I. Somatoform Disorders
   A. Preoccupied with health or body appearance.

      No identifiable medical condition causing the physical complaints.

   B. Hypochondriasis refers to physical complaints without a clear cause, and particularly severe anxiety focused on the possibility of having a serious disease.

      1. Hypochondriasis shares many features with panic disorder and other anxiety disorders, and rates of comorbidity with such disorders (and mood disorders) are high.

      2. The essential problem in hypochondriasis is anxiety, but usually present to physician because of physical complaints.

      3. Another important feature of hypochondriasis is that reassurance from numerous doctors that the person is healthy has, at best, only a short-term positive effect. Often such persons will return to the same or other doctors on the assumption that the doctor missed something initially in ruling out medical reasons for the symptoms. This disease conviction has become a core diagnostic feature of hypochondriasis.

         a. hypochondriasis vs illness phobia

         b. hypochondriasis - more likely to misinterpret physical symptoms, display higher rates of checking behaviors, have higher levels of trait anxiety, and have a later age of onset than those with illness phobia.

      4. Little is known about the prevalence of hypochondriasis in the general population.

         Approximately 3% of medical patients may meet criteria for hypochondriasis

         Sex ratio is 50:50

         May emerge at any time, with peak periods in adolescence, middle age (40s and 50s) and after age 60.

         Chronic course.

         Focus of symptoms varies from culture to culture.
5. **Etiology:**

Hypochondriasis is believed to be caused by distorted **cognitive** or **perceptual and emotional factors**. For example, persons with hypochondriasis tend to interpret ambiguous stimuli such as minor pain as threatening. These cognitive distortions and increased self-focusing tend to create anxiety and subsequently more physical symptoms in the person, which exacerbates the symptoms. Persons with hypochondriasis also have a restricted concept of health as being totally symptom free.

a. Other etiological factors may include **genetic vulnerabilities**, **overreaction to stress**, a tendency to view negative life events as unpredictable and uncontrollable, and **modeling of illness behaviors by others**.

b. In addition, persons with hypochondriasis may develop the disorder in the context of a **stressful life event**, experience a disproportionate incidence of familial disease during **childhood**, and/or **receive substantial attention for illness-related behaviors**.

6. Little is known about **treating hypochondriasis**. Recent studies suggest that cognitive-behavioral treatments, incorporating **identifying and challenging illness-related misinterpretations of physical symptoms**, **showing patients how to voluntarily produce the symptoms**, **coaching patients to rely less on reassurance**, and **stress management**, seem to be helpful. Little evidence exists to support traditional psychodynamic treatment for hypochondriasis. More recent approaches attempt to offer **more substantial and sensitive reassurance than is typical** in a physician’s office and with some encouraging results.

C. **Somatization disorder** (known as **Briquet’s syndrome** until 1980), involves an extended history of physical complaints before age 30 and substantial impairment in social or occupational functioning. The textbook presents the case of Linda to illustrate somatization disorder.

1. Persons with somatization disorder are concerned about the symptoms themselves, not what they might mean. Moreover, they show little urgency to respond to, or take action about, their symptoms, despite feeling continually weak and ill. In somatization disorder, the symptoms become a major part of the person’s identity. The DSM-IV requires that the person report 8 symptoms to meet diagnostic criteria, whereas the diagnosis **undifferentiated somatoform disorder** is reserved for persons who report fewer than 8 symptoms.
2. Somatization disorder is rare, and prevalence rates range from 4.4% (in a large city) to 20% of a large sample of primary care patients.

Typical age of onset is adolescence.

Often unmarried women of lower socioeconomic status.

Chronic course.

3. Somatization disorder shares features with hypochondriasis, including a history of family illness or injury during childhood.

Data are mixed regarding genetic contributions, although somatization disorder is strongly linked in family studies to antisocial personality disorder (ASPD).

a. Some evidence suggests that somatization disorder and ASPD (as well as substance abuse and attention deficit hyperactivity disorder) share a neurobiologically-based disinhibition syndrome.

People with these disorders may possess a weak behavioral inhibition system (BIS) that does not control the behavioral activation system (BAS). The BAS is a brain system that underlies impulsivity, thrill-seeking behavior, and excitability, whereas the BIS is involved in sensitivity to threat or danger and avoidance of situations or cues suggesting that threat or danger is imminent.

Many behaviors and traits associated with somatization disorder also seem to reflect short-term gain (i.e., active BAS) and insensitivity for long-term problems (i.e., weak BIS).

b. Another possibility (not mentioned in book) childhood trauma – physical and/or sexual abuse.

c. The major difference between somatization disorder and ASPD, however, may involve level of dependency. Whereas males tend to display aggression and ASPD, females tend to display dependency and little aggression. Therefore, gender socialization may direct a specific biological vulnerability.
4. **Treatment of somatization disorder** is exceedingly difficult, and no treatment exists with demonstrated effectiveness. Treatment of somatization disorder typically involves attempts to reduce the person's tendency to visit numerous medical specialists according to the "symptom of the week."

Use of a **gatekeeper physician**, one assigned to screen all physical complaints and decide on whether further evaluation is warranted, can be helpful.

Additional attention is directed at reducing the supportive consequences of relating to significant others on the basis of physical symptoms.

D. **Conversion disorders** refer to physical malfunctioning without any physical or organic pathology to account for the malfunction, especially in sensory-motor areas. Examples include paralysis, aphonia (i.e., difficulty speaking), mutism, analgesia, seizures, blindness, loss of sense of touch, globus hystericus (i.e., sensation of lump in throat), and astasia-abasia (i.e., weakness in legs and loss of balance). Most conversion symptoms suggest some kind of neurological disease, but can mimic the full range of physical functioning. Conversion disorder is illustrated in the textbook with the case of Eloise.

1. Freud popularized the term "conversion," believing that anxiety from unconscious conflicts is somehow converted into physical symptoms to find expression (i.e., anxiety is displaced onto a more acceptable object, in this case physical problems).
2. Several differences exist among those with conversion disorder, actual physical disorder, malingering (i.e., deliberately faking symptoms), factitious disorder (i.e., symptoms are feigned and under voluntary control, but without any obvious reason for doing so aside from assuming the sick role and to gain attention)

factitious disorder by proxy (i.e., caregiver making others sick; sometimes referred to as Munchausen's syndrome by proxy).

a. First, as with somatization disorder, conversion disorder is often (but not always) marked by la belle indifference, or a general apathy toward one's symptoms.

b. Second, conversion symptoms are usually precipitated by some stressful event.

c. Third, those with conversion disorder often function normally but display little insight into this ability. Still, an awareness of sensory and motor information is disturbed. ("Blind" people who can maneuver out of the way of obstacles, but aren't aware that they can see them. Those who are faking will score below chance on tests of vision.)

d. In general, those with conversion disorder are dissociated from sensory-motor awareness, whereas those who mangle or have a factitious disorder attempt to fake this effect (e.g., by faking blindness) and often look worse than blind persons who perform at chance levels on visual discrimination tasks.

3. Unconscious processes are salient features of conversion disorders. It is known that persons with small localized damage to certain parts of their brains can identify objects in their field of vision, but without awareness that they could, in fact, see. The textbook presents the case of Celia to illustrate this concept.
4. Conversion disorders are rare, and prevalence estimates in neurological settings range from 1 to 30%, whereas in epilepsy setting the range is between 10 and 20% of cases.

Seen primarily in women and typically develop during adolescence or shortly thereafter.

More often in less educated, lower socioeconomic status groups where knowledge about disease and medical illness is not well developed.

Conversion reactions are not uncommon in soldiers exposed to combat.

Symptoms often disappear, but return later in the same or similar form when a new stressor occurs.

5. The **Freudian psychodynamic** view postulates four basic processes in the development of conversion disorder:
   a. Experience of a traumatic event, or unacceptable unconscious conflict.
   b. The person represses the unacceptable conflict and resulting anxiety, thereby making it unconscious.
   c. Anxiety continues to fester and increase and threatens to emerge into consciousness. The person converts the conflict into physical symptoms, and thereby relieves the pressure of having to deal directly with the conflict. The reduction in anxiety is the **primary gain** or reinforcing event that maintains the conversion symptom. Primary gain accounts for the la belle indifference as the conversion resolution of the conflict would not be upsetting to the patient.
   d. Individual receives greatly increased attention and sympathy from loved ones. Freud considered attention/avoidance to represent **secondary gain**.

6. Little data exists to support Freud’s account; though the role of trauma does have support. A modification of Freud’s approach stipulates that, following the traumatic event, patients develop symptoms purposefully but detach this motivation from consciousness. The behaviors are subsequently maintained by negative reinforcement. (Movie—Tommy)

7. Treatment of conversion disorder is similar to treatment for somatization disorder. A core strategy is to identify and attend to the trauma or stressful life event and to remove sources of secondary gain. The therapist may also work to reduce reinforcing or supportive consequences of the conversion symptoms (i.e., secondary gain).
E. **Pain disorder** refers to a disorder where there may have been initial clear reasons for pain, but where psychological factors play a large role in the persistence of pain. It is difficult to judge cases where the causes were primarily physical vs. psychological. An important feature of pain disorder is that the pain is real and it hurts. The textbook presents the cases of a medical student and a woman with cancer to illustrate pain disorder.

F. Persons with **body dysmorphic disorder (BDD)** (or imagined ugliness) display a preoccupation with some imagined defect in appearance despite reasonably normal appearance. That is, the focus is on physical appearance. Reaction to perceived distortions in facial features is common. These persons are fixated on mirrors, engage in suicidal behavior, display ideas of reference (i.e., thinking that events in the world are somehow related to them and their imagined defect) and avoidance, and experience severe disruption in daily functioning. The condition was previously known as dysmorphophobia (i.e., fear of ugliness). The textbook illustrates BDD with the case of Jim.

   1. The predominant focus of attention in adolescence is skin and hair. The disorder is largely influenced by cultural standards of beauty. Examples include skin condition, facial width, slope of nose, and lip, neck, and foot size.

   2. Many persons with BDD become fixated on mirrors and frequently check their appearance. Others show a phobic fear and avoidance of mirrors. Suicidal ideation, attempts, and suicide completion are frequent consequences of BDD.

   3. Best estimates of the **prevalence of BDD** are that it is more common than previously thought and that it tends to run a lifelong, chronic course if left untreated. BDD is seen equally in males and females, few marry, and age of onset ranges from early adolescence through the 20s, peaking at age 18 or 19. BDD is not seen frequently in mental health settings as BDD sufferers frequently seek out plastic surgeons.

   4. **Little is known about the causes or treatment of BDD**, including whether BDD runs in families, biological and predisposing vulnerabilities. Obsessive-compulsive disorder tends to co-occur with BDD and both disorders share similar features (e.g., intrusive thoughts, checking). There are two and only two treatments for BDD with any evidence of effectiveness.

      a. **SSRIs**, such as clomipramine (Anafranil) and fluvoxamine (Luvox) provide relief for some people; both drugs also work for OCD.

      b. **Cognitive-behavior therapy**, specifically exposure and response prevention, has been successful with BDD and of course OCD.
5. BDD is big business for plastic surgeons. As many as 25% of persons requesting plastic surgery meet criteria for BDD. Persons with BDD do not benefit from plastic surgery, and preoccupation with imagined ugliness may actually increase following plastic surgery.

II. Dissociative Disorders

A. **Dissociative disorders** characterize alterations or detachments in consciousness or identity involving either dissociation or depersonalization. Dissociative disorders include depersonalization disorder, dissociative amnesia, dissociative fugue, dissociative trance disorder, and dissociative identity disorder. Each involves extreme manifestations of normal variants of depersonalization and derealization experiences.

1. **Depersonalization** involves distortion in perception such that a sense of reality is lost. Symptoms of unreality are characteristic of dissociative disorders because depersonalization is a psychological mechanism whereby one dissociates from reality.

2. **Derealization** involves losing a sense of the external world (e.g., things may seem to change shape or size; people may appear dead or mechanical).

3. Feelings of depersonalization and derealization are also part of other disorders, including panic and acute stress disorder.

B. **Depersonalization disorder** is a very rare condition involving severe and frightening feelings of unreality and detachment such that they dominate an individual's life and interfere with normal functioning. The textbook illustrates depersonalization disorder with the case of Bonnie.

1. Primary problem involves depersonalization and derealization.

2. Limited data suggest that 50% of persons suffering from depersonalization disorder also have another mood or anxiety disorder; mean onset is approximately 16.1 years of age, and the disorder tends to be chronic.

3. Depersonalization is related to a distinct cognitive profile, reflecting cognitive deficits in attention, short-term memory, and spatial reasoning. Such persons are easily distracted. Such deficits correspond with reports of tunnel vision (i.e., perceptual distortions) and mind emptiness (i.e., difficulty absorbing new information).
C. **Dissociative amnesia** represents several forms of psychogenic memory loss and is most often found in females. The textbook illustrates dissociative amnesia with the case of "The Woman Who Lost Her Memory."

1. Those with **generalized amnesia** are unable to recall anything, including their identity. Generalized amnesia may be lifelong or may extend from a period in the more recent past.

2. More common is **localized or selective amnesia**, or a failure to recall specific (usually traumatic) events during a specific period of time. In most cases of amnesia, the forgetting is very selective for traumatic events or memories rather than being generalized.

D. **Dissociative fugue** is related to dissociative amnesia. Persons with dissociative fugue just take off, and later find themselves in a new place, unable to remember why or how they got there, including an inability to recall their past. Often a new identity is assumed. Dissociative amnesia and fugue usually begin in adulthood, with rapid onset and dissipation, and most are female. Dissociative fugue is illustrated in the textbook with the case of the misbehaving sheriff.

1. A related non-Western variation of dissociative fugue is called amok (as in running amok). This disorder is most often seen in males and involves a trancelike state where the person often brutally assaults or sometimes kills persons or animals. Such persons usually do not remember the episode.

E. **Dissociative trance disorder** represents a condition that differs in important ways across cultures. In this condition, the symptoms resemble those of other dissociative disorders, with the exception that dissociative symptoms and sudden changes in personality are attributed to possession of a spirit known to a particular culture. This disorder is more common in women and is often associated with some life stressor. Dissociative traces commonly occur in India, Nigeria, Thailand, and other Asian and African countries. This condition is considered abnormal only if the trance is undesirable and pathological by members of the particular culture.

F. **Dissociative identity disorder (DID)** involves the adoption of new identities (as many as 100) inside one body and mind. These identities often display their own unique set of behaviors, voice, and posture. Often the identities have little or no knowledge of each other. The defining feature of DID is dissociation of certain aspects of personality. The textbook illustrates DID with the case of Jonah–Bewildering Blackouts.

1. The term **alter** refers to the different identities or personalities in DID. Many patients have at least 1 impulsive alter, and cross-gendered alters are not uncommon.

2. A **host** is typically the identity that seeks treatment and the identity that tries to keep fragments of identity together; though the host often ends up becoming overwhelmed in the process. The host identity often develops later than the other identities.
The term switch refers to the transition from one personality to another. Often a switch is instantaneous and may include physical transformations (e.g., posture, facial patterns).

Can DID be faked?

a. First, evidence indicates that persons with DID are suggestible, creating the possibility that personalities are developed from therapists' leading questions. The example of Kenneth Bianchi is presented. The symptoms of DID could be accounted for by therapists who inadvertently suggest the existence of alters to suggestible individuals—a model known as a sociocognitive model.

b. A recent survey of American psychiatrists showed little consensus on the scientific validity of DID.

c. Objective tests suggest that many people with fragmented identities are not consciously or voluntarily faking (e.g., optical changes, measures of visual acuity, eye muscle balance).

d. Persons with DID, unlike malingerers, are more likely to hide their symptoms.

Average number of identities in DID patients is close to 15. The ratio of females to males with DID is high (9:1). Onset of DID is almost always in childhood, often as young as 4 years of age. DID tends to run a chronic lifetime course if left untreated. Good prevalence data are lacking, but estimates range from 3 to 6% in the United States. DID is associated with high rates of comorbidity.

Almost all patients presenting with DID have histories of horrible, unspeakable, child abuse, usually sadistic sexual or physical abuse. It is believed that DID is rooted in a natural tendency to escape or dissociate from the unremitting negative affect associated with severe abuse. A lack of social support during the abuse is also important. There is a growing opinion that DID is a very extreme subtype of PTSD, with an emphasis on the process of dissociation in DID over anxiety as in PTSD. DID is unlikely to develop after age 9.

Persons who eventually develop DID may have a tendency to be highly suggestible or possess the ability to have a creative fantasy life. For example, 50% of those with DID remember imaginary playmates in childhood. It is therefore possible that those who are suggestible or hypnotizable are also those who can use dissociation as a defense against traumatic events (i.e., autohypnotic model).

There is a biological vulnerability to DID, but it is difficult to pinpoint. For example, about half of patients with temporal lobe epilepsy display some type of dissociative symptoms (e.g., development of a new identity fragment). In addition, dissociative
symptoms are not associated with trauma in seizure patients, as they are with DID patients without seizure disorders.

9. Evidence supporting the existence of distorted or illusory memories comes from experiments by Elizabeth Loftus and her colleagues. The evidence across numerous studies suggests that memories can be planted by strong suggestions by authority figures.

G. Persons with dissociative amnesia and fugue state usually get better on their own without treatment and remember what they have forgotten. For DID, however, the focus is on reintegration of identities and much of the treatment follows similar treatments for PTSD. The fundamental treatment goal with DID is to identify cues or triggers that provoke memories of trauma and/or dissociation and to neutralize them. The patient must also confront and relive the early trauma and gain control over memories of the horrible events. There is no evidence that hypnosis is a necessary part of treatment.
CASE REPORT

MH, a 28 year old male, presented to the psychiatric unit in a distressed and intoxicated state. He described a seven week history of increasing dysphoria associated with marked suicidal ideation which he related to two recent bereavements. Firstly the tragic death of his wife in a road traffic accident seven weeks previously, followed three weeks later by the death of his mother due to a "heart attack". He also indicated that his wife had been seven months pregnant at the time of the accident and went on to describe in graphic detail the traumatic experience of having to identify her body. Mental state examination revealed a depressed mood and a preoccupation with feelings of anger related to the loss of his wife. He described feelings of hopelessness and reported that he had on one occasion "walked in front of a bus" and on another had been restrained from "jumping into the river". He reported an alcohol intake in excess of forty units per day since the death of his wife but denied previous excessive alcohol consumption.

Past history revealed that M was the youngest of fourteen children. Three of these had died as the result of "a motorbike accident", "leukemia" and "cancer of the throat" respectively. He described a happy childhood without emotional difficulties in a stable and supportive family. A student of moderate academic ability, he was a good mixer and left school at fifteen having obtained the group certificate. He then worked as a mechanic in his home town but became increasingly estranged from his family because he was "a bit wild and hot headed". He emigrated to the United Kingdom where he worked as an electrician. Soon after this he met his wife and they married after a brief courtship. He reported having little contact with his family in Ireland apart from a brief period when he returned home after the sudden death of his father five years previously.

He denied any past psychiatric or medical history and stated that he had been in excellent psychological health until "suddenly everything was taken away". He was admitted with a diagnosis of a severe adjustment reaction secondary to the recent bereavements.

The initial hospital course was relatively uneventful. M's case was viewed sympathetically by staff members who were supportive and concerned. He refused permission for his family to be contacted stating that they wouldn't be interested. His suicidal ideas gradually abated over a four week period and he was discharged with a plan for active follow up including bereavement counselling. He re-presented two weeks later in a distressed and intoxicated state. He reported depressed mood associated with anergia, early morning wakening, anorexia and weight loss. He was commenced on antidepressant medication and made a rapid recovery and was discharged after a three week admission.

M re-presented one week later with a similar clinical picture and was re-admitted. Two days later the hospital was contacted by M's landlord who had "heard that M had been killed in a car accident". When questioned about this M offhandedly explained that his landlord had just got mixed up. He again refused permission for his family to be contacted. Liaison with his bereavement counsellor revealed that he had resisted anything other than the most superficial discussion of events and had especially objected to producing any photographs of his wife. Soon after M, who had hitherto been an affable and co-operative patient became threatening
and hostile with the staff and left hospital against medical advice. At this point the validity of M's story was questioned and, because of concern about the patient's threatening mental state, attempts were made to contact his family through the police in his stated home town.

His family were successfully contacted and indicated that M was in fact single and that his mother and eleven siblings were all alive and well. The three siblings previously reported as deceased were in fact non-existent. Further questioning revealed that he had no known previous psychiatric contact and had resided in his home town until four months previously when he had left after an argument at work over a forged cheque. He had no other forensic history.

M re-presented five days later reporting the same history as previously but with a number of minor omissions including the story of his three deceased siblings. He also changed the date of his marriage. He was confronted with these inconsistencies and with the details of the collateral history from his family. He adamantly maintained that his story was true but refused admission. One year later he has not re-presented.

http://www.priory.com/psych/fact.htm
Multiple Personality Disorder

(Dissociative Identity Disorder)

by Paul R. McHugh

When this essay was written, Paul McHugh, MD, was Henry Phipps Professor of Psychiatry and Director of the Department of Psychiatry and Behavioral Science at the Johns Hopkins Medical Institutions in Baltimore.

Prompted by the unexpected flourishing of this extraordinary diagnosis, students often ask me whether multiple personality disorder (MPD) really exists. I usually reply that the symptoms attributed to it are as genuine as hysterical paralysis and seizures and teach us lessons already learned by psychiatrists more than a hundred years ago.

Consider the dramatic events that occurred at the Salpetriere Hospital in Paris in the 1880s. For a time the chief physician, Jean-Martin Charcot, thought he had discovered a new disease he called "hystero-epilepsy," a disorder of mind and brain combining features of hysteria and epilepsy. The patients displayed a variety of symptoms, including convulsions, contortions, fainting, and transient impairment of consciousness. Charcot, the acknowledged master of Parisian neurologists, demonstrated the condition by presenting patients to his staff during teaching rounds in the hospital auditorium.

A skeptical student, Joseph Babinski, decided that Charcot had invented rather than discovered hystero-epilepsy. The patients had come to the hospital with vague complaints of distress and demoralization. Charcot had persuaded them that they were victims of hystero-epilepsy and should join the others under his care. Charcot's interest in their problems, the encouragement of attendants, and the example of others on the same ward prompted patients to accept Charcot's view of them and eventually to display the expected symptoms. These symptoms resembled epilepsy, Babinski believed, because of a municipal decision to house epileptic and hysterical patients together (both having "episodic" conditions). The hysterical patients, already vulnerable to suggestion and persuasion, were continually subjected to life on the ward and to Charcot's neuropsychiatric examinations. They began to imitate the epileptic attacks they repeatedly witnessed.
Babinski eventually won the argument. In fact, he persuaded Charcot that doctors can induce a variety of physical and mental disorders, especially in young, inexperienced, emotionally troubled women. There was no "hystero-epilepsy." These patients were afflicted not by a disease but by an idea. With this understanding, Charcot and Babinski devised a two-stage treatment consisting of isolation and counter suggestion.

First, "hystero-epileptic" patients were transferred to the general wards of the hospital and kept apart from one another. Thus they were separated from everyone else who was behaving in the same way and also from staff members who had been induced by sympathy or investigatory zeal to show great interest in the symptoms. The success of this first step was remarkable. Babinski and Charcot were reminded of the rare but impressive epidemic of fainting, convulsions, and wild screaming in convents and boarding schools that ended when the group of afflicted persons was broken up and scattered.

The second step, countersuggestion, was designed to give the patients a view of themselves that would persuade them to abandon their symptoms. Dramatic countersuggestions, such as electrical stimulation of "paralyzed" muscles, proved to be unreliable. The most effective technique was simply ignoring the hysterical behavior and concentrating on the present circumstances of these patients. They were suffering from many forms of stress, including sexual feelings and traumas, economic fears, religious conflicts, and a conviction (perhaps correct) that they were being exploited or neglected by their families. In some cases their distress had been provoked by a mental or physical illness. The hysterical symptoms obscured the underlying emotional conflicts and traumas. How trivial a sexual fear seemed to a patient in whom convulsive attacks produced paralysis and temporary blindness every day!

Staff members expressed their withdrawal of interest in hysterical behavior subtly, in such words as, "You're in recovery now and we will give you some physiotherapy, but let us concentrate on the home situation that may have brought this on." These face-saving countersuggestions reduced a patient's need to go on producing hysteroepileptic symptoms in order to certify that her problems were real. The symptoms then gradually withered from lack of nourishing attention. Patients began to take a more coherent and disciplined approach to their problems and found a resolution more appropriate than hysterical displays.

The rules discovered by Babinski and Charcot, now embedded in psychiatric textbooks and confirmed by decades of research in social psychology, are
being overlooked in the midst of a nationwide epidemic of alleged MPD that is wreaking havoc on both patients and therapists. MPD is an iatrogenic behavioral syndrome, promoted by suggestion, social consequences, and group loyalties. It rests on ideas about the self that obscure reality, and it responds to standard treatments.

To begin with the first point: MPD, like hystero-epilepsy, is created by therapists. This formerly rare and disputed diagnosis became popular after the appearance of several best-selling books and movies. It is often based on the crudest form of suggestion. Here, for example, is some advice on how to elicit alternative personalities (alters, as they have come to be called), from an introduction to MPD by Stephen E. Buie, M.D., who is director of the Dissociative Disorders Treatment Program at a North Carolina hospital:

"It may happen that an alter personality will reveal itself to you during this [assessment] process, but more likely it will not. So you may have to elicit an alter... You can begin by indirect [sic] questioning such as, 'Have you ever felt like another part of you does things that you can't control?' If she gives positive or ambiguous responses ask for specific examples. You are trying to develop a picture of what the alter personality is like...At this point you may ask the host personality, "Does this set of feelings have a name?"...Often the host personality will not know. You can then focus upon a particular event or set of behaviors. 'Can I talk to the part of you that is taking those long drives in the country?'"

Once patients have permitted a psychiatrist to "talk to the part...that is taking these long drives," they are committed to the idea that they have MPD and must act in ways consistent with this self-image. The patient may be placed on a hospital service (often called the dissociative service) with others who have given the same compliant responses. The emergence of the first alter breaches the barrier of reality, and fantasy is allowed free rein. The patient and staff now begin a search for further alters surrounding the so called host personality. The original two or three personalities proliferate into 90 or 100. A lore evolves. At least one alter must be of the opposite sex (Patricia may have Penny but also must have Patrick). Sometimes it is even suggested that one alter is an animal. A dog, cat, or cow must be found and made to speak! Individual alters are followed in special notes for the hospital record. Every time an alter emerges, the hospital staff shows great interest.

The search for fresh symptoms sustains the original commitment while cultivating and embellishing the suggestion. It becomes harder and harder for
a patient to say to the psychiatrist or to anyone else, "Oh, let's stop this. It's just me taking those long drives in the country."

The cause of MPD is supposed to be childhood sexual trauma so horrible that it has to be split off (dissociated) from the host consciousness and lodged in the alters. Patient and therapist begin a search for alters who remember the trauma and can identify the abusers. Thus commitment to the diagnosis of MPD is enhanced by the sense that a crime is being exposed and justice is being done. The patient now has such a powerful vested interest in sustaining the MPD enterprise that it almost becomes an end in itself.

Certainly these patients, like Charcot's, have many emotional conflicts and have often suffered traumatic experiences. But everyone is distracted from the patient's main problems by a preoccupation with dramatic symptoms, and perhaps by a commitment to a single kind of psychological trauma. Furthermore, given that treatment may become interminable when therapists concentrate on fascinating symptoms, it is no wonder that MPD is regarded as a chronic disorder that often requires long stretches of time on dissociative units.

Charcot removed his patients from the special wards when he realised what he had been inventing. We can do the same. These patients should be treated by the same methods Charcot used--isolation and countersuggestion. Close the dissociation services and disperse the patients to general psychiatric units. Ignore the alters. Stop talking to them, taking notes on them, and discussing them in staff conferences. Pay attention to real present problems and conflicts rather than fantasy. If these simple, familiar rules are followed, multiple personalities will soon wither away and psychotherapy can begin.

http://www.psycom.net/mchugh.html
Neurochemical deficits with hypochondriasis and some other somatoform disorders (eg, BDD) appear similar to those of depressive and anxiety disorders. For example, the aforementioned obsessive-compulsive spectrum described by Hollander et al in 1992 includes OCD, BDD, anorexia nervosa, Tourette syndrome, and impulse control disorders (eg, trichotillomania, pathological gambling). Although only preliminary data have been reported on these neurochemical deficits, such deficits may explain why symptoms overlap, why the disorders are commonly comorbid, and why treatments may parallel one another (eg, SSRIs).

http://www.emedicine.com/MED/topic3122.htm

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OCD

Related Spectrum Disorders

It is now hypothesised that OCD is closely related to a variety of other spectrum or related disorders (Hollander, 1997). Up to 10% of the U.S. population may suffer from an OCD spectrum disorder, compared with 2% to 3% with OCD. Spectrum disorders all involve some degree of compulsive or impulsive behaviour and consist of any of three specific core symptoms:

- pre-occupation or obsession with specific bodily sensations or appearance (e.g., body dysmorphic disorder, depersonalisation, eating disorders, hypochondriasis);
- selected neurologic disorders (e.g., Tourette’s syndrome, Sydenham’s chorea, torticollis, autism) often involving basal ganglia dysfunction presenting with repetitive behaviours;
- and impulsivity or certain types of impulse control disorder (e.g., sexual compulsions, trichotillomania, pathological gambling, kleptomania, and self-injurious behaviour).

Spectrum disorders share other characteristics with OCD including features such as age at onset, clinical course, family history, and response to SSRIs and behavioural therapy. Such behaviours can be placed on a risk-aversive/impulsive spectrum. At the compulsive end of the scale are an exaggerated sense of harm and a heightened sense of risk while at the impulsive end is an underestimation of harm and risk, causing behaviour that is dangerous or otherwise troublesome.

Both compulsivity and impulsivity involve difficulty in delaying or inhibiting repetitive behaviours, although the mechanisms of action differ. Compulsive behaviours are an attempt to reduce anxiety and discomfort, while impulsive actions are an attempt to obtain pleasure, arousal, or gratification.

Both genders demonstrate impulsive behaviour; men may gamble, intermittently explode with anger, set fires and act out sexual behaviour and women are more likely to steal, pull out their hair, injure themselves, shop compulsively and binge eat. It is not clear whether such gender differences are hormonal, cultural., or a combination of both (Hollander, 1997).

It seems that the SSRIs can successfully treat the OCD-related disorders. We will now discuss the role of citalopram in these disorders.

http://www.brainexplorer.org/ocd/OCD_Related_%20Spectrum_%20Disorders.shtml
Magnesium

Magnesium supplementation enjoys a broad reputation as having a calmative effect on anxiety symptoms and stress levels. Research is just beginning to highlight magnesium's effect on anxiety, with interesting results. One researcher observed decreased levels of nervousness as well as insomnia symptoms in patients supplemented with 200 milligrams of magnesium in combination with 400 milligrams calcium and an association between magnesium deficiency and anxiety symptoms was noted. A different investigation looked at the use of magnesium in post-surgical patients and its effectiveness in alleviating pain. Patients were infused with magnesium both during and following surgery and were evaluated for anxiety levels. Patients receiving the magnesium infusion required significantly less pain medication (morphine and fentanyl) in comparison to the control group that received no magnesium. Additionally, the magnesium group reported less anxiety as well. Magnesium deficiency is reportedly common in the typical American diet, with one major survey determining that adequate magnesium is lacking in nearly 72% of diets, and that nearly half of the population consumes less than 75% of the Recommended Daily Allowance (RDA) of magnesium. Furthermore, one third of these people consumed less than 50% of the RDA for magnesium. Those taking oral contraceptives, diuretic medicines and who overuse laxatives may be at risk of magnesium deficiency. Supplementing with magnesium is a simple and inexpensive means to further reduce anxiety.