Conversion Disorder (DSM-IV-TR #300.11)

Conversion disorder is characterized by the occurrence of certain signs or symptoms that are clearly inconsistent with what is known about anatomy and pathophysiology. For example, the patient may complain of blindness, yet cortical visual evoked potentials are normal. Or a patient may complain of complete anesthesia of the left upper extremity and go on to describe the boundary of the anesthesia as being a clear-cut line encircling the elbow. Other common complaints include hemiplegia, deafness, and seizures.

On close inspection the specific symptomatology in each case corresponds with the patient’s particular conception of how an illness might manifest itself. Take for example a patient who complains of such unsteadiness that walking is impossible; the patient’s conception of the malady, however, simply does not encompass the symptomatology evident at bedside examination that the physician’s knowledge of pathophysiology would predict. Thus, although the patient stumbles and lurches in the attempt to cross from chair to the bed, in bed there is no truncal ataxia and no deficiency on finger-to-nose or heel-to-knee-to-shin testing.

Patients who find themselves with such symptoms, however, are not to be confused with malingerers or those with factitious illness. Patients who suffer conversion symptoms do not intentionally feign such symptoms, as malingerers do; they experience them as genuine, and their distress over them may be as genuine as that of the patient whose unsteadiness is produced by a midline cerebellar tumor.

A synonym for conversion disorder is “hysterical neurosis, conversion type.” Both terms are to a degree unfortunate. The term “conversion” has its roots in psychoanalysis and connotes a specific etiologic theory that has not been substantiated. “Hysterical,” though an ancient term, has so many different meanings and is such a pejorative term that it might best be allowed to rest in peace.

The lifetime prevalence of conversion disorder is not known with certainty, and estimates range from 0.01% to 0.5% of the general population; it is more common in females, with female to male ratios ranging from 2:1 up to 10:1.

ONSET

Although conversion disorder may appear at any time from early childhood up to old age, most patients experience their first symptoms during adolescence or early adult years. In most cases the actual onset of symptoms is abrupt and typically follows a major stress in the patient’s life.

CLINICAL FEATURES

In general, at any given time most patients with conversion disorder have only one symptom. Some of the more common ones are listed in the box below, and most of these are described in detail below.

Conversion anesthesia may occur anywhere, but it is most common on the extremities. One may see a typical “glove and stocking” distribution; however, unlike the “glove and stocking” distribution that may occur in a polyneuropathy, the areas of conversion anesthesia have a very precise and sharp boundary, often located at a joint. The same nonphysiologic sharp boundary may be seen in conversion hemianesthesia, wherein the boundary precisely bisects the body on a sagittal plane. Other anomalies may appear on examination. Patients who complain of total lack of all sensory modalities (including vibratory sense) in the hand or foot may nevertheless have intact position sense at the index finger or the great toe. Furthermore, patients who complain of a similar complete lack of feeling in their legs are nonetheless able to walk normally and have a negative Romberg test. Some patients, when asked to close their eyes and say “yes” if they feel something and “no” if they don’t, will reliably say “no” every time the “anesthetic” area is touched. Deep tendon reflex testing fails to show the expected hyporeflexia. In doubtful cases somatosensory evoked potentials may be helpful. Anesthesia may also occur on the cornea and on the palate. Indeed in the nineteenth and early twentieth centuries these symptoms were accorded a special place among the “stigmata” of “hysteria.”

In conversion paralysis one may see the same sort of anomalous boundary as seen in conversion anesthesia. For example, the weakness may extend up to the elbow and end precisely there. In conversion hemiplegia, other abnormalities may be seen; for example, though weak for many months the affected arm may hang limply at the side, rather than displaying the typical physiologic flexion posture. Patients with conversion hemiplegia may also display the “wrong-way tongue” sign wherein the protruded tongue, instead of deviating toward the hemiplegic side, as in “true” hemiplegia, deviates instead toward the normal side. Furthermore, on observation of gait one finds that the weakened leg is dragged, rather than circumducted. On formal testing of muscle strength in the lower extremities, an attempt to elicit Hoover’s sign may be helpful. Here, with the patient recumbent, the examiner’s hands, palms up, are placed under the heel of the weakened leg and likewise under the heel of the unaffected leg,
and the patient is then asked to exert as much effort as possible in raising the affected leg from the bed. In the case, say, of weakness secondary to a stroke, although the affected leg does not move, the examiner feels considerable pressure upon the hand underneath the unaffected leg as the patient puts maximum effort into the task. In cases of conversion paralysis, however, Hoover’s sign is present, and the examiner does not feel any pressure on the hand underneath the unaffected leg. Finally, in conversion paralysis the Babinski sign is absent, and the deep tendon reflexes are not increased.

In conversion paraplegia, one finds normal, rather than increased, deep tendon reflexes, and the Babinski sign is absent; in doubtful cases the issue may be resolved by demonstrating normal motor evoked potentials.

In conversion ataxia (or, as it has been classically called, astasia-abasia), a patient, upon attempting to stand or walk, lurches and staggers forward, arms flinging and trunk swaying, always barely making it to the safety of bed or chair. Yet when examined in bed, one finds no limb or truncal ataxia.

Conversion tremor tends to be coarse and irregular and generally disappears when the patient is distracted.

Conversion seizures, also known as “hysterical fits” or “non-epileptic seizures,” may mimic either grand mal or complex partial seizures.

Conversion grand mal seizures display multiple anomalies. Their onset is gradual rather than sudden; if the patient cries out at the onset it usually consists of intelligible screaming rather than an inarticulate cry. The movements during a conversion seizure are extremely varied but generally purposeful. The patient may thrash about, strike at the walls, or break one piece of furniture over another, all in contrast to the rhythmic, simple tonic-clonic activity seen in most grand mal seizures. Most patients do not bite their tongue during a conversion seizure, and only those with considerable medical sophistication pass urine. Most conversion seizures end gradually rather than abruptly, and afterwards patients display neither confusion nor somnolence.

Conversion seizures of the complex partial type may be more difficult to diagnose. One differential point is that true complex partial seizures typically start with a motionless stare or with automatisms (such as chewing) before the onset of complex behavior, whereas conversion seizures of the complex partial type typically begin with complex behavior.

Other findings helpful in distinguishing conversion seizures from “true” seizures include a post-ictal Babinski sign, an elevated prolactin or neuron-specific enolase level, and a positive ictal or post-ictal EEG. Babinski signs are almost universal after “true” grand mal seizures, and are seen also in about one-fifth of patients after complex partial seizures. Prolactin and neuron-specific enolase levels are generally elevated after either type of seizure, and both are useful to obtain 15 to 30 minutes post-ictally. Surface EEGs are always appropriate: the ictal EEG is always abnormal during a grand mal seizure and almost always so during a complex partial seizure; post-ictal EEGs generally also show slowing after either type of seizure. As negative findings accrue (i.e., the absence of a Babinski sign, normal prolactin and neuron-specific enolase levels, and normal ictal and post-ictal EEGs) the likelihood that the event in question was a pseudoseizure increases. In doubtful cases, one may consider placebo induction, for example with an infusion of normal saline.

In conversion deafness the blink reflex to a loud and unexpected sound is present, thus demonstrating the intactness of the brain stem. Should one suspect the vanishingly rare bilateral cortical deafness, a brain stem auditory evoked potential will resolve the issue.

Bilateral conversion blindness may be suspected when the patient, though complaining of recent onset of blindness, neither sustains injury while maneuvering around the office nor displays any of the expected bruises or scrapes. The pupillary reflex is present, thus demonstrating the intactness of the optic nerve, chiasm, tract, lateral geniculate body, and mesencephalon. Should one suspect cortical blindness, a visual evoked potential will resolve the question.

In cases of monocular conversion blindness, one need only demonstrate two things to make the correct diagnosis: first, that the peripheral fields are full in the unaffected eye; and second, that the pupillary response is normal in both eyes.

Conversion aphonia may be suspected when the patient is asked to cough, for example, during auscultation of the lungs. In contrast with other aphonias, the cough is normally full and loud.

A synonym for “globus hystericus” might be conversion dysphagia; however, this term fails to convey the essence of the patient’s chief complaint, which is a most distressing sense of having a lump in one’s throat. Finding that the patient can swallow solid food with little difficulty or even that swallowing liquid may ease the discomfort argues strongly for a diagnosis of globus. In doubtful cases a chest
x-ray, video esophagram, and esophageal manometry may be required to rule out other conditions.

Conversion parkinsonism differs from most “true” parkinsonian conditions in several respects: onset is usually abrupt and the course nonprogressive, in contrast with the gradual onset and progressive downhill course seen in conditions such as Parkinson’s disease or diffuse Lewy body disease; cogwheeling is absent, and although there may be a loss of associated arm movements when walking, the arm is typically held at the side, rather than in flexion; and finally, tremor, rather than diminishing with action, may actually increase in severity. In doubtful cases, and provided that appropriate help is available, one may vigorously check for retropulsion with a sudden, unexpected and forceful push back-forward to the patient’s chest: if the patient with conversion parkinsonism does begin to fall back, one often sees an extreme loss of balance accompanied by a telltale rapid and fluid upswing of the arms.

In conversion syncope one fails to see autonomic changes, such as pallor. Furthermore, the conversion faint itself often has a “swooning” character to it, which typically heightens the drama of the moment and never results in injury.

In conversion coma a number of distinctive findings may be present, including the following: fluttering of the eyelid when the eyelashes are lightly stroked, resistance to eyelid opening and an abrupt closure of the lids when they are released, and normal pupillary responses. In doubtful cases one may resort to a surprise touch to the face with an ice cube. If the issue is still in doubt, a normal EEG will help resolve the issue.

Other conversion symptoms, of course, are possible. Like the foregoing, most suggest central or peripheral nervous system disease, such as strokes, tumors, and the like. Conversion anosmia, nystagmus, ocular bobbing, convergence spasm, facial weakness, and ageusia may occur. Conversion symptoms suggesting disease outside the nervous system, such as conversion vomiting, pseudocyesis, or cough, are symptoms suggesting disease outside the nervous system, such as strokes, tumors, and the like. Conversion anosmia, nystagmus, ocular bobbing, convergence spasm, facial weakness, and ageusia may occur. Conversion symptoms suggesting disease outside the nervous system, such as conversion vomiting, pseudocyesis, or cough, are decidedly uncommon. Regardless of the symptoms, however, the physician with enough ingenuity is generally able to demonstrate how each particular conversion symptom “violates” the laws of anatomy or pathophysiology.

Patients with conversion disorder may or may not display the classic “la belle indifférence,” the attitude of casual disregard for normally alarming symptoms such as blindness or hemianesthesia. Even when present, this sign is not reliable because it may be confused with the studied stoicism seen in patients with other illnesses or with the symptom of anosognosia.

Conversion disorder may occur in isolation; however, in many instances a personality disorder is also seen, most commonly histrionic, passive-aggressive, borderline, or, in males, antisocial. Dysthymia or major depression may also occur concurrently.

**COURSE**

Conversion disorder may pursue either an episodic or chronic course, with the initial conversion symptom remitting spontaneously, often within weeks or months. In such cases a subsequent episode may be expected in the years to come; should such a subsequent episode occur, the conversion symptom itself may be different from the initial one.

A minority of patients experience their conversion symptoms chronically, this tends to be the case with an associated personality disorder.

**COMPLICATIONS**

Should patients take to bed or restrict their activities because of conversion symptoms, jobs may be lost and relationships strained. Potentially dangerous diagnostic procedures, such as arteriography, may be undergone. In chronic cases of conversion paralysis, disuse atrophy or contractures may occur.

**ETIOLOGY**

Conversion symptoms are more common among the uneducated and unsophisticated; the actual conversion symptom itself is generally a reflection or extension of symptoms that the patient has seen in another or has personally experienced.

In most instances, consequent upon the appearance of the conversion symptom there is a reduction in the patient’s level of anxiety. Close inspection reveals that conversion symptoms are not, however, premeditated—they simply happen—and although observers may feel a “purpose” is behind them, the patient himself is unaware of any such thing. Many clinicians feel that the symptom itself may be a kind of “sign language,” or a sort of hieroglyphic that conveys what the patient is unable to put into words.

Recent PET scanning has demonstrated that in patients with conversion hemiplegia or hemianesthesia, there is a decreased activation of the contralateral basal ganglia and thalamus. The pathophysiologic relevance of this, however, is unclear. It may represent a premorbid susceptibility to the development of conversion symptoms or might, in turn, merely be epiphenomenal and unrelated to the underlying cause or causes.

**DIFFERENTIAL DIAGNOSIS**

The diagnosis of conversion disorder cannot be made unless one can demonstrate with certainty that the patient’s symptomatology clearly violates the laws of anatomy and pathophysiology. To demonstrate merely that no explanation for the symptom can be found is not sufficient, as many diseases may present in a most subtle and deceptive way. Examples include multiple sclerosis, systemic lupus erythematosus, polyarteritis nodosa, and sarcoidosis. Certain disorders, such as early torsion dystonia, akinetic seizures, or supplementary motor seizures, may tax the skills of even the most experienced diagnostician. Furthermore, it is not uncommon for patients with conversion seizures to also have “true” seizures. Where doubt persists after an exhaustive workup, one should defer on diagnosis and observe the patient over time until the clinical picture crystallizes. This is especially the case when the patient’s complaint is of pain. Although conversion pain does exist, such a diagnosis should be entertained only after a scrupulously exhaustive examination.
When the symptoms fall clearly outside the realm of anatomic possibility, one must then rule out both malingering and factitious illness. In both these cases, in contrast to conversion disorder, the patient premeditates the symptoms and intentionally feigns them with a clearly conceived purpose in mind. In malingering the purpose is to avoid some unpleasantness, such as jail. In factitious illness the purpose is to be a patient and under medical care. Demonstrating such purposefulness and premeditation is difficult and often requires prolonged observation and repeated interviews.

At this point, assuming that one has demonstrated both that the symptom lies outside the bounds of anatomic possibility and that it is neither premeditated nor does it serve any purpose that the patient is aware of, then one may with reasonable confidence say that the patient has a conversion symptom. Before making a diagnosis of conversion disorder, however, one must rule out two other disorders that may produce conversion symptoms, namely schizophrenia and Briquet’s syndrome. Schizophrenia is distinguished by the bizarreness, hallucinations and delusions characteristic of that illness. In Briquet’s syndrome one generally finds not merely one conversion symptom at a time, as is the case in conversion disorder, but multiple conversion symptoms. Furthermore, in Briquet’s syndrome the conversion symptoms are only a part, and often a minor part at that, of a much larger fabric of multiple complaints referable to multiple organ systems other than the nervous system.

TREATMENT

After the diagnosis is made, one should inform the patient in a gentle and nonjudgmental, yet quietly authoritative, way that neither the examination nor the diagnostic tests have revealed any damage to the brain or nerves. One may then confess honestly that, although medicine does not know the cause of the symptoms, it is nevertheless known that patients tend to recover in a few weeks. With such support and reassurance, a majority of patients will experience a remission during a hospital stay, and this is especially likely when the conversion symptoms have been of acute onset and short duration, and were preceded by an obvious psychosocial precipitant. In certain instances a few sessions with a physical therapist who is knowledgeable about these patients may expedite the remission, often providing a sort of “face saving” device. At all costs one must avoid pejorative statements such as “there is nothing really wrong with you,” as these only serve to undermine the physician-patient relationship.

When these measures fail, alternative techniques may be used. Hypnosis may effect a remission; however, early relapses tend to occur. Another approach involves viewing the symptoms as a kind of “sign language,” deciphering what the sign means, and then assisting the patient in putting that meaning into words and taking appropriate action. Such an approach is often labor intensive, yet the clinical impression is that it may produce solid results.

BIBLIOGRAPHY


