></td>
<td></td>
<td>Explicit, though unproven. Amygdala could be responsible for orbitofrontal cortex deficit.</td>
<td>Potential, as “acute psychic distress” may be responsible for frontal cortex impairment.</td>
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*Note. SNS = sympathetic nervous system; GABA = gamma-aminobutyric acid.*

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sympathetically mediated emergency or stress reactions described by Cannon (1928); among contemporary theorists, autonomic dys-
function or sympathetic hyperactivity is emphasized by Fink and
Taylor (2003) and is central to Gurrera’s (1999) formulation of NMS.

**Implications and Future Directions**

When Kahlbaum (1874/1973) first proposed the concept of catatonia over 125 years ago, there was an awareness of its
connection to stressful and frightening events. That awareness
was lost until a few decades ago, when Gallup and Maser (1977) and
Perkins (1982) reintroduced it. The current conceptualization of
catatonia as a fear response deriving from TI is consistent with
their positions as well as with Kahlbaum’s original formulation,
evolutionary-based conceptions of depression, and much of the
recent research on the phenomenology, treatment response, and
pathophysiology of catatonia. In addition, positions recently articu-
lated by Georg Northoff (2002), the most prominent cognitive
neuroscientist working in this area, and Patricia Rosebush (2003;
see also Rosebush & Mazurek, 1999), who has the largest corpus
of catatonia cases, are entirely consistent with this thesis. Although
this position thus has significant empirical support, many questions
remain.

First, the prevalence and precise nature of the affect underlying
catatonia is still to be determined. If catatonia is overwhelmingly
associated with mixed affective states in mania, as Krueger and
Braeunig (2000) asserted, is it the depression that “drives” the
response? Does anxiety or agitation underlie “irritability” when the
latter is seen in mania with catatonic features? Is anxiety often
missed in catatonic stupor because psychomotor retardation does
not typically suggest anxiety? Systematic research, involving both
rating scales and interviews, is clearly needed to address these
questions.

Second, as there are strain differences in the propensity for
expression of TI (Gallup, Ledbetter, & Maser, 1976; Mills &
Faure, 1991), there may well be personality or physiological
predispositions to catatonia. Gurrera (1999) suggested a genetically
based sensitivity to stress could lead to the hyperactive SNS
seen in NMS, which Fink and Taylor (2003) endorsed for malig-
nant catatonia. If such a predisposition exists, however, it need not
be genetic. Heim and Nemeroff (2001) have found adverse child-
hood experiences to induce “a persistent sensitization of stress-
responsive neural circuits” (p. 1024), particularly related to mixed
depression–anxiety states (Heim et al., 2000). Further, early ad-
verse experiences may lead to not only increased SNS sensitivity
but, as Schore (2001) contended, excessively pruned connections
between the right orbitofrontal cortex and the amygdala and hy-
pothalamus, making it more difficult for the former to modulate
the latter structures. The consequences of this would be that even
low-level stressors could generate “unmodulated terrifying and
painful” emotional experiences, triggering “fear-freeze” behav-
ioral responses (Schore, 2001, p. 227). Future research will need to
establish whether early adverse experiences, common in major
depression (Kendler, Kessler, Neale, Heath, & Eaves, 1993), are
also frequent in individuals who manifest catatonic states.

Additionally, if catatonia is primarily a fear response as hypo-
thesized, attempts to code the facial expressions of persons in the
midst of catatonic episodes, with an approach such as the Facial
Action Coding System (Ekman & Friesen, 1978), should result in
a preponderance of anxious or fearful expressions. It should be
recalled that the frequent presence of fearful faces inspired the
name for the condition on which Kahlbaum (1874/1973) based his
concept of catatonia—melancholia attonita.

Finally, if Porges’s polyvagal theory does apply to catatonia as
proposed, there should be evidence for decreased VVC activity in
catatonia. VVC activity can reliably be measured by assessing an
individual’s respiratory sinus arrhythmia rate (Porges, 2001; Sa-
har, Shalev, & Porges, 2001). Respiratory sinus arrhythmias are
minor fluctuations in heart rate, coincident with respiratory
rhythms, which allow an individual to engage and disengage from
the social environment with minimal energy expenditure. One
would predict low activity during catatonic states and possibly
between episodes, as low VVC activity could indicate a vulnera-
bility to catatonic episodes.

Catatonia is not the only possible link between psychiatric
conditions and animal defense reactions. There are aspects of other
disorders potentially explainable by a progression from fight–
flight to TI, with perceived changes in level of danger, or predatory
imminence. Nijenhuis, Vanderlinden, and Spinphoven (1998) have
proposed just such a model for dissociative disorders, and post-
traumatic stress disorder, with its cyclic alterations between intru-
sive and avoidance symptoms, seems a good fit as well (Horowitz,
1978). Indeed, both dissociation and avoidance symptoms, which
allow the avoidance of distressing and arousing information, would appear to be the psychological equivalent of Dixon’s (1998)
cutoff behaviors in TI. Finally, could bipolar disorder, which some
see as integrally related to catatonia (Fink & Taylor, 2003; Ries,
1985), also be modeled on alterations between episodes of TI and
fight–flight? Depression has already been linked to TI (Dixon,
1998), and as some see manic excitement as parallel to catatonic
excitement (Fink & Taylor, 2003), such a proposal could clearly be
mounted. Thus, catatonia may be the best but is certainly not the
only example of the usefulness of exploring animal and evolution-
ary models of behavior as a means to understand psychiatric

disorders.

It is proposed here that the concept of catatonia as a fear
response provides more explanatory power than the dominant view
of catatonia as a motor disorder. Although the latter is superficially
accurate, it is unable to adequately explain many aspects of cata-
tonia, such as the presence of anxious and fearful expressions, the
potential lethality, the communicative abnormalities, and the pres-
ence of undirected aggressive behavior. Catatonia as a fear re-
response can explain all of the above in addition to the motor
disturbances. Conceptualizing catatonia as a motor disorder is a
relic of “black box” materialism, deriving from an inability to see
humans as responsive to the situations in which they find them-

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