THE ENCYCLOPEDIA OF NEUROPSYCHOLOGICAL DISORDERS

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The Encyclopedia of

NEUROPSYCHOLOGICAL DISORDERS
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The Encyclopedia of Neuropsychological Disorders

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To my wife and children for your love and support: You are my everything.
   To my parents, for your encouragement and many life lessons,
   among them the importance of hard work.
   
   CAN

I dedicate this book to my three daughters,
   Sarah, Whitney, and Heather.
   
   RSD

To my wife Mary, with all of my love.
   
   AMH
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Preface

The Encyclopedia of Neuropsychological Disorders was created as a reference manual of disorders that have a biological-psychological interaction. In many ways the title of this text can be seen as a misnomer. As professionals, we speak of neurological disorders and psychiatric disorders, but the concept of neuropsychological disorders is not readily used by the medical and psychological professions. The book makes this distinction in the discussion of the potential neuropsychological sequelae for an array of recognized neurological, psychiatric, as well as other medical disorders. As such, the book provides firm bases for numerous health care professionals to better understand and treat neurological, psychiatric, and neuromedical patients. While the book offers a wide array of disorders, the crux of the discussion is directed toward the fields of neuropsychology, neuropsychiatry, and behavioral neurology. Indeed, what we know of the functioning of normal and diseased brains has grown more in the last four decades than any other time in history. And, because there is an increased appreciation of the potential impact on the central nervous system by various unsuspected disorders, the variety of presentations now seen by clinicians in the neurosciences is steadily expanding. Along with this knowledge comes vast improvements in the approaches to diagnosis and treatment by professionals across a wider band of specialties. As a consequence, there is need for a concise and synthesized discussion of the presenting features of recognized disorders. With this in mind specifically, we sought to create a reference work in which individual disorders are discussed with the fields of neuropsychology, neuropsychiatry, and neurology in mind, covering those domains of clinical relevance with emphasis placed on empirically based information.

The internet can be a powerful tool, but in many ways it can be difficult to determine the reliability of that information. Thus, empirical backing of the information shared was of greatest importance. The product is designed to be useful to both veteran clinicians as well students in training. In all, the text includes a structured coverage of the clinical and neuropsychological features, neuropathological/pathophysiological correlates, diagnostic considerations, and methods of treatment for nearly 300 recognized disorders and diseases across the lifespan.
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The Encyclopedia of

Neuropsychological Disorders
**Acute Disseminated Encephalomyelitis**

**DESCRIPTION**

Acute disseminated encephalomyelitis (ADEM) is classified as a demyelinating disease of the central nervous system (CNS) and has onset in all age ranges, with more cases found in the pediatric population. Empirical studies suggest that ADEM is an immune-mediated inflammatory process that predominately involves the white matter of the brain. Onset may manifest spontaneously, or, in most cases, it is triggered by a systemic viral infection such as mumps, rubella, varicella-zoster, herpes simplex virus, hepatitis A virus, and coxsackie virus. There are rare cases in which vaccinations have triggered this disease.

Symptoms are characterized by rapid onset of encephalopathy with or without meningeal signs and focal/multifocal neurological signs (Alper, Heyman, & Wang, 2008; Marchioni et al., 2008; Sejvar, 2008; Tenembaum, 2007). Altered mental status, as minimal as lethargy or as severe as coma, may be present. Some focal neurologic deficits may be present, specifically extrapyramidal or pyramidal signs, which are shown in 60–90% of cases. Hemiplegia is present in 50–75% of cases, cranial nerve deficits in 7–45%, and concomitant spinal cord involvement in 25% (Sejvar, 2008). These symptoms are generally short lived with resolution over weeks or months. Complete recovery has been reported at 57–89% (Alper et al., 2008).

Although ADEM shares similar characteristics to other CNS disorders, it can be distinguished because of its rapid onset, progression of the illness, rapid remission, and the characteristic pattern and distribution of brain lesions on a MRI. However, none of these differences are pathognomonic of ADEM making diagnosis unclear. Furthermore, symptoms of ADEM are most similar to first episodes of multiple sclerosis (MS), which makes it difficult to distinguish (Marchioni et al., 2008; Sejvar, 2008). ADEM can be classified into two types. The classic form is characterized by brain and spinal cord involvement, and there is a site-restricted form that is characterized by pure encephalitis, myelitis, cerebellitis, and optic neuritis. Peripheral nervous system (PNS) involvement is suggested in 5–43% of cases (Marchioni et al., 2008).

**NEUROPATHOLOGY/PATHOPHYSIOLOGY**

Neuroimaging studies have suggested that ADEM is a result of an inflammatory autoimmune response with resultant CNS inflammation and demyelination. A lack of biological markers make the pathophysiological basis of the condition unclear. Though the pathophysiological underpinnings of this response are unclear, one suggested explanation for ADEM, as well as other inflammatory CNS and PNS disorders, is that factors stimulate the immune system to produce antigen-specific humoral and/or cellular immunity (Sejvar, 2008; Tenembaum, 2007).

A few mechanisms have been offered to explain the process of immune stimulation induced by an infection or vaccination. "Molecular mimicry" is a term used to describe the involvement of epitopes of a virus, vaccine, or other antigenic stimulus in developing immune antibodies and/or T cells that cross-react with epitopes on myelin or axonal glycoproteins of nerves. Another mechanism describes the initial event as the binding of cross-reactive antibodies and subsequent damage to oligodendrocytes. Also, the introduction of sequestered myelin antigens into the circulation could damage myelin cells and therefore incite autoimmunity (Sejvar, 2008).

**NEUROPSYCHOLOGICAL/CLINICAL PRESENTATION**

In most cases, the clinical course of ADEM involves a rapid onset and resolution of the disorder while resulting deficits develop within days. Predominately spontaneous recovery (of neurological deficits) occurs over a period of weeks to months. Some cases result in persistent motor deficits, cognitive impairment, and recurrent seizures. In general, outcome is favorable in adult ADEM (Sejvar, 2008). One study of adult ADEM followed patients diagnosed with ADEM over a period of 30 months and found some residual
mild cognitive impairment, as shown by problems with memory and concentration, and speech (Hollinger, Sturzenegger, Mathis, Schroth, & Hess, 2002). Therefore, the literature supports that in adult cases of ADEM, long-lasting neuropsychological and behavioral deficits are minimal or nonexistent.

Long-term neuropsychological or behavioral impairment in children with history of ADEM seems to be minimal or nonexistent as well. Although cognitive and motor deficits predominate in the initial stages of ADEM in adults, children may initially present with behavioral disturbances prior to diagnosis of ADEM. One study reported two cases where the child initially presented with symptoms of abnormal behavior such as irritability, violent tendencies, and behaviors mimicking delusions, suggestive of acute psychotic disorder. This suggests a different psychological manifestation of the disorder in some children. However, both these cases had positive outcomes and no residual cognitive, academic, or neuropsychological deficits were found (Krishnakumar, Jayakrishnan, Beegum, & Riyaz, 2008). This is consistent with the current literature that states serious complications are rare in childhood ADEM. However, some studies have found that the rate of relapse is considerable in this population (Anlar et al., 2003; Hollinger et al., 2002).

DIAGNOSIS

The major differential diagnosis of ADEM is MS. Interestingly, it has been suggested that 10–30% of patients initially diagnosed with ADEM end up developing MS (Sejvar, 2008). Two more differential diagnoses are also important: that is, infectious meningoencephalitis of possibly treatable etiology and acute brain swelling (Hollinger et al., 2002; Schwarz, Mohr, Knauth, Wildemann, & Storch-Hagenlocher, 2001).

MRIs generates similar results for MS. An MRI image of a case with ADEM would typically include widespread, bilateral, and asymmetric involvement of the white matter, deep gray nuclei, and spinal cord (Beleza et al., 2008). This is similar to MRI findings of 18% of children who are ultimately diagnosed with MS, making it indistinguishable (Alper et al., 2008; Callen et al., 2008). Several neuroimaging studies have made suggestions on how to distinguish between ADEM and first episodes of MS. Callen et al. (2008) proposed that the criteria for pediatric MS should include any two of the following: ≥ 2 periventricular lesions, presence of black holes, or absence of diffuse bilateral legion distribution pattern. It has also been suggested that, in many cases, the presence of encephalopathy is a strong indicator for the diagnosis of ADEM (Alper et al., 2008).

Recently, four patterns of MRI results in ADEM have been identified. These include ADEM with small (<5 mm) lesions, with large confluent lesions with edema and mass effect, with symmetric bilateral thalamic involvement, and acute hemorrhagic encephalomyelitis with features of hemorrhage within demyelinating lesions. However, correlation of the above to clinical outcome has not been found (Sejvar, 2008).

It is not sufficient to rely solely on one diagnostic procedure when diagnosing ADEM. Some minimal requirements have been identified that include a preceding infection, a monophasic disease course, neurological findings that indicate a disseminated CNS disease, and an absence of metabolic or infectious disorders (Hollinger et al., 2002). The classic definition of ADEM does not consider the PNS aspect of the disease. This type is found in 5–43% of cases and is associated with worse prognosis for functional recovery after the first episode and likelihood of relapse (Marchioni et al., 2008).

Relapse of ADEM is an issue of concern particularly in pediatric ADEM. Currently, relapse has been reported to occur in 5–25% of cases (Marchioni et al., 2008; Tenembaum, 2007). There are two categories of this relapsing form of ADEM. The disease can be classified as “recurrent” if it recurs at least 2 months after its onset, and the lesions affect the same area as in the first episode. ADEM can be classified as multiphasic, if the lesions present dissemination in space and time (Marchioni et al., 2008; Supplej et al., 2008).

TREATMENT

The treatment of choice for ADEM lies in different immunosuppressive or immunomodulatory strategies. These options are thought to be effective under the assumption that ADEM is the result of an autoimmune response against CNS structures. Some medical treatment examples of this nature are high-dose corticosteroids, plasmapheresis, or intravenous immunoglobulins. Corticosteroids are considered to be the first choice and most widely used treatment for ADEM. Treatment regimen, in most cases, includes intravenous methylprednisolone or dexamethasone for 3–5 days, followed by an oral taper for 3–6 weeks. Steroid treatment may be administered. However, this has been associated with relapse. These treatments combined with rehabilitation for any cognitive or neurological impairment or residual physical
deficits, such as cognitive, occupational, and physical therapy, should result in full recovery of the disorder in a relatively short period of time (Hollinger et al., 2002; Sejvar, 2008).

Miriam Jocelyn Rodriguez
Charles Golden


ADRENOLEUKODYSTROPHY

DESCRIPTION

Adrenoleukodystrophy, also referred to as ALD, is one of a group of genetic disorders referred to as leukodystrophies. These disorders are so named as they correspond with deterioration of myelin within the central nervous system (CNS). ALD specifically is characterized by the widespread demyelination of white matter throughout the CNS and atrophy in the adrenal gland secondary to a buildup of very long chain fatty acids (VLCFAs). Formerly classified along with other presentations under the umbrella of Schilder’s disease (Beers, Porter, & Jones, 2006), it is now recognized as an independent metabolic encephalopathy (Ropper & Brown, 2005).

Two different subtypes fall under the umbrella of ALD. The most common is the X-linked form, which corresponds with a genetic abnormality on the sex chromosome, thus affecting young males rather than females. Onset is usually before age 7 (approximately 4–10 years of age) but may present in young adults in the early 20s as a more slowly developing disorder. The child cerebral form is the most common form and also the most severe. In some cases, it may occur in neonatal stages of development as an autosomal recessive type that represents the second subtype.

Symptoms present across cognitive, sensorimotor, academic, emotional, and behavioral domains as discussed under Neuropsychological/Clinical Presentation. As the disease progresses, neural impulses become further broken and thus greater functional impairments are seen. Given the progressive nature of the disease, prognosis for the childhood form is poor with individuals usually succumbing 1–10 years after initial symptom onset. In comparison, the adult form does not usually onset until the early to late 20s, sometimes early 30s, and is associated with a far slower and milder progressive course. Nevertheless, prominent cerebral deterioration can be seen. The disease is still noted far more in males compared with females, although an even milder form of adult-onset ALD has been noted in females who are carriers of the disease.

NEUROPATHOLOGY/PATOPHYSIOLOGY

ALD most commonly presents as an X-linked recessive trait that corresponds with an inability to
metabolize VLCFAs and subsequently their accumulation in the brain and adrenal glands. This leads to demyelination and progressive dysfunction of the adrenal gland, corresponding with the clinical presentation of ALD as discussed below. In the X-linked ALD, the genetic abnormality has been mapped to the Xq28 region resulting in the mutation of the ALDP gene, which encodes a peroxisomal membrane protein member of the ATP-binding cassette family (Maertens & Dyken, 2007).

Several pathological abnormalities in ALD patients have been noted. First, they have elevated levels of cerebrospinal fluid’s protein content that corresponds with their inability to efficiently metabolize VLCFAs. The adrenal glands may be hard to identify as they are commonly severely atrophic and correspond with adrenal insufficiency. In addition, adrenal biopsies in the zona fasciculata and reticularis show microvacuoles, striated cytoplasms, and many ballooned cytoplasmic cortical cells (Lake, 1997; Maertens & Dyken, 2007).

MRI scans reveal diffuse demyelination and CT scans may show hyperdense or hypodense regions in the parieto-occipital white matter, with the frontal lobes being less involved. As such, on the cortical surfaces, the brain and spinal cord are usually normal, but on slicing, the involved white matter is gray and firm, and in most cases, there is a caudorostral progression of the leukodystrophy (Lake, 1997). This white matter degradation presents bilaterally and generally symmetrically. Still, pathological review suggests particular susceptibility of the optic nerves, the fornix, hippocampal commissure, posterior cingulum, posterior limbs of the internal capsule, and the corpus callosum with characteristic contrast enhancement at the rim of the lesions (Lake, 1997; Maertens & Dyken, 2007; Powers, 1985).

NEUROPSYCHOLOGICAL/CLINICAL PRESENTATION

The disease is progressive in nature and is thus associated with a gradually expanding clinical picture in terms of functional compromise. Again, the childhood-onset form is not only the most common but also the most severe type. Beginning between the ages of 4 and 10, a gradual onset of behavioral and functional changes may be seen. Children may begin to act out more, appearing regressive in their behavioral regulation. Motoric deficits may also begin to be seen in the form of diminished coordination and eventually disturbance of gait and spasticity. Independent ambulation eventually becomes impossible, and in some instances, even quadriplegia is seen, and thus individuals become wheelchair bound (Maertens & Dyken, 2007). Increasing difficulties in memory and school performance are also seen early on. As the disease continues to progress, individuals often develop dysphagia, deafness, blindness (due to demyelination of the entire visual pathway), aphasia, and mental disorientation leading to dementia (Beers et al., 2006; Dox, Melloni, Eisner, & Melloni, 2002; Rowland, 2005). In addition, seizures may develop but do so in less than one-third of patients (Naidu & Moser, 1994). Other symptoms include characteristic signs of adrenal failure. This includes salt craving, hyperpigmentation in skin folds, vomiting, and fatigue. Eventually, deterioration culminates in a vegetative state (Maertens & Dyken, 2007).

There is also an adolescent-onset form, in which the signs and symptoms of cerebral involvement are the same as those seen in the childhood form but instead develop between the ages of 10 and 21 (i.e., the period between childhood onset and adult onset) (Lake, 1997).

In the adult-onset form, the disease is primarily characterized by motoric changes early on. Individuals will demonstrate progressive rigidity that leads to a decrease in gross motor magnitude, amplitude, and coordination. Preferential compromise of the lower extremities can at times be seen leading to paresis or paralysis, whereas the upper limbs are largely spared (Lake, 1997). Ataxia is also commonly noted. The array of motor anomalies is directly related to the disease’s infiltration of spinocerebellar regions (Maertens & Dyken, 2007). Spastic paraparesis tends to occur later in life (i.e., 30s–40s), again with notation of a slow progression (Ropper & Brown, 2005). Psychiatric disturbances and seizures are also commonly seen (Maertens & Dyken, 2007). Finally, cognitive deterioration can occur in the adult-onset form, although the progression is far slower and tends not be of the same degree of severity. When presenting in women who are carriers of the disease, these features are milder; however, it is worth noting that neurological symptoms have been reported to occur in less than 50% of these women (Ropper & Brown, 2005).

DIAGNOSIS

Diagnosis of ALD is often multifaceted, although some methods are more favored and definitive than others. Specifically, identification of an excess of VLCFAs and than other biochemical abnormalities noted previously is the major method of making the diagnosis (Maertens & Dyken, 2007) and may be viewed as the definitive means of diagnosis. Low serum sodium and chloride levels and elevated potassium levels are also noted on laboratory workup corresponding with degradation of the adrenal glands (Ropper & Brown, 2005).
genetic testing can identify the abnormality as discussed previously and has been suggested in early identification and even prenatal diagnosis. Diagnosis during the first trimester can be accomplished by the measurement of VLCFA ester in chorionic villus samples and by restriction fragment length polymorphism using a DNA probe (Boué et al., 1985).

Although the above are considered definitive, it is noted that often these laboratory tests are only carried out after previous clinical workup including taking of a thorough history, neurological and/or neuropsychological workup, and neuroimaging suggest the potential of ALD. When considering ALD, both CT and MRI scans are viewed as helpful clinical tools (Lake, 1997). Commonly, MRI scans can demonstrate the diffuse white matter disease that is the hallmark of the presentation, which in combination with noted clinical features can lead to the aforementioned laboratory tests.

TREATMENT

Prognosis for patients with ALD is poor with those stricken with the disease succumbing to its effects within 1–10 years of symptom onset. Neurological deterioration progresses until patients finally enter a vegetative state; however, patients generally die from adrenal crisis or other causes soon after. Although the disease course cannot be halted, there is some evidence to support methods of slowing progression and addressing symptoms. Adrenal replacement therapy prolongs life and occasionally effects a partial neurological remission. Lorenzo’s oil (glyceryl trierucate/glycerol trioleate: 4/1) therapy can diminish the frequency and severity of neurological disability if started early, before any symptoms appear (Lake, 1997). Bone marrow transplantation has also demonstrated some benefit when done early, but the efficacy is limited (Krivit, Lockman, Watkins, Hirsch, & Shapiro, 1995). Specifically, although the bone marrow transplantation seems to cure the biochemical defects, it does not affect radiological or neurological defects. All cases show neurological progression regardless of transplantation (Beers et al., 2006). The procedure also carries risk of mortality and morbidity and is not recommended for those whose symptoms are already severe or who have the adult-onset or neonatal forms.

Oral administration of docosahexanoic acid may help infants and children with neonatal ALD. In addition, when administered before the age of 6, a diet enriched with monounsaturated fatty acids and devoid of long chain fatty acids has been said to slow the progress of the disease. Therapeutic plasma exchanges are successful in reducing VLCFA but without altering the clinical course.

Although the above are considered options for treatment, in many ways still therapy is primarily directed at supportive care and treatment including physical therapy, psychological support, and special education.

J. Aaron Albritton
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ADULT ANXIETY DISORDERS

DESCRIPTION

It may appear inappropriate to characterize psychopathology associated with anxiety as a neuropsychologically based condition, since traditionally the
anxiety disorders have been thought to be the result of environmental stressors, and, particularly within a psychoanalytic framework, stressors occurring during early life. However, with recent developments in neuroscience, a literature has developed that has been supportive of the view that there are neurobiological aspects to at least some of these disorders. Anxiety is an affect characterized by unpleasant features involving physical and psychological experiences. Physically there may be disturbed breathing, increased heart activity, trembling, paralysis, and sweating. Psychologically there are unpleasant feelings and sensations, and a sense of apprehension. Traditionally, anxiety is distinguished from fear, in that fear is associated with objective real or threatened danger, whereas anxiety is more associated with unreal or imagined danger. Although anxiety itself is a commonly experienced and normal human emotion, some individuals experience anxiety or fear to such a degree that it interferes with their lives. At the point when anxiety becomes debilitating, it is likely that the person would be diagnosed with one of the anxiety disorders as a psychiatric diagnosis currently described and defined in the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR; American Psychiatric Association [APA], 2000).

The term Anxiety Disorders in the DSM-IV is actually an umbrella that covers all pathological disorders that are characterized by intense anxiety, fear, or stress. These include Panic Disorder With or Without Agoraphobia, Specific Phobia, Social Anxiety Disorder, Obsessive-Compulsive Disorder, Posttraumatic Stress Disorder, Acute Stress Disorder, and Generalized Anxiety Disorder. Anxiety disorders are often found to be comorbid with mood disorders or other anxiety disorders, but comorbid conditions, most notably substance abuse disorders and major depression (Stein & Chavira, 1998). Obsessive-Compulsive Disorder (OCD) is characterized by obsessive thoughts that provoke anxiety and/or compulsions or repetitive behaviors that help to neutralize anxiety. People with OCD feel that their compulsive behaviors reduce the stress brought on by the obsessive thoughts, but these may or may not be logically related. In order to be considered OCD, the prevalence of these thoughts and actions must impair the person’s life in social or occupational functioning.

Posttraumatic Stress Disorder (PTSD) is a condition that may arise after a severely traumatic event. Symptoms include reexperiencing the event, feeling great anxiety or tension when reminded of the event, increased arousal and avoidance of stimuli that may be associated with the traumatic event. PTSD may be significantly disabling and has been reported to cause significant work production losses in the United States (Zatzick et al., 2008). The symptoms of Acute Stress Disorder (ASD) are similar to those of PTSD but occur immediately following the traumatic event and typically have a shorter duration.

Generalized Anxiety Disorder (GAD) is characterized by a pervasive, excessive, and uncontrollable continuous worry about life events and activities.
It affects women twice as much as men. Patients may also suffer from physical ailments that manifest their worry, such as fatigue, restlessness, and muscle tension. GAD is the most common anxiety disorder and is also one of the most common comorbid disorders with other anxiety and mood disorders (Kessler, Chiu, Demler, Merikangas, & Walters, 2005). Patients with GAD tend to have a chronic course with early onset (Newman, 2000).

Anyone that presents with significant anxiety symptoms or phobic avoidance that does not fully meet DSM-IV criteria for any of the above-specified disorders can be classified as having Anxiety Disorder NOS (Not Otherwise Specified).

NEUROPATHOLOGY/PATHOPHYSIOLOGY

The neuropathology and pathophysiology of the anxiety disorders are inextricably associated with the area of the neurobiology of stress and fear, which have an enormous literature. There is extensive research evidence indicating that stress has consequences for brain function based both on animal model and human studies. A good example is the now classical work of Sapolsky on how autonomic changes, notably involving glucocorticoids, influence the functioning of the hippocampus (Rodrigues, LeDoux, and Sapolsky, 2009) and the extensive work of LeDoux on the amygdala and fear conditioning (e.g., Delgado, Jou, LeDoux, & Phelps, 2009).

Some of the neurobiology of anxiety disorders is genetic or developmental, and some relates to interaction with the environment (Stein, Jang, Taylor, Vernon, & Livesly, 2002). A portion of the neurobiology of the anxiety disorders has to do with developmental vulnerabilities, whereas other aspects are considered to be the consequences of stress. Areas of the brain involved appear to be in the limbic system, with major emphasis on the amygdala and hippocampus. There are also neurochemical considerations. For example, many researchers believe that the glutamate receptor, the primary excitatory neurotransmitter in the brain, is a key contributor to mood and anxiety disorders (Sanacora, Kendell, Fenton, Coric, & Krystal, 2004; Zarate et al., 2004). To this effect, several studies have been conducted looking to find symptom relief in mood and anxiety disorders with a variety of glutamate modulators (Mathew et al., 2008; Sanacora et al., 2004; Zarate et al., 2004).

Neuropathologies and pathophysiology may differ according to type of anxiety disorder. In PD, patients have been found to have anatomical changes in the form of decreased anterior cingulate cortex (ACC) volume (Asami et al., 2008). Childhood Separation Anxiety Disorder often predates PD, and it has been shown that both disorders predict heightened sensitivity to inhaled CO2. Researchers have found that genetic predispositions are correlated with both this developmental trajectory and CO2 sensitivity and interact with the environmental event of parental loss as a significant mediating factor in both disorders (Battaglia et al., 2009). Researchers have found correlations with PD diagnosis and certain chemicals in the brain, as well: For instance, serotonin receptor binding is significantly reduced in patients as compared with healthy controls, and this dysfunction improves in treated patients (Nash et al., 2008).

Individuals with SP have greater activation responses in the insula and ACC to pictures of phobic avoidance than controls (Straube, Glauser, Dilger, Mentzel, & Miltner, 2006). In SAD, there is significant neurological evidence for the precipitating brain areas in which symptoms occur. Functional magnetic resonance imaging (fMRI) studies have shown consistently that the ACC is involved in experiencing physical pain but more recently some researchers have found the same area to also be involved in pain or discomfort arising from social situations, such as perceiving group exclusion (Eisenberger, Lieberman, & Williams, 2003). These same researchers also showed that ACC activation during social exclusion was correlated with self-reports of distress.

There has been extensive neurobiological research regarding PTSD involving both human studies and animal models. Summarizing an enormous literature, there appears to be a consensus that the amygdala, insula, and ACC all play crucial roles in PTSD symptomatology (Damsta, Kosel, & Mousally, 2009). Neurobiological studies now implicate both developmental or genetic and acquired aspects.

Wakizono et al. (2007) found that there is reduction in inhibition of memory circuits for fear extinction in animals due to exaggerated amygdala response associated with hypofunctioning medial prefrontal cortex (PFC) and hippocampus, but strain differences were found. Thus, in animal models of PTSD it appears that individual characteristics that mediate responses to stress and therefore the onset of PTSD can be genetic or environmental or an interaction of these two factors. A twin study showed that both exposure to trauma such as violent crime and ensuing PTSD symptoms are mediated by genetics and environment, whereas exposure to trauma via accidents and natural disasters are influenced only by shared environmental factors (Stein et al., 2002).

Bremner et al. (2003) is among many others that have found that characteristic features of brain
dysfunction in PTSD patients (see also Gurvits et al., 1996; Liberzon et al., 1999). For example, while remembering negatively emotionally valenced words in a memory test, women with PTSD show very different fear and emotion brain pathways than women without PTSD, in a network that has been implicated in PTSD problems in previous studies. Patients with PTSD have shown decreased response to reward receipt, which may underlie decreased motivation associated with the disorder, most notably in animals exposed to inescapable shock (Maier & Seligman, 1976; Sailer et al., 2008). These deficits have been shown in fMRI studies to be associated with the PFC, which is responsible for decision making, and the nucleus accumbens, which is important in the reward pathway (Krawczyk, 2002; Sailer et al., 2008).

The severity of GAD symptoms are highly correlated with salivary cortisol levels (Mantella et al., 2008). This evidence points to hypothalamic-pituitary-adrenal (HPA) axis dysfunction, which is associated with constant stress and worry and theorized to cause continual activation of the HPA axis. It is also speculated that the amino acid N-acetylaspartate’s (NAA) occurrence in the hippocampus is related to severe anxiety. A recent study measuring levels of hippocampal NAA while treating GAD patients with Riluzole (an approved treatment for amyotrophic lateral sclerosis) showed that these levels dropped significantly during and after the course of treatment as compared to non-anxious controls, and great symptom relief was also seen (Matthew et al., 2008).

NEUROPSYCHOLOGICAL/CLINICAL PRESENTATION

It is generally understood that individuals with anxiety disorders typically do not have apparent neuropsychological deficits and that cognitive changes, when present, tend to be subtle and mild. Indeed, individuals with OCD may be exceptionally bright. The major descriptive considerations for these individuals are associated with personality, lifestyle, and symptoms associated with anxiety. Apparent cognitive features of these individuals may relate to cognitive style, such as the preoccupation with detail seen in OCD or amnesia for traumatic events seen in ASD or PTSD. People with anxiety disorders typically have very low quality of life, as 20% to 59% of patients diagnosed on the spectrum of anxiety disorders score at least two standard deviations below the community norm on a Quality of Life Scale (Rapaport, Clary, Fayyard, & Endicott, 2005).

Not all anxiety disorders present in the same way. In a study using the startle blink reflex, Grillon, Ameli, Goddard, Woods, and Davis (1994) showed that patients with PD overgeneralize conditioned fear to stimuli providing a danger cue. That is, they respond with a fear response to stimuli more distant from a conditioned danger cue than is the case for normal controls. Decreased function of short-term memory and attention has also been reported in people with PD (Gordeev, 2008). Onset of situational phobias is much later in life than the other subtypes. Animal phobics, however, do not have a fear of being harmed so much as an intense disgust (Lipsitz, Barlow, Manuzza, Hofmann, & Fyer, 2002).

A study performed by Rapee and Lim (1992) had observers rate the performances of people with SAD in social situations, and the subjects also rated their own performances. The observers rate the subjects much higher than the subjects rated themselves, showing that people with SAD have some cognitive distortions about their performance in social situations. Other studies have shown, however, that people with SAD actually do perform worse in social situations than people with an anxiety disorder (Thompson & Rapee, 2002; Voncken & Bögens, 2008), although these deficits are seen only in interactive social situations, not in solo actions such as speech giving (Voncken & Bögens, 2008). Voncken and Bögens (2008) showed one cognitive distortion that is characteristic of social anxiety sufferers — there is a great discrepancy between how patients perceive themselves and what they believe others want them to be. This theory provides logic as to why SAD patients are fearful in social situations — because they feel unable to live up to others’ expectations, and are constantly failing others.

Clinicians have long seen a great deal of heterogeneity in OCD, but a recent meta-analytic study found that while obsessions and compulsions range quite widely, they can be reduced to symmetry in ordering, forbidden thoughts, cleaning, and hoarding (Bloch, Landerers-Weisenberger, Rosario, Pittenger, & Leckman, 2008). Research has shown that patients with OCD also respond more extremely and more quickly to startling images (Kumari, Kaviani, Raven, Gray, & Checkley, 2001). Unfortunately, OCD symptoms tend to be stable across time (Mataix-Cols et al., 2002).

There are numerous studies of cognitive function in OCD, particularly involving memory and executive function. Performance on the Wisconsin Card Sorting Test in those with OCD has shown worse perseveration than found in normal controls; visuospatial deficits have also been found (Bucci et al., 2007). Others have
identified a cognitive profile characterized by weaknesses in motor function and processing speed and strengths in language, verbal memory, and reasoning/problem-solving (Burdick, Robinson, Malhotra, & Szeszko, 2008). A suggested basis for the deficits found is altered corticostriatal functional connectivity involving the dorsal striatum and lateral PFC.

Although the severity of PTSD symptoms has been shown to be negatively correlated with the strength of one’s social support network (Scarpa, Haden, & Hurley, 2006), researchers have recently discovered that social support does not mediate time to remission of symptoms (Laffaye, Cavella, Drescher, & Rosen, 2008). Some of these symptoms are neuropsychological in nature. There has been substantially more research conducted in the neuropsychological aspects of PTSD than any other anxiety disorder. The general consensus appears to be that even when one accounts for intelligence, education, alcoholism, and depression (Bremer et al., 1995, 2004; Samuelson et al., 2006; Vasterling et al., 2002), there is impairment of verbal declarative memory and attention associated with PTSD itself. These deficits appear to coexist with the common traumatic and negative emotionally valenced memories generally found in PTSD and the preference for remembrance of trauma-related material (Elzinga & Bremer, 2002). PTSD sufferers also experience early amnesia immediately after trauma exposure, which is correlated with later severity of symptoms (Granja et al., 2008).

ASD is very similar to PTSD except that it remits much more quickly. A diagnosis of ASD following a traumatic event is a high-risk factor for developing PTSD, which has a much longer course (Brewin, Andrews, Rose, & Kirk, 1999; Fullerton, Ursano, & Wang, 2004). Symptoms of PTSD have been noted in elderly veterans who experienced the “Bataan Death March” during World War II (Goldstein, van Kammen, Shelly, Miller, & van Kammen, 1987). Unfortunately, although it is established in the literature that a diagnosis of ASD predicts later diagnosis of PTSD, much more research needs to be conducted to see if early, aggressive treatment of ASD can stave off a diagnosis of PTSD (McKibben, Bresnick, Wiechman, & Fauerbach, 2008). ASD is highly correlated with dissociative symptoms (Ginzburg, Solomon, Dekel, & Bleich, 2006), which are characterized by a disruption in a person’s consciousness, memory, and/or identity (APA, 2000). Persons that experience some kind of trauma and then proceed to blame others for their suffering are much more likely to develop ASD (Lambert, Difede, & Contrada, 2004), although the reasoning behind this phenomenon has yet to be illuminated.

Patients with GAD have been found to negatively evaluate their internal experiences (such as thoughts, emotions, and sensations) and use worry as a means of escaping and avoiding these experiences (Roemer, Orsillo, & Salters-Pedneault, 2008). Researchers now believe that patients with GAD not only have problems with worrying, but worry about their worry (called meta-worry), as they view their worrisome thoughts as dangerous and uncontrollable (Wells & Carter, 2002). It is believed that the implications for treatment from this new theory are that patients must learn to view worrying as a negative strategy for dealing with threat and also learn new behavioral strategies for appraising threats in the environment (Well & Carter, 1999). As with most anxiety disorders, GAD has been found to be associated with very low quality of life (Pollack et al., 2008). Although patients with GAD report experiencing negative thoughts that are unpleasant and anxiety-provoking, they have been shown not to present with signs of hyperarousal measured by skin conductance and heart rate (Upatel & Gerlach, 2008). Although individuals with GAD often have difficulties concentrating, there does not appear to be a specific cognitive literature on this disorder separate from phobias, PTSD, and OCD. Apparently, the disorder is so heterogeneous, often including the symptoms of other anxiety disorders and other comorbidities, that there can be no specific neuropsychological profile.

DIAGNOSIS

The Anxiety Disorders are a class of mental disorders, and there is a general consensus in the professional community to accept the diagnostic criteria contained in the various editions of the DSM-IV-TR. The latest version (DSM-5) is in preparation at this writing. Therefore, diagnoses are made by determining whether the client meets the required number of criteria contained in the manual. Most clinicians use an interview and history review to determine whether those criteria are met, but a more precise and scientifically acceptable method is to use one of the structured psychiatric interviews. The most commonly used procedure at present is the Structured Clinical Interview for DSM-IV-TR (SCID) (First, Spitzer, Gibbon, & Williams, 2005) that will shortly be replaced by the SCID for DSM-5. Use of the SCID generally is a requirement for publication of diagnoses in professional and scientific journals and may be used in general clinical practice at the discretion of the psychologist or psychiatrist in routine clinical practice. It may be useful to supplement the SCID with one of many anxiety
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scales available and with psychological personality testing. The most commonly used anxiety scales are the Beck Anxiety Inventory, the Hamilton Anxiety Rating Scale, and the Spielberger State-Trait Anxiety Inventory (STAI). Numerous scales have been developed for PTSD including the Trauma Symptom Inventory, two Mississippi Scales, one for civilians and the other for the military, the PTSD Checklist, and others. The diagnosis of an anxiety disorder is therefore ideally made using a structured interview to evaluate presence of DSM-IV-TR criteria, and special scales as indicated. These criteria are briefly reviewed in what follows.

As indicated by the DSM IV-TR, clients are diagnosed with PD if they have recurrent panic attacks with four or more of the following symptoms: palpitations or accelerated heart rate, sweating, shaking, shortness of breath, feeling of being choked, chest pain, nausea, feeling faint, derealization or depersonalization, fear of losing control or going crazy, fear of dying, numbness or tingling, and/or chills or hot flushes (APA, 2000). Also, at least one panic attack must have been succeeded by a month or more of one or more of the following symptoms: worry of additional attacks, what they may mean or signify, and/or a change in behavior due to the attack. These attacks cannot be related to agoraphobia or due to the effects of a medical condition or substance. PDA follows the same criteria as for PD, but panic attacks are often caused by agoraphobia. One could also be diagnosed as having agoraphobia without history of PD.

For SP, clinicians must specify whether the client suffers from Animal, Natural Environment, Blood-Injection-Injury, Situational, or Other type.

In SAD the person meets criteria for phobia and knows that the fear is excessive, but the social or performance situations are either avoided or endured with intense distress (DSM IV-TR). In order for this to be diagnosable, the avoidance of fearful situations must significantly interfere with the person's daily life, occupational or social functioning, or have great distress. These symptoms cannot be accounted for by substances or any other mental disorder. Some overlapping diagnoses are Panic Disorder With or Without Agoraphobia, Body Dysmorphic Disorder, or Schizoid Personality Disorder. The fear or criticism can also not be related to a different, medical disorder, such as abnormal eating behavior in the presence of an eating disorder. There is a Generalized specifier if the person fears almost all social situations, not simply a few stressful ones.

In order to be diagnosed with OCD, one must have either obsessions or compulsions, or both. Obsessions are recurrent thoughts, impulses, or images that are thought to be intrusive and inappropriate and cause distress, which are not just excessive worries about real problems. They cannot be suppressed, ignored, or distracted away, and the patient must know these thoughts are from his own mind, not from an outside source. Compulsions are defined repetitive behaviors or mental acts that one feels compelled to complete in response to an obsession, or according to extremely strict rules. Some common examples of compulsions are hand washing, checking, and repeating words silently. These behaviors are intended to reduce distress or prevent a terrible event, but they are not connected in a realistic way to the observer. Also, at some point the patient has been aware that these compulsions and obsessions are excessive or unreasonable, and they take up more than an hour of the patient's day, everyday, and cause marked stress or interference in domains of daily life, such as occupational and social functioning.

Some obsessions and compulsions are considered separate disorders, such as obsession with one's appearance seen in Body Dysmorphic Disorder and obsession with food seen in Eating Disorders. Clinicians can specify whether a patient has Poor Insight, if the patient does not see these problems as excessive for most of an episode.

Regarding PTSD, witnessing a traumatic event that made the patient feel that his life was threatened or an event that included serious injury or death of another is the first criteria that must be fulfilled for a diagnosis of PTSD. The patient's response must have involved intense fear and helplessness. Throughout the course of the disorder, a patient will reexperience this event recurrently in one or more ways, including intrusive memories, upsetting dreams, “flashbacks,” or a reliving of the event, perhaps in hallucinatory form, with distress at the exposure to stimuli that remind him of the event, and experience physiological fear. Patients avoid any stimuli that is associated with the precipitating event, in three or more of the following ways: avoiding thoughts, feelings, or talking; avoid places, people, or activities; inability to remember a part of the event; anhedonia; asociality; blunted affect; sense of no future. Arousal is indicated by two or more of the following: insomnia, irritability, difficulty in attention, hypervigilance, exaggerated startle response. The various PTSD scales document these symptoms in detail. All of them must last for longer than 1 month, and the illness must cause significant impairments in some areas of functioning. PTSD is considered Acute if it lasts for less than 3 months and Chronic if it lasts for longer. Usually, trauma survivors show symptoms within 6 months of the event, but if not, they are considered to have...
Delayed Onset. In ASDs the symptom picture is similar, but the condition should last no more than 1 month from experiencing the extreme stressor.

People diagnosed with GAD not only worry excessively, but they meet the criteria for worrying more days than not for at least 6 months about a multitude of events or activities. GAD patients find it difficult, if not impossible, to control their feelings of worry, and this anxiety is associated with at least three of the following symptoms: restlessness, fatigue, difficulty concentrating or “mind going blank,” irritability, muscle tension, or significant sleep disturbance of any kind. This anxiety cannot be due to another physical or mental disorder, or to the effects of a substance. As always, this worry and anxiety must cause the client significant amounts of distress or impairment in domains of their life, such as social or occupational.

Formal neuropsychological testing is of particular interest in the cases of OCD and PTSD where there are extensive literatures regarding brain function. Emphasis is generally placed on memory and executive function, individuals with these diagnoses typically having normal language and usually average or above general intelligence. Even in these cases, referral for neuropsychological assessment is relatively infrequent, and is generally made because of suspicion of some other disorder, usually substance abuse or a general medical condition. The clinician should not be surprised to find normal neuropsychological results in many individuals with anxiety disorders. The neuropsychological deficits reported in the literature often involve functions that require specific tests to evaluate, many of which are not contained in standard neuropsychological batteries. The task of the clinical neuropsychologist is often that of identifying a disorder of brain function associated with some other illness in the context of an anxiety disorder. A typical question involves whether impaired function may be a product of disabling anxiety. In any event, the most useful clinical procedures appear to be tests of executive abilities and memory (Fenger et al., 2005; Kuelz, Hohagen, & Vodeholzer, 2004).

TREATMENT

Treatment for all anxiety disorders is mostly the same, as psychologists and psychiatrists tend to use cognitive behavioral therapy (CBT), medications, and sometimes psychotherapy.

Behavioral therapy methods have been commonly used. Behavioral methods were mainly used in combination with other treatments but not much alone (Goisman et al., 1993). Formal psychotherapy for PD, social phobia, and GAD has all decreased in the past decades, but cognitive and behavioral treatments are still far less used than psychodynamic therapies (Goisman, Warshaw, & Keller, 1999). However, the use of orthodox psychoanalysis, commonly used early in the 20th century, is rarely used today.

One popular and effective type of CBT is in vivo exposure, in which an anxious patient is put in a feared situation over and over, learning how to deal with their responses with the help of the therapist. This is much more effective than imaginative exposure therapy (Emmelkamp, 2002) yet now researchers are finding that exposure therapy given in the form of virtual reality is just as effective as in person in vivo exposure therapy (Parsons & Rizzo, 2008). Exercising as an adjunct to CBT in treating anxiety disorders, even walking, has been found to have a significant effect on relieving symptoms rather than treatment with CBT alone (Merom et al., 2008). Unfortunately, several studies have shown through prospective experiments that patients with any anxiety disorders are very likely to have a chronic course of the disease, and that sufferers of social phobia had the smallest chance of recovery (Bruce et al., 2005).

A new theoretical perspective on PTSD and phobias (including SAD) is that a neutral stimulus has been found to be associated with some great fear. Therefore, accelerating the cognitive process known as extinction, which replaces the fear associated with something neutral or positive over time, is a new way of looking at treatment for PTSD and phobias. An interesting study conducted by Marchand, Roberge, Primiano, and Germain (2009) found that there were no statistical differences in PD treatment outcome for CBT, exposure therapy, or supportive therapy. More recently, there has been a focus on patients’ subjective views of what helps them recover.

Subjects with SPs showed great activation reduction in associated brain areas after only two sessions of exposure therapy (Straube, 2006), but not enough studies have been conducted to tease out the differences in therapy application to the different anxiety disorder subtypes (Choy, Fyer, & Lipsitz, 2006). In vivo exposure therapy has shown strong effects in ameliorating symptoms, but patients tend to drop out (Choy et al., 2006).

Exposure therapy for SAD often consists of therapist and client working together to make a ranked list of fearful situations, since each person’s fear hierarchy is different. Then the client learns the nature of social anxiety, cognitive restructuring strategies, goes through in vivo exposure, and receives social skills training. There are standard protocols for therapists’ use (Antony, Craske, & Barlow, 2006; Antony &
 Exposure therapy along with the drug D-cycloserine, and extinction, and new research findings show that within the amygdala is very important in learning effective with SAD and other anxiety disorders. Baldwin, Bobes, Stein, Scharwachter, & Faure, 1999). are found when treated with paroxetine (2004; also see the greatest rates of remission from anxiety disorders treating anxiety disorders, as Ballenger showed that commercial name Seroquel, has much promise for tor (SSRI) type of antidepressant often known by its name clomipramine and venlafaxine, to be quite successful in relieving symptoms of OCD (for meta-analytic reviews, see Dello’osso, Nestadt, Allen, & Hollander, 2006; Fineberg & Gale, 2005). Although transcranial magnetic stimulation has been showing promise as a treatment in other mental disorders, as of yet there is little evidence for its therapeutic use in OCD (Alonso et al., 2001; Martin, Barbanoj, Pérez, & Sacristán, 2003). Psychosurgery is still considered controversial by many medical and lay parties (Moran, 2004), but cingulotomies are still performed as a last-resort surgical effort to alleviate OCD symptoms, and some studies show improvements with few side effects in patients several years afterward (Dougherty et al., 2002). Different forms of CBT have been found to be helpful: though stress management training sounds plausible, in vivo exposure and ritual prevention forms of CBT have been found to be much more successful (Simpson et al., 2008).

PTSD is often treated with CT or CBT, and several studies have empirically shown them to be effective (Rabe, Zoellner, Beauducel, Maercker, & Karl, 2008; Sibrandij et al., 2007). Rabe et al. actually documented changes in brain activity after a course of CT treatment, using electroencephalogram readings. Previous studies have shown heightened right hemisphere activity for feelings of anxiety, and in this study Rabe et al. showed a significant decrease in right anterior activity upon viewing a trauma-related image, whereas the same area brain activity did not decrease in wait-list controls (2008). Another study showed that if PTSD is treated with CBT within 3 months after trauma exposure, recovery is accelerated (Sibrandij et al., 2007). Although PTSD symptoms have been shown empirically to respond well to cognitive behavioral and trauma-focused therapies, patients do not respond well to psychodynamic, supportive, nondirective, or hypnotherapy (Bisson & Andrew, 2007). Interestingly, one experiment showed that acupuncture is significantly better at treating PTSD than group CBT (Hollifield, Sinclair-Lian, Warner, & Hammerschlag, 2007).

For GAD, individual acceptance-based behavior therapy (ABBT) that alters the intensity and/or frequency of internal experiences has arisen as a conglomerate of several other types of therapy, such as CBT for GAD, dialectical behavior therapy, and acceptance and commitment therapy. ABBT has shown promise (Roemer & Orsillo 2007; Roemer et al., 2008). The therapy involves heightening patients’ awareness of their anxious response habits and why avoiding experiences that cause such feeling make their stress worse. Clients self-monitor their progress between sessions and are encouraged to practice mindfulness everyday as well as conduct written exercises toward the same end. Mindfulness meditation in the context of mindfulness-based CT has been shown to alleviate these residual symptoms (Evans et al., 2008).
In summary, an exceptionally wide variety of behavioral and pharmacological approaches have been taken to the treatment of the anxiety disorders. Treatments of both types used individually or combined have had some success, and, in general, one could conclude that the anxiety disorders are quite treatable, with good outcomes. As new treatments emerge, such as mindfulness meditation, they are certain to be applied and evaluated.

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